BIOLOGY, MEDICINE, AND SURGERY OF SOUTH AMERICAN WILD ANIMALS

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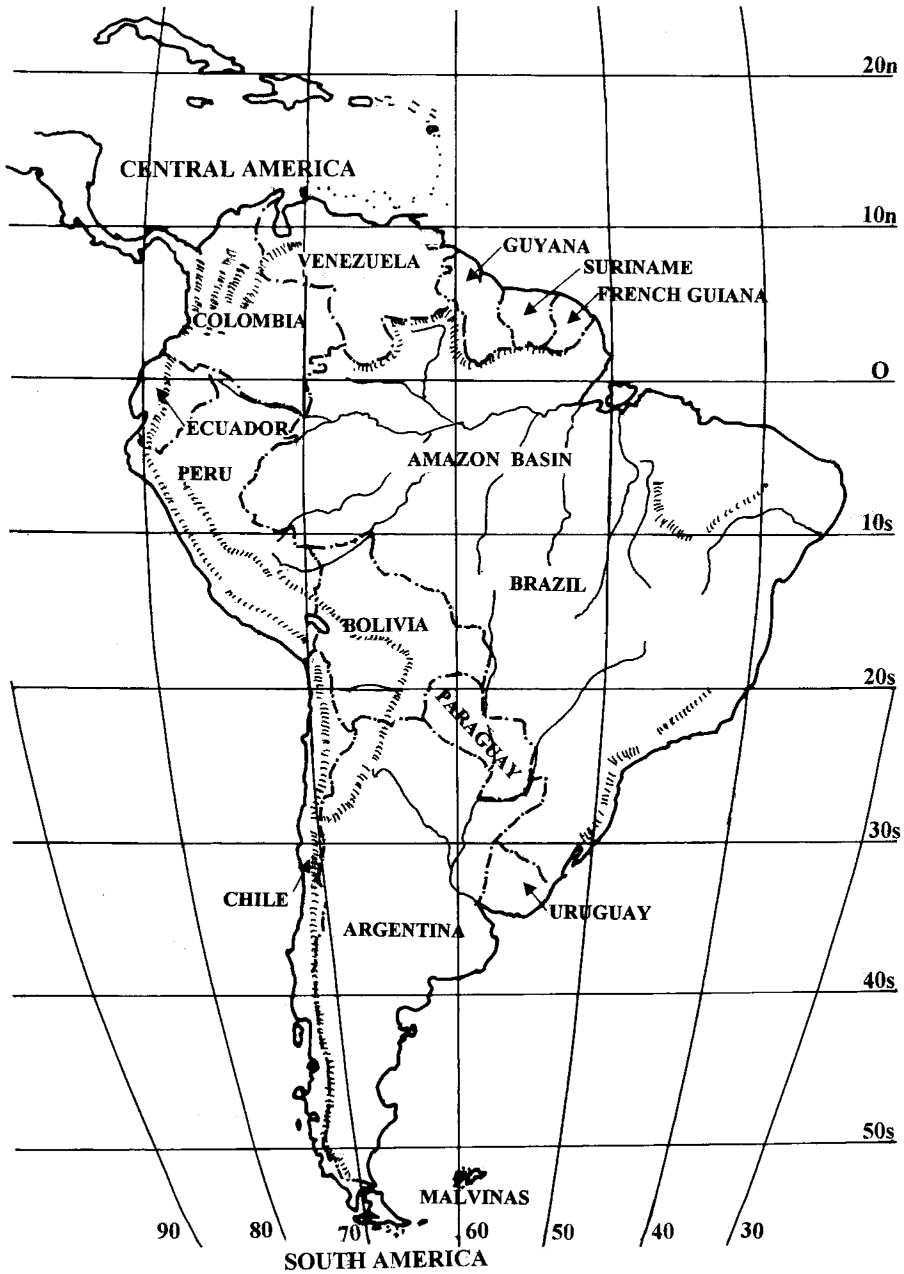

Iowa State University Press / Ames

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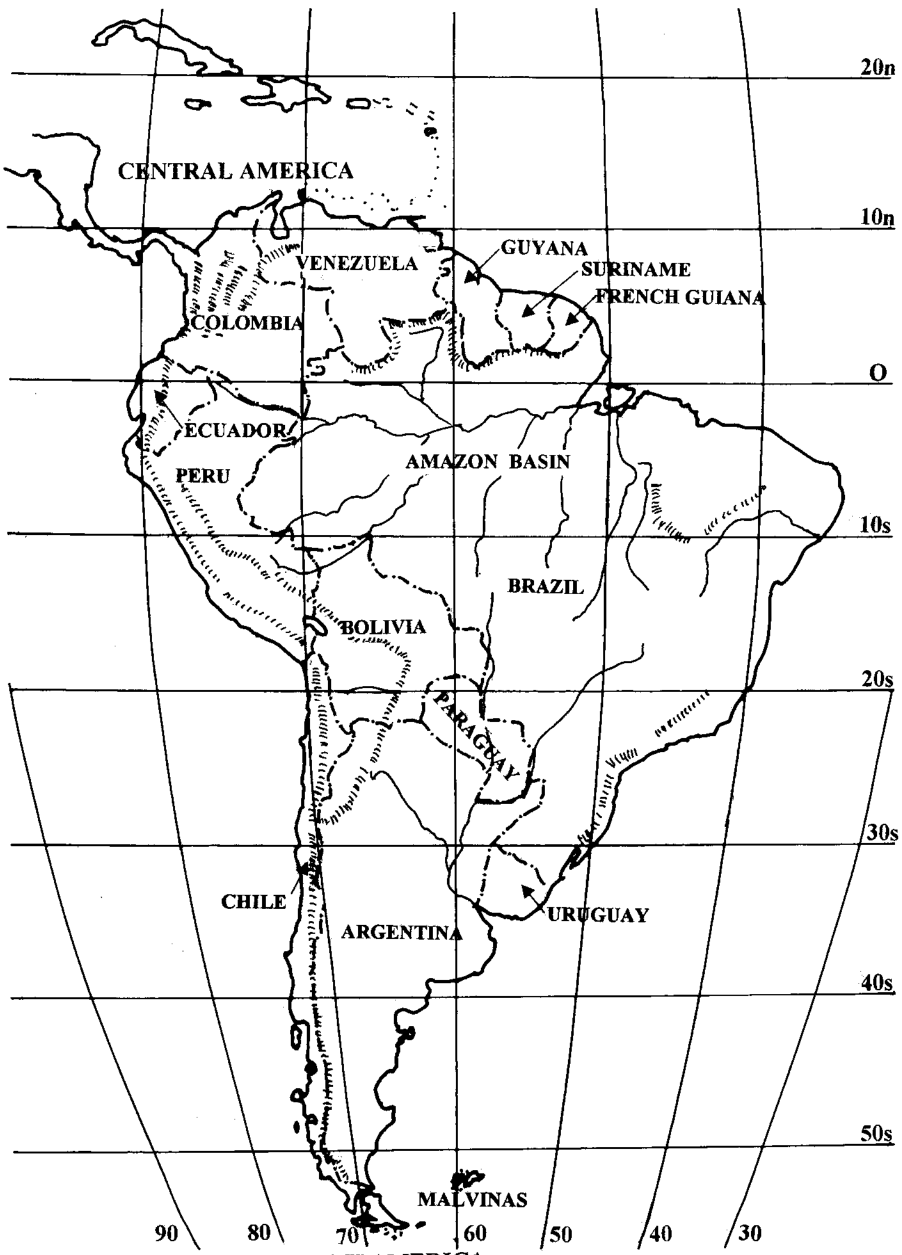

SOUTH AMERICA

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Contents

Special Topics

Preface

South America has many wonderful wild animals that are worthy of the special care given them by biologists and veterinarians. Governments in South America are beginning to recognize the importance of their wild fauna. Zoos in South America and worldwide have exhibited South American animals for decades, to the delight of the public. Concern about the continued existence of certain species has prompted a surge in the number of scientists conducting studies to ascertain as much as possible about the lives of wild animals in the free-ranging state. Zoo biologists and veterinarians are participating in cooperative captive propagation programs.

Wild animals are being managed in semi–free-ranging situations, conditions for the production of meat and fiber for human use. Endangered species of wild animals are being given special consideration by private aviculturists, zoos, rehabilitation centers, and government agencies. Captive propagation is practiced for release back into the wild (Andean condor). Some projects are international in scope (golden lion tamarin).

The time is at hand to share the experiences of South American biologists and veterinarians with the world. Numerous scientists, clinicians, administrators, rehabilitators, and academicians have contributed to this book. Except for a few North American participants, the authors are South American. The coverage of specific animal groups varies, either because little information is available or there is a lack of authors who have experience with the taxa.

The editors and authors are pleased to present this overview of the biology, medicine, and surgery of South American wild animals. It is a labor of love and dedication that wild animals may be better understood. We hope that the health and well-being of animals, both in captivity and in the free-ranging state, will be improved.

—Murray E. Fowler, D.V.M
—Zalmir S. Cubas, D.V.M

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Thanks to the many institutions, agencies, and spouses who have supported the authors in furthering the cause of wild animals in South America or wherever they may be exhibited throughout the world.

A special note of thanks is due to Dr. Zalmir Cubas, who first mentioned that the time was right for this book and then proceeded to organize the authors for the various topics.

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—Murray E. Fowler, DVM
—Zalmir S. Cubas, DVM

Amphibians/Reptiles: Classes Amphibia and Reptilia

1
Class Amphibia (Amphibians): Frogs, Toads

Sérgio Rangel Pinheiro

INTRODUCTION

The South American continent is rich in varied biotypes and a complex and little known diversity of animal and plant life. The amphibian class is large and diverse in shape, size, spot pattern, and colors. There are extreme examples among the frogs from Brazil, such as the smallest terrestrial vertebrate of South America, the little Rã-do-Foliço-da-mata (***Psyllophyrne didactyla***), with cryptic colors that match the environment (Atlantic forest in southeastern Brazil). The male is about 1.5 cm long, and the females are even smaller—about 1 cm. At the other extreme are the giant Brazilian toads of the genus ***Bufo;*** giant females collected from the caatinga of the state of Bahia (northeastern Brazil) weighed as much as 0.8 kg and measured about 30 cm long (measured in the resting position—the back legs were not expanded).

Other examples are the jewels from the rain forests, the Dendrobatids of the genera ***Epipedobates***, ***Phyllobates***, and ***Dendrobates***, known for their small size, varied brilliant colors, and high toxicity. In spite of their beauty, these amphibians don't seem to arouse much interest in zoos and commercial breeders. Some commercial breeders raise exotic amphibians, mainly for the pet trade in Brazil, Argentina, and Uruguay. The genera ***Xenopus*** and ***Hymenochyrus*** and the aquatic salamanders of the genera ***Ambyostoma*** (Axolotl) and ***Cynops*** are most commonly produced.

South American zoos successfully exhibit several species of amphibians, because they easily adapt to captivity. Concentration is on the larger species or those with exotic shapes. Generally, South American amphibians are small and not attractive (with the exception of Dendrobatids and Brachycephalins) and have no appeal for zoo visitors, who want to see large or at least odd animals with bizarre shapes and colors. Species of the genera ***Bufo*** (common toad), Figure 1.1, ***Ceratophrys*** (horned frogs), Figures 1.2 and 1.3, ***Prynoyas*** (milk frogs), Figure 1.4, and ***Pipa*** (Surinam toad) are most commonly raised in zoos. When the public is exposed to such animals in herpetariums, it helps to change false beliefs about frogs and similar animals.

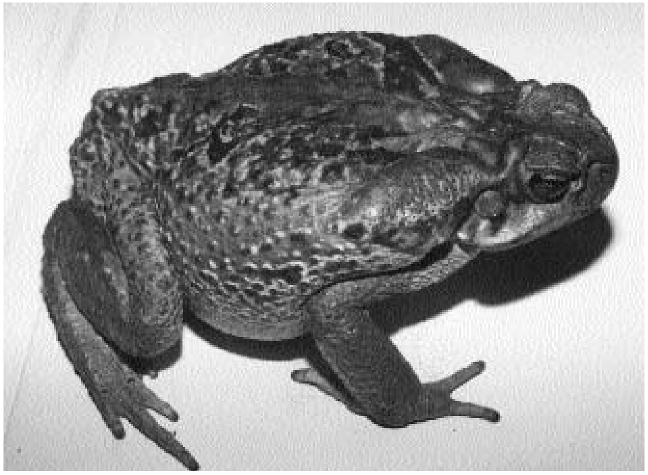


FIGURE 1.1. Common toad, female, *Bufo paracnemis*.

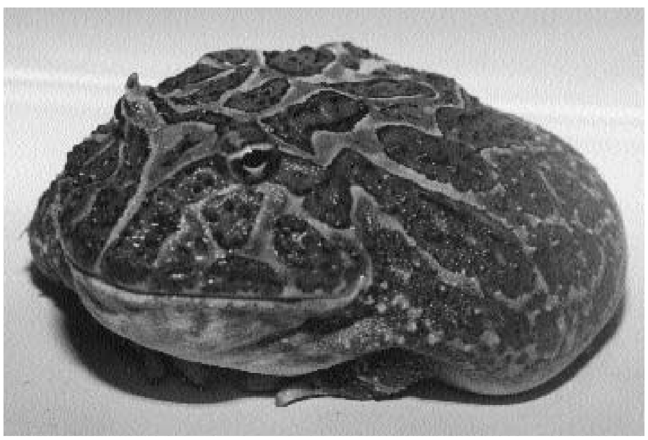


FIGURE 1.2. Horned frog, female, *Ceratophrys cnwelli*.

FIGURE 1.3. Horned frog, *Ceratophrys varia*.

FIGURE 1.4. Milk frog, female, *Prynoyas venulosa*.

BIOLOGY

Taxonomy

South American amphibian fauna is rich in anurans, but relatively poor in Gymnophionas and Urodelas, and only one species of the family Pletodontidae, ***Bolitoglossa altamazonica***, has been described. This small South American amphibian has been little studied and is rarely seen because it lives in bromeliads on the tops of trees in the Amazon jungle, the Zona da Mata, Minas Gerais, in southeastern Brazil, and Vale do Rio Dolce. There are no records of ***Bolitoglossas*** in captivity.

Gymnophionas

DISTRIBUTION AND HABITAT Known in Brazil as "blind snakes" or "two-headed snakes;" these animals are unjustly feared by the rural population. Gymnophionas are divided into two groups; terrestrial and aquatic.

Terrestrial Gymnophionas from the Caeciliidae family are divided into two principal genera. The genus ***Siphonops*** is composed of several species, all much alike in size and color. They are amphibians with a worm form and are fossorial, nocturnal, and completely terrestrial. They grow up to 40 cm long and live in the Cerrado and the Atlantic forests all over the continent. In Brazil, most species are blue-gray with white rings (***Siphonops anullatus*** and *S.* ***paoloensis***). The second genus, ***Pseudosiphonops***, is Amazonian, with a biology identical to the genus described previously. These also have a worm form and small size, about 26 cm, but are brown with light rings.

HOUSING Terrariums for these animals should be made of glass except for a plastic web cover (for aeration).

The substrate should be composed of 30% damp humus, 40% damp sphagnum moss, and 30% dry leaves. It's advisable to spray water into the enclosure every day to maintain the humidity around 70%.

FEEDING This group feeds on terrestrial worms, Coleoptera larvae, flatworms, ant eggs, pupas, and termites. They have a protractile tentacle located between the vestigial ocular region and the nasal area, which is used for orientation, to search for food (through smelling chemical substances), or to find a mate. In captivity these animals accept worms, insects, or even beef.

REPRODUCTION Gymnophiona species are easy to breed in captivity. Eggs are transparent, joined by connective tissue. Eggs hatch within 2 weeks and hatchlings are cared for by the mother, who acquires a whitish color resulting from the secretion of a substance that is attractive to the newborn, enticing them to remain interlaced on the mother's body for approximately 10 days. When the color of the adult female returns to its original hue, the young, now evidently larger, leave the mother and begin an independent life.

The aquatic group of Gymnophionas includes species from the Typhlonectidae family, genera ***Typhlonectes***, ***Chthonerpeton***, and ***Potomotyphlus***. They have been poorly studied and are little known. They, too, are of worm form, but are exclusively aquatic. They show no external branchias, but have internal ones. This species develops totally in water. The members of this family in South America are found in Brazil, Uruguay, Venezuela, and Argentina. Reproduction is viviparous or oviparous. Their natural diet is composed of small aquatic invertebrates (crustaceans, insects, worms, and mollusks).

Anurans

DISTRIBUTION AND HABITAT Anurans exhibit the greatest diversity among South American amphibians. The most important families and genera will be discussed. The anurans live in diverse environments: underground, in the tops of trees, in water and on the forest floor. In Brazil, anurans are found in all ecosystems: restingas, caatingas, Atlantic forests, Amazon forests, Cerrado, and cultivated fields. Most species depend on water to survive.

TAXONOMY

- Family Hylidae (tree frogs),Genera: ***Hyla***, ***Phrynoyas***, ***Trachycephalus***, ***Osteocephalus***, ***Sphaenorhyncus***, ***Gastrotheca***, ***Phyllomedusa***, among others—Tree frogs are typical representatives of arboreal habitats. The ventouses on the tips of their toes enable them to cling to trunks and branches. Their preferred habitat is forested regions, and their biology is totally related to water. The great majority are nocturnal. They feed on small arthropods. Larger species of ***Hyla***, ***Phrynoyas***, ***Trachycephalus***, and ***Osteocephalus*** genera are able to feed on small vertebrates. A rare event of fruit-eating behavior of ***Hyla truncata***, a small species living in a terrestrial bromeliad, was recorded from the Maricá's Restinga, in the state of Rio de Janeiro.
- Family Bufonidae (toads),Genera: ***Bufo***, ***Melanophryniscus***, ***Ranphophyrne***, among others—***Bufo*** species are terrestrial animals with biology little related to water (using it only for reproduction). This family consists of animals ranging in size from the small ***Melanophryniscus*** that live in cold and dry highland regions to the giant ***Bufos*** from the hot and humid regions of northern and northeastern Brazil. They have glands all over their bodies that secrete toxins, but the most important poison producers are the parathyroids, located behind the eyes. Species of this family are widespread throughout South America and frequently figure in regional folklore.

  Seven species of ***Bufo*** occur in Brazil; ***Bufo arenarum*** ssp (southeast, south and northeast), ***B. crucifer*** (southeast), ***B. ictericus*** (south and southeast); ***B. paracnemis*** (southeast, northeast and central west), ***B. typhonius*** (north), ***B. pigmaeus*** (southeast), and ***B. marinus*** (north).
- Family Pipidae (Pipa toad),Genus: ***Pipa***––Species of this genus are exclusively aquatic and have no eyelids or tongue. They have gustatory papilla on the tips of the front toes. Two of the most important Brazilian species are ***Pipa pipa*** (north) and ***Pipa carvalhoi*** (northeast).
- Family Microhylidae (wood toads),Genera: ***Elachistocleis; Dermatonotus; Arcovomer; Zacaenus*** and ***Stereocyclops***, among others. These species are small to medium anurans with a round body shape and short legs. They live primarily in subterraneous habitats, the great majority living under the substratum of cerrado, fields and woods. They are inactive during the day, and are awakened by rain at night to feed and mate.

  This is a numerous and interesting family. In Brazil they are found in all ecosystems, including the littoral where the substratum is sandy with a high percentage of salt.
- Family Dendrobatidae (poison frogs), Genera: ***Dendrobates***, ***Epipedobates***, and ***Phylobates***, among others—These frogs occur in the Amazon tropical forests. These graceful, small, brightly colored frogs have adherent ventouses on the tips of their toes. They live on rock formations or bushes in the forest.

Some species live near water or exclusively in bromeliads. They usually feed on ants and termites and seem not to have any predators. These species do not tolerate low temperatures; the ideal diurnal temperature is around 26°C, reduced to 22°C at night.

Members of this family excrete extremely poisonous cutaneous toxins and should be handled carefully, preferably with disposable gloves.

- Family Leptodactylidae (common frogs and horned frogs), Genera: ***Ceratophrys***, ***Crossodactylus***, ***Hylodes***, ***Toropa***, ***Odontophrynus***, ***Eleuterodactylus***, among others—Some species are diurnal, some nocturnal. They are called “false frogs” because of the absence of interdigital membranes. The biology of most species is associated with water, nevertheless there are exceptions, such as members of the genus ***Eleuterodactylus***, which have terrestrial development. Females of this genus dig a small cavity in the forest floor, where, after amplexus with the male, they lay a small number of transparent eggs that already contain embryos. Within two weeks the embryos are totally developed and emerge from the incubation chamber in the ground as small but completely formed frogs.
- Family Pseudidae (water frogs), Genus: ***Pseudis***—Common in southern Brazil, Uruguay, and Argentina, these little frogs are aquatic with well-developed interdigital membranes in the back feet that enable them to be excellent swimmers. Oddly, in this genus, although adult frogs barely reach 6 cm in length, the tadpole is the largest of all South American species. The most common species are ***Pseudis minuta*** (Uruguay) and ***Pseudis paradoxa*** (southeastern Brazil).

Currently, there are no known South American scientific works reporting studies of the life of amphibians in the wild or of amphibian conservation projects by South American zoological institutions. On the other hand, taxonomic studies and biogeographic studies have been conducted by the herpetology sections of important South American museums.

MANAGEMENT IN CAPTIVITY

HOUSING Robust species such as ***Bufo ictericus***, ***Bufo paracnemis***, and the large horned frogs ***Ceratophrys ornata***, ***Ceratophrys cranwelli***, and ***Ceratophrys varia***, require wide, well-ventilated cages or terrariums with lateral ventilation. Webbing walls or covers must be kept from direct contact with the animals. In particular, recently captured individuals are easily stressed and in their eagerness to escape may traumatize the nostrils or legs. Webbing should be placed only in the upper part of the side wall, and the lower part should be of glass, which is not abrasive and more suitable for animals while adapting.

The substrate should be nonabrasive. The ideal substrate for ***Ceratophrys*** and ***Bufo*** spp. is a composition of humus, not too damp (40% humidity), sphagnum moss (25% humidity), and dried leaves. A substrate made with equal parts of these items is excellent for these large, heavy anurans. The substrate should be at least 10 cm deep to allow the animals to bury themselves. The large amount of feces and urine produced necessitate changing the substrate every 10 days.

Tree trunks and artificial plants may provide decorative elements. All material used for captive amphibians should be processed in an autoclave to eliminate contamination.

A large, easily cleaned water container should be provided and cleaned often. Frogs may spend several days in the water during the molting process.

It is necessary to avoid cleaning products that leave toxic residues. Such residues may be fatal to these animals, because they are highly susceptible to chemical toxins. An ideal disinfectant is a light solution of povidone iodine (2%).

It may be advisable to spray warm water (25–27°C) over the animals once a day, making sure not to wet the enclosure to the point of transforming it into a swamp. Excess humidity may cause morbidity in big anurans. These animals cannot tolerate low temperatures. It is recommended that an even temperature of 24–26°C be maintained in the terrarium. The temperature may be reduced at night to 20–22°C.

Totally aquatic species such as ***Pipa carvalhoi*** and ***P. pipa*** should be kept in a tropical aquarium with water at a pH of 6.4 to 6.8 and a temperature of approximately 28°C. They prefer aquariums with many plants and low to moderate illumination. The aquarium should be covered with a plastic web for oxygenation and to prevent escapes. In a dry environment, aquatic anurans dehydrate rapidly and may die within a few hours.

Arboreal species such as those in the family Hylidae (genera ***Phyllomedusa***, ***Hyla***, ***Trachycephalus***, ***Osteocephalus***, ***and Phrynoyas***, among others) should receive care in captivity that approximates conditions in their natural habitat. They need large, tall cages built of wood or metal, completely lined with plastic webbing that is installed loosely to prevent abrasive accidents. Bushy vegetation with large leaves should be placed in the cage, along with a large water container made of plastic, if possible, for hydration, copulation, and oviposition.

The Dendrobatidae require a large, ventilated terrarium planted with spineless bromeliads. In Sao Paulo, a layer of fern trunks is placed on the bottom of the terrarium, into which bromeliads and small epiphytes are fixed. A shallow water container (about 3 cm deep) is sufficient for these small species (they barely reach 4 cm) to bathe. The substrate should be composed of one part chopped flowers, two parts humus (25% moisture), and one part sphagnum moss (25% humidity). Tree trunks and rocks finish the decoration. The terrarium must have artificial cold illumination during the daily photoperiod (a minimum of 8 hours), since the majority of these species are diurnal.

Ceratophrys tadpoles are voracious predators and must be kept isolated, because they are cannibalistic. They should be kept in small individual containers with the water temperature never below 24°C. Recently metamorphosed young frogs should continue to be kept separated in small cages with the same structures as those for adults.

FEEDING ***Bufo*** species and large horned frogs feed on live prey. Zoos or other institutions that intend to work with this group of animals should maintain breeding colonies of mice, crickets, and cockroaches that may be offered to the animals according to their size and age. ***Ceratophrys*** and ***Bufos*** usually have great appetites and should be fed once a week (twice a week at the most to prevent obesity).

Bufo tadpoles are vegetarian, scraping algae from stones and other submersed surfaces in lakes and ponds. These tadpoles may be kept in large groups and fed "green water" (algal water) rich in phytoplankton and palatable microorganisms. They may be offered algae and aquatic plants from an aquarium. Some species accept a special ornamental fish ration. Spirulina algae may also be offered. They grow rapidly, gaining weight daily. Metamorphosis takes place within three months, and the small toads may then be fed with crickets, cockroaches, and fruit flies. As they grow, larger prey may be offered. Young ***Bufos*** may live in colonies, but should be separated according to size to prevent cannibalism.

Ceratophrys tadpoles are voracious. They should be fed daily with small fish or tadpoles of ***Rana catesbiana***. They also accept earthworms or tubifex worms. Young horned frogs should be fed with crickets, cockroaches, and newborn mice, sprinkled with calcium and powdered vitamin complex to ensure good growth.

Members of the Pipidae family have vestigial vision and gustatory papilla on their front toes and taste food by touch. They have no tongue, so food or prey is captured with the sensorial legs and carried to the mouth. Adult amphibians may be fed small live fish, earthworms, tubifex worms, a commercial fish diet, and even small pieces of fresh beef. They are hardy and undemanding. Feeding of tadpoles is discussed in the reproduction section, because there is species variation in their requirements. Specimens of different sizes should not be kept in the same aquarium because cannibalism is common among the Pipidae.

Arboreal species, such as members of the Hylidae family, should be fed once a week with newborn mice and mealworm larvae offered in pots in the middle of the vegetation next to the animals' shelter. Crickets and cockroaches should be released into the terrarium weekly, the amount depending on the number and voracity of the frogs.

Tadpoles of the Dendrobatidae should be fed with algal water and infusions. Newly metamorphosed frogs may be kept in a colony, and be fed with small insects (crickets, cockroaches, ants, termites, and fruit flies).

REPRODUCTION

- Leptodactylidae—***Ceratophrys*** females are bigger than the males. Breeding males vocalize a lot and show great strength in the front legs, preparatory to amplexus (clasping the females). Both in ***Bufos*** and ***Ceratophrys***, coitus takes place in the water. When the male practices inguinal amplexus, the female releases an enormous number of ovules that are immediately fertilized by the active male. After copulation, the spawn should be transferred to a separate tank of water with the same conditions as the breeding tank. This water should be well aerated using an aquarium compressor connected to an aerator. Within six or seven days, the developed tadpoles hatch.
- Pipidae—Pipidae reproduce well in captivity. Males and females are similar to each other in appearance. Sex may be determined by the following characteristics: females are thinner than males and voiceless. The robust males have well-developed front legs to accomplish inguinal amplexus. In addition, males have a strong and vibrating voice that may heard several meters away from the aquarium during the reproductive period (from September to March in Brazil). After amplexus, each female (of both species) deposits hundreds of fertilized eggs over its own back. The female moves away from the male, and gradually the eggs are absorbed by a special cutaneous tissue that encloses them completely. Incubation lasts approximately 60 days. At this point, the two species differ greatly in the reproductive process.

  Pipa carvalhoi are small frogs (8–12 cm, males barely smaller than females). They live in northeastern Brazil, in the states of Bahia and Pernambuco. After 60 days in the chamber on the back of their mother, tadpoles begin to emerge through the pores, tail first. They complete metamorphosis in 4 months. They can be raised in colonies because they are filter feeders, and cannibalism is not a problem among the newborn. However, tadpoles must be separated from their mother as soon as they emerge, because adult frogs are extremely voracious cannibals.

Initially, tadpoles should be fed with an infusion of artemia, brine shrimp. As soon as they are large enough, they will accept a flake ration used for ornamental fish. When metamorphosis is complete, they may be fed tubifex worms, scrapings from beef, or a commercial ration. Later they may be fed an adult ration adapted to the size of the young ***Pipas***.

Pipa pipa are larger than ***P. carvalhoi***. They grow as long as 30–40 cm in length with the legs fully stretched. It is found in northern Brazil. After 60–90 days of incubation, completely metamorphosed frogs emerge through the pores of the female's back. It is one of the most bizarre scenes ever seen in aquariums; an enormous frog standing still on the bottom of the tank as hundreds of tiny frogs emerge, which must be removed immediately, because their voracious parents will eat them. After they are separated, the progeny will accept mosquito larvae, tubifex worms, scrapings from beef, and a commercial ration for ornamental fish. Because they are from the Amazon region, the water temperature of their aquarium should not drop below 24°C or they will die. ***Pipas*** may live as long as 15 years in captivity.

- Hylidae—Reproduction occurs from November to April. Installation of an artificial rain system will stimulate copulation. Ovipositing will take place inside the water container of the cage. When ovipositing is complete, the water container should be removed and placed in a clean cage kept at about 22°C. The water should be oxygenated with an aquarium compressor connected to an aerator. Tadpoles of the Hylidae family have various eating habits. Some are filter feeders (***Phyllomedusa***); some are vegetarian (***Hyla***, ***Trachycephalus***, ***and Osteocephalus***); and others rasp algae (***Phrynoyas and Sphaenorhyncus***). All species of this family are tropical and cannot tolerate temperatures below 22°C.
- Dendrobatidae—To facilitate ovipositing, small plastic plates with a layer of water 3 cm deep are placed on the bottom of the terrarium. Small ceramic vases with side entrances for the small frogs are placed on the plates. Amplexus is performed inside the ceramic chamber, as is egg laying. The plates (with the vases) should be removed to an oxygenated container. Hatching occurs within 48 hours. Metamorphosis is complete in 3 months.

REFERENCES

1. Breen, J.F. 1974. Encyclopedia of Reptiles and Amphibians. Neptune City, New Jersey, T.F.H. Publications.
2. Capula, M. 1989. Guide to the Reptiles and Amphibians of the World. New York, Simon and Shuster.
3. Dauner, E. 1982. El Terrerio: Construccíon, Mantenimiento, Cría u Reproduccíon de los Animales. Barcelona, Spain, Editorial De Vecchi.
4. Heselhaus, R.; and Schimidt, M. 1994. Harlequin Frogs: A Complete Guide. Neptune City, New Jersey, T.F.H. Publications.
5. Hunziker, R. 1994. Horned Frogs. Neptune City, New Jersey, T.F.H. Publications.
6. Matz, G.; and Vanderhaege, M. 1979. Guia del Terrario. Barcelona, Spain, Omega.
7. Obst, F.J.; Richter, K.; and Jacob, U. 1998. The Completely Illustrated Atlas of Reptiles and Amphibians from the Terrarium. Neptune City, New Jersey, T.F.H. Publications.
8. Wall, J.G. 1994. Jewels of the Rainforest—Poison Frogs of the Family Dendrobatidae. Neptune City, New Jersey, T.F.H. Publications.
9. Zimmermann, E. 1995. Reptiles and Amphibians. Neptune City, New Jersey, T.F.H. Publications.

2
Class Reptilia, Order Crocodilia (Crocodilians): Caimans, Crocodiles

BIOLOGY AND CAPTIVE MANAGEMENT

Flavio de Barros Molina

INTRODUCTION

Living crocodilians, order Crocodilia, comprise 22 species included in eight genera and three families: Alligatoridae (*Alligator, Caiman, Paleosuchus,* and *Melanosuchus*), Crocodilidae (*Crocodylus, Osteolaemus,* and *Tomistoma*), and Gavialidae (*Gavialis*). There are 10 species/subspecies of crocodilians in South America (Table 2.1) and according to the International Union for the Conservation of Nature (IUCN) three of these species are considered threatened (critically endangered, endangered, or vulnerable)[16] (see Table 2.1). *Caiman latirostris* is not on this list but is considered threatened in Brazil.[3]

TABLE 2.1. Distribution of South American crocodilians by country with IUCN status

Scientific Name	Distribution	IUCN
Caiman crocodilus apaporiensis	CO	
Caiman crocodilus crocodilus	PE, EC, VE, CO, GU, FG, SU, BR, TR	
Caiman crocodilus fuscus	MX, CA, CO, EC, VE	
Caiman crocodilus yacare	BR, BO, AR, UR?, PA	
Caiman latirostris	AR, BO, BR, PA, UR	
Melanosuchus niger	BO, EC, PE, CO, BR, FG, GU	EN
Paleosuchus palpebrosus	BO, PE, EC, CO, VE, GU, SU, FG, BR, PA	
Paleosuchus trigonatus	BO, PE, CO, VE, GU, SU, BR	
Crocodylus acutus	US, MX, CA, EC, PE, VE, CO, TR	VU
Crocodylus intermedius	VE, CO	CR

Source: See references 5, 14, 16, 31, and 33. AR, Argentine; BO, Bolivia; BR, Brazil; CA, Central America; CO, Colombia; EC, Ecuador; FG, French Guyana; GU, Guyana; MX, Mexico; PA, Paraguay; PE, Peru; SU, Suriname; TR, Trinidad; UR, Uruguay; US, United States, VE, Venezuela; ?, unconfirmed distribution; CR, critically endangered; EN, endangered; VU,vulnerable.

The objective of this chapter is to discuss management techniques developed for breeding South American species. Excellent reviews of these and other related topics can be found in the references.[10,30,36]

BIOLOGY

Crocodilians may be found in various ecosystems, especially in the tropics. They live in close association with aquatic habitats, such as rivers, ponds, swamps, and estuaries, and usually spend part of the day in the water, and part on land. *Crocodylus porosus* is adapted to saltwater in northern Australia and southeast Asia.

As ectothermic vertebrates, they rely on an external heat source to control body temperature. Basking behavior is a key factor in the thermoregulation process. Crocodilians spend most of the day basking on land (atmospheric basking). To maintain their body temperature within the appropriate range they frequently move from land to water (and vice versa). Water can be used as a heat source (as during aquatic basking) or a heat sink. During atmospheric and aquatic basking they change position many times exposing different body parts to an environmental heat source. According to Lang[18] and Mazzotti[24] heat exchange is greatly influenced by solar radiation and conduction in the water. Thermal behavior and body temperature are affected by internal (e.g., nutritional status, age, size, infection, and reproductive state) and external factors (e.g., climatic conditions and social interactions).[18] Thermal selection varies among the species of crocodilians [24], and little is known about it for most of the species. Preferred body temperature seems to be between 31.3 and 32.5°C for *Crocodylus johnstoni,*[17] 30 and 33°C for *Alligator mississippiensis,*[18] 28.5 and 36.2°C for *Caiman crocodilus,*[7] and 28 and 34.5°C for *Caiman latirostris.*[26]

Crocodilians are opportunistic carnivores, eating a variety of prey items, including insects, mollusks, fish, frogs, reptiles, birds, and mammals. They have sharp conical teeth well adapted to puncturing and tearing prey. Most species are nocturnal hunters.[28] Crocodilian diets vary with habitat, size, and age. Hatchlings eat insects, tadpoles, frogs, snails, crabs, shrimp, and small fishes. Subadults and adults eat mainly fishes, crabs, reptiles, birds, and mammals.[28] They are ambush predators and, according to Pooley,[28] may show an advanced social feeding behavior. This can be seen in a group of crocodiles cooperatively snapping at a school of fish or feeding on a large mammal carcass.

Crocodilians differ from all other reptiles by showing a complex social behavior. They communicate with sounds, postures, motions, odors, and touch.[19] As explained by Lang,[19] communication starts in the egg and continues throughout life. Embryos near hatching time possibly may vocalize to synchronize the hatching of the clutch. Hatchlings and young may vocalize to maintain group cohesion or, in a dangerous situation, to alert others and attract the attention of adults. Typically, adults react by attacking the possible predator.

Species-specific social signals and displays help to define and maintain dominance hierarchy in the group.[19] Dominant individuals have large body size and are aggressive.[19,22] They control access to mates, nest sites, food, and living space.[19] Subadult males and small females have low-ranking positions in hierarchies. Dominance hierarchies in females are usually related to nest-site access.[19] Crocodilians are territorial. Dominant males exclude other males from their territories.[19] Females may defend their own territories or just their nest sites. Territorial behaviors may be seen year round or only during the reproductive season, depending on the species and locality.

Sexual dimorphism in South American caimans and crocodiles is related to size, with males attaining a larger size than females (see Table 2.2). Mating behavior of

TABLE 2.2. Maximum adult size and reproductive parameters of South American crocodilians[10,14,31,33]

Scientific Name	Maximum Adult Size (m)	Nest Characteristic	Eggs per Clutch
Caiman crocodilus apaporiensis	2.09?	Mound nest	20–50 eggs
Caiman crocodilus crocodilus	2.5	Mound nest	14–40 eggs
Caiman crocodilus fuscus	2.0[a]	Mound nest	15–30 eggs
Caiman crocodilus yacare	3.0	Mound nest	20–40 eggs
Caiman latirostris	3.5	Mound nest	20–60 eggs
Melanosuchus niger	6.0	Mound nest	18–75 eggs
Paleosuchus palpebrosus	1.5	Mound nest	12–25 eggs
Paleosuchus trigonatus	1.7	Mound nest	10–17 eggs
Crocodylus acutus	6.0	Hole or mound nest	19–81 eggs
Crocodylus intermedius	6.0	Hole nest	15–70 eggs

Source: See references 10, 14, 31, 33. ?, largest specimen reported.[5] [a]Average size.

Caiman crocodilus crocodilus (in the llanos of Venezuela) and *Caiman crocodilus yacare* (in the pantanal of Brazil) occurs at the end of the dry season.[22] In both localities the species nest during the flooding season; the breeding season seems to be related to rainfall cycles and river levels.[22] *Melanosuchus niger, Paleosuchus palpebrosus,* and *Paleosuchus trigonatus* seem to nest at the end of the dry season and the beginning of the rainy season; *Caiman latirostris* nests in the Southern Hemisphere spring.[22] *Crocodylus acutus* nests during the northern spring, and *Crocodylus intermedius* during the northern winter.[33]

All members of the Alligatoridae family make mound nests; many members of the Crocodilidae family make hole nests[22] (see Table 2.2). *Paleosuchus trigonatus* often nests beside termite mounds.[21] Clutch size is related to the size of the female, usually varying from 14 to 60 eggs per clutch in *Caiman* species and subspecies, from 10 to 25 eggs per clutch in *Paleosuchus* species, from 18 to 75 eggs per clutch in *Melanosuchus niger,* from 19 to 81 eggs per clutch in *Crocodylus acutus,* and from 15 to 70 eggs per clutch in *Crocodylus intermedius*[10,14,31,33] (see Table 2.2).

The eggs of *Caiman crocodilus, Paleosuchus palpebrosus,* and *Paleosuchus trigonatus* generally develop and hatch when incubated between 28 and 32°C.[22] Incubation time is variable, usually between 42 and 100 days.[14,21,33] Sex determination in crocodilians is temperature dependent.[20] Only females are produced at low temperatures, mostly females (or only females) are produced at high temperatures, and varying proportions of males and females are produced at intermediate temperatures.[20]

Many species of crocodilians show maternal care. Females may defend their nest against predators, they may open the nests and carry the hatchlings to water, and they may protect the young for weeks or months.[19,22] At least in captivity, males of some species also show these behaviors.[22]

MANAGEMENT IN CAPTIVITY

Crocodilians show no external sexual dimorphism. Sexing is possible by cloacal probing.[4] They may be marked by making a hole in the scales of the single caudal crest following a predetermined code. The use of natural marks, especially those at the jaws and tail, is another option for identification. Measuring should be done according to Medem's recommendations.[25]

The capture and restraint of crocodilians may be dangerous for both human and reptile. It must be done carefully and only if really necessary. Such a stressful situation can cause the death of a crocodilian, as a result of lactic acid production. Wise[37] presented techniques for the physical restraint of crocodilians.

The minimum area of the exhibit and other specifications may be determined by national law, as in Brazil.[15] Crocodilians usually may be kept in groups, depending on the area available and the number, size, and sex of individuals to be enclosed. As hierarchic and territorial reptiles, they may exhibit aggressive behavior, especially if both adult males and females are maintained in the group. The introduction of a new adult specimen into an already formed group is always a problem, because the newly introduced animal will challenge its new companions or will be challenged by them. Usually there is no problem when young caimans are introduced, even into a group of adults, but the best choice, whenever possible, is to form a group of young crocodilians and allow them to grow up together. This option seems to minimize antagonistic interactions.

It is important to understand the hierarchy of a captive group. Much can be learned by observing crocodilian behavior. Individuals basking in an unusual place may be individuals that have been excluded by dominant specimens. Interspecific aggressions have been observed at São Paulo Zoo, Brazil (*Paleosuchus palpebrosus* with *Caiman* sp. and *Caiman latirostris* with *Caiman crocodilus yacare*), and a few incidents led to the death of some caimans.

The exhibit must have a diversity of environments to improve behavioral performance of caimans and crocodiles, and simulating a natural environment will contribute to public education. A sandy area exposed to the sun is important for basking, and an area planted with trees and bushes offers options for heat sinking. A sandy area is also important for the nesting behavior of crocodile females (hole nesters); a soil area covered by dead leaves and branches is important for the nesting behavior of caiman females (mound nesters) (see Table 2.2). The exhibit must have a pond. The pond depth should depend on the species and size of the individuals involved, but must be deep enough to allow the male to place himself over the female during copulation. The substrate used in the pond must be smooth to avoid damage of the crocodilians' belly and the borders must form soft acclivities that will not make it difficult for them to leave the water. Such aquatic plants as *Eichhornia* and *Pistia* may be placed in the pond.

Air and water temperature depends on the species and the season. Air temperature for *Caiman* spp. maintained in outdoor exhibits at São Paulo Zoo usually varies between 15 and 30°C, especially important for atmospheric basking. Extremes of 5 to 35°C may be registered. Water temperature usually varies between 18 and 25°C, especially important for aquatic basking. Extremes of 13–28°C may be registered. Crocodilians must always have access to adequate microenvironments to thermoregulate. Indoor exhibits should be equipped with heaters to create a thermal gradient.[32] Crocodilians maintained in indoor exhibits, including aquariums, must be periodically exposed to natural unfiltered sunlight or artificial ultraviolet light of appropriate wavelength (ultraviolet B; UVB) for the endogenous synthesis of vitamin D_3.[1,12,13] Gehrmann[13] discusses the light requirements of captive reptiles (including the detrimental effects of prolonged exposure to UVB from sunlamps).

Caimans (*Caiman* spp.; *Paleosuchus* spp.) can be kept together with turtles but problems may occur, probably as a result of overpopulation. At São Paulo Zoo, on a few occasions a caiman (especially *Caiman crocodilus crocodilus*) has attacked a turtle (*Phrynops hilarii* and *Trachemys scripta elegans*). The following procedures are suggested when maintaining a mixed exhibit: (a) avoid overpopulation of any of the species involved, (b) keep all the reptiles well fed, and (c) know and monitor the caimans' hierarchy.

Food usually offered in captivity consists of chicks, mice, fish, and invertebrates. Muscle meat (e.g., beef) is not nutritionally balanced and should be avoided. Chicks and mice are good options. Fresh whole fish (including head, viscera, scales, and bones) are not calcium deficient. An enzyme, thiaminase, is found in some species of fish and becomes active after death. Thiaminase destroys vitamin B_1 and may cause a deficiency.[1,12] Thawed frozen fish should be supplemented with thiamin.[1]

Hatchlings and young caimans readily accept mealworm larvae and adults, crickets, and cockroaches. Mealworms, crickets, and cockroaches must be supplemented with a calcium source,[1,12] (e.g., powdered calcium carbonate,[12]), and mealworm larvae should be offered only sporadically because of its high level of fat[1] (see Table 3-1 in Frye[12]). Frequency of feeding should be determined by the temperature at which crocodilians are kept. During warm months they may be fed twice a week; during cold months they should be fed once a week. Young animals must be fed more frequently.

REPRODUCTION, ARTIFICIAL INCUBATION

To achieve the best results with artificial incubation, the eggs must be removed from the nests within 24 hours of laying.[10] It is advisable to keep the eggs in the same position as they were found in the nest.[10,27,35] After being measured, the eggs must be placed in incubators held at the appropriate temperature and humidity. The average temperature of the egg cavity in crocodilian nests varies from 28 to 32°C; the average humidity varies from 70 to 100%.[10]

The incubation substrate should include nesting material[10,23] or vermiculite.[8,9,27] The origin of the nesting material must be checked to avoid a possible contamination of the eggs. A 1:1 ratio of vermiculite and tap water, by mass, is suggested for use with reptile eggs in general.[27] Closed egg containers must be periodically opened for ventilation.[27] The eggs must be regularly inspected and the development of the opaque banding pattern must be monitored.[10,35] Rotten eggs should be removed. Sometimes hatchlings must be assisted during the hatching process.[23]

Proper methods for incubating reptile eggs are discussed by Packard and Phillips.[27] A simple incubator can be made of a Styrofoam box supplied with common lamps and a thermostat as used at Escola Superior de Agronomia "Luiz de Queiroz"/Universidade de São Paulo (ESALQ/USP) (for *Caiman latirostris* eggs)[34] and São Paulo Zoo (for *Caiman crocodilus crocodilus* eggs).[8,9]

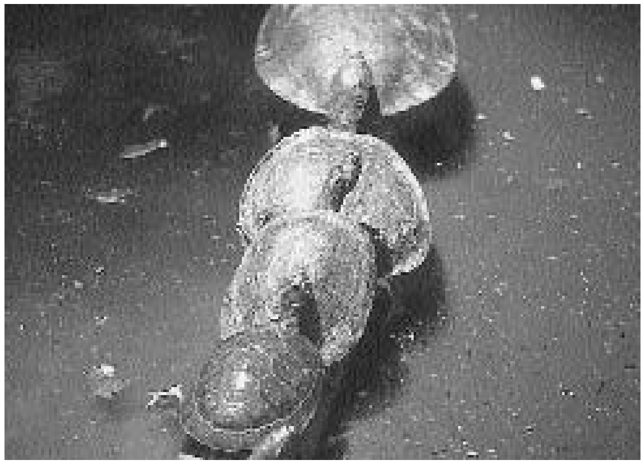
Hatchlings may be identified by photographs or line drawings of the head, particularly the jaws and the tail. They should be measured by Medem's method.[25] Normal hatchlings are usually fed for the first time during their second or third week. They can be kept in small outdoor enclosures or in 1000-L tanks (also maintained outside). At São Paulo Zoo, these tanks were in use for turtles before 1985. They are not expensive and are adapted from the types commonly used for water reservoirs in houses. The internal space may be divided into areas, one-fourth sand and three-fourths water at a depth of 15 cm. Young that are longer than 40 cm total length are best maintained in outdoor enclosures.

REFERENCES

1. Allen, M.E.; and Oftedal, O.T. 1994. The nutrition of carnivorous reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 71–82.
2. Bellairs, A. d'A. 1987. The Crocodilia. In G.J.W. Webb, S.C. Manolis, and P.J. Whitehead, eds., Wildlife Management: Crocodiles and Alligators. Chipping Norton, Australia, Surrey Beatty & Sons, pp. 5–7.
3. Bernardes, A.T.; Machado, A.B.M.; and Rylands, A.B. 1990. Fauna brasileira ameaçada de extinção [Threatened Brazilian fauna]. Belo Horizonte, Fundação Biodiversitas.
4. Brazaitis, P.J. 1968. The determination of sex in living crocodilians. British Journal of Herpetology 4(3):54–58.
5. Brazaitis, P.J. 1973. The identification of living crocodilians. Zoologica 58(3–4):59–101.
6. Buffetaut, E. 1989. Evolution. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 26–41.
7. Diefenbach, C.O.C. 1975. Thermal preferences and thermoregulation in *Caiman crocodilus*. Copeia 1975(3):530–540.

8. Duarte, P.G.; Molina, F.B.; Lula, L.A.B.M.; and Rocha, M.B. 1993. O manejo de jacarés na Fundação Parque Zoológico de São Paulo: Algumas observações sobre a reprodução e o desenvolvimento inicial dos filhotes de jacaretinga, *Caiman crocodilus crocodilus* (Linnaeus, 1758) [Caiman management at São Paulo Zoo: Some observations on the reproduction and initial development of *Caiman crocodilus crocodilus* young]. In L.M. Verdade, I.U. Packer, M.B. Rocha, F.B. Molina, P.G. Duarte, and L.A.B.M. Lula, eds., Anais do III Workshop sobre Conservação e Manejo do Jacaré-de-papo-amarelo (*Caiman latirostris*). Piracicaba, SP, ESALQ/USP. pp. 15–50.
9. Duarte, P.G.; Molina, F.B.; Lula, L.A.B.M.; and Rocha, M.B. 1994. Maintenance of Brazilian crocodilians at São Paulo Zoo. In Proceedings of the 49th Annual Conference of the IUDZG(The World Zoo Organization. São Paulo, Brazil, IUDZG, pp. 7–13.
10. Ferguson, M.W.J. 1985. Reproductive biology and embryology of the crocodilians. In C. Gans, F. Billett, and P.F.A. Maderson, eds., Biology of the Reptilia, Vol. 14. Development A. New York, John Wiley & Sons, pp. 329–491.
11. Fowler, M.E. 1978. Metabolic bone disease. In M.E.Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 55–76.
12. Frye, F.L. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry. Malabar, Florida, Krieger Publishing.
13. Gehrmann, W.H. 1994. Light requirements of captive amphibians and reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 53–59.
14. Groombridge, B. 1987. The distribution and status of world crocodilians. In G.J.W. Webb, S. C. Manolis, and P.J. Whitehead, eds., Wildlife Management: Crocodiles and Alligators. Chipping Norton, Australia, Surrey Beatty & Sons , pp. 9–21.
15. IBAMA. 1989. Legislação sobre zoológicos [Legislation about zoos]. Brasília, Instituto Brasileiro do Meio Ambiente e dos Recursos Naturais Renováveis.
16. IUCN. 1996. 1996 IUCN Red List of Threatened Animals. Gland, Switzerland, IUCN.
17. Johnson, C.R. 1973. Behaviour of the Australian crocodiles, *Crocodylus johnstoni* and *C. porosus*. Zoolological Journal of the Linnaeus Society 52:315–336.
18. Lang, J.W. 1987. Crocodilian thermal selection. In G.J.W. Webb, S.C. Manolis, and P.J. Whitehead, eds., Wildlife Management: Crocodiles and Alligators. Chipping Norton, Australia, Surrey Beatty & Sons, pp. 301–317.
19. Lang, J.W. 1989. Social behavior. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 102–117.
20. Lang, J.W.; and Andrews, H.V. 1994. Temperature-dependent sex determination in crocodilians. Journal of Experimental Zoology 270(1):28–44.
21. Magnusson, W.E. 1989. Termite mounds as nest sites. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, p. 122.
22. Magnusson, W.E.; Vliet, K.A.; Pooley, A.C.; and Whitaker, R. 1989. Reproduction. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 118–135.
23. Marques, E.J.; and Monteiro, E.V.L. 1997. Manejo e criação do *Caiman crocodilus yacare* no Pantanal Mato-grossense [Management and rearing of *Caiman crocodilus yacare* in the Pantanal Mato-grossense]. In C. Valladares-Pádua, R.E. Bodmer, and L. Cullen, Jr., eds., Manejo e conservação de vida silvestre no Brasil. Brasília, CNPq/Sociedade Civil Mamirauá, pp. 95–105.
24. Mazzotti, F.J. 1989. Structure and function. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 42–56.
25. Medem, F. 1976. Recomendaciones respecto a contar el escamado y tomar las dimensiones de nidos, huevos y ejemplares de los Crocodylia y Testudines [Recommendations respecting scale count, and nest, egg, and specimen measurements in Crocodilia and Testudines]. Lozania (20):1–17.
26. Molina, F.B.; and Sajdak, R.A. 1993. Observações sobre a preferência térmica e o comportamento de termorregulação no jacaré-de-papo-amarelo, *Caiman latirostris,* em cativeiro: Variações ontogenéticas e algumas comparações com outras espécies de jacarés neotropicais [Observations on the thermal preference and thermoregulation behavior of the Broad-snouted caiman, *Caiman latirostris,* in captivity: Ontogenetic variations and some comparisons with other species of neotropical caimans]. In L.M. Verdade, I.U. Packer, M.B. Rocha, F.B. Molina, P.G. Duarte, and L.A.B.M. Lula, eds., Anais do III Workshop sobre Conservação e Manejo do Jacaré-de-papo-amarelo (*Caiman latirostris*). Piracicaba, SP, ESALQ/USP, pp. 93–132.
27. Packard, G.C.; and Phillips, J.A. 1994. The importance of the physical environment for the incubation of reptilian eggs. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 195–208.
28. Pooley, A.C. 1989. Food and feeding habits. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 76–91.
29. Pough, F.H.; Heiser, J.B.; and McFarland, W.N. 1996. Vertebrate Life, 4th Ed. Prentice-Hall.
30. Ross, C.A. Ed. 1989. Crocodiles and Alligators. New York, Facts On File.
31. Ross, C.A.; and Magnusson, W.E. 1989. Living crocodilians. In C.A. Ross, ed., Crocodiles and Alligators. New York, Facts On File, pp. 58–73.
32. Sajdak, R.A.; Molina, F.B.; Jansen, R.; and Abransom, C. 1993. Behavioral thermoregulation in Broad snout caiman *Caiman latirostris* in captivity with comments on behavioral enrichment. In Proceedings of the Great Lakes Regional Conference of the American Association of Zoological Parks and Aquariums (AAZPA). Duluth, Minnesota, AAZPA, pp. 512–519.

33. Steel, R. 1989. Crocodiles. London, Christopher Helm.
34. Verdade, L.M.; Michelotti, F.; Rangel, M.C.; Cullen, L., Jr.; Ernandes, M.M.; and Lavorenti, A. 1992. Manejo dos ovos de jacaré-de-papo-amarelo (*Caiman latirostris*) no CIZBAS/ESALQ/USP [Management of broad-snouted caiman eggs at CIZBAS/ESALQ/USP]. In L.M. Verdade and A. Lavorenti, eds., Anais do II Workshop sobre Conservação e Manejo do Jacaré-de-papo-amarelo (*Caiman latirostris*). Piracicaba, SP, ESALQ/USP, pp. 92–99.
35. Webb, G.J.W.; Manolis, S.C.; Dempsey, K.E.; and Whitehead, P.J. 1987a. Crocodilian eggs: A functional overview. In G.J.W. Webb, S.C. Manolis, and P.J. Whitehead, eds., Wildlife Management: Crocodiles and Alligators. Chipping Norton, Australia, Surrey Beatty & Sons, pp. 417–422.
36. Webb, G.J.W.; Manolis, S.C.; and Whitehead, P.J. Eds. 1987b. Wildlife Management: Crocodiles and Alligators. Chipping Norton, Australia, Surrey Beatty & Sons.
37. Wise, M. 1994. Techniques for the capture and restraint of captive crocodilians. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 401–405.



3 Class Reptilia, Order Chelonia (Testudinata) (Chelonians): Turtles, Tortoises

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INTRODUCTION

Turtles are easily distinguishable from all other reptiles by their characteristic morphology: a body encased by a bony shell covered with horny scutes of epidermic origin. The shell consists of the dorsal carapace and the ventral plastron, firmly fused together in most species. Neural and costal bony plates of the carapace are fused to the neural arches of some vertebrae and to the ribs, respectively. Shell morphology reflects turtle ecology, with tortoises usually having a domed carapace. Detailed discussions of turtle anatomy and physiology may be found in the references.[1,11,49]

About 250 living species of turtles are known worldwide.[49] In South America there are 40 species of freshwater turtles, 4 species of tortoises (Table 3.1), and 7 species of marine turtles.[28] According to the International Union for the Conservation of Nature (IUCN), 22 of these species are considered to be threatened (critically endangered, endangered, or vulnerable).[27] Natural populations of Amazonian species in the *Podocnemis* genus are being protected by the efforts of local conservation projects being developed in Brazil[8] and neighboring countries[31,47] and apparently are recovering. Conservation projects are also important in the protection of marine turtle nesting beaches in Brazil[33] and other South American countries.[51]

BIOLOGY, MANAGEMENT, AND FREE-LIVING POPULATIONS

Flavio de Barros Molina

Feeding Habits and Behavior

Chelidae species are predominantly carnivorous, eating a variety of prey items, including crustaceans, insects, and fish.[12,16,35,43,50,52,56,57] *Chelus fimbriatus* is completely carnivorous, and some species may be highly omnivorous, e.g., *Phrynops rufipes* and a northern population of *Phrynops geoffroanus*.[16,32]

Species in the family Pelomedusidae are primarily herbivorous, eating mostly aquatic plants and fallen fruit,[3,12,16,48,52] but they also consume some mollusks, crustaceans, insects, and fish.[48,52] In captivity, some species will take meat and fish.[12,52]

TABLE 3.1. Distribution of South American freshwater turtles and tortoises. IUCN status of threatened and near threatened species

Scientific Name	Distribution	IUCN
Acanthochelys chacoensis	AR	VU
Acanthochelys macrocephala	BR/PA/BO	NT
Acanthochelys pallidipectoris	AR/PA/BO?	VU
Acanthochelys radiolata	BR	NT
Acanthochelys spixii	BR/UR/AR/PA?	NT
Chelus fimbriatus	BR/GU/FG/SU?/VE/CO/EC/PE/BO/TR	
Hydromedusa maximiliani	BR	VU
Hydromedusa tectifera	BR/UR/AR/PA?	
Phrynops dahli	CO	CR
Phrynops geoffroanus	BR/GU/VE/CO/EC/PE/BO/PA/AR?	
Phrynops gibbus	BR/GU/FG/SU/VE/CO/EC/PE/TR	
Phrynops hilarii	BR/UR/AR/PA?/BO?	
Phrynops hogei	BR	EN
Phrynops nasutus	FG/SU/BR?	
Phrynops raniceps	BR/VE/CO/PE/BO	
Phrynops rufipes	BR/CO/PE?	NT
Phrynops tuberculatus	BR	
Phrynops vanderhaegei	BR/PA/AR/UR?/BO?	NT
Phrynops williamsi	BR/UR/AR/PA	
Phrynops zuliae	VE	VU
Platemys platycephala	BR/GU/FG/SU/VE/CO/EC/PE/BO	
Peltocephalus dumerilianus	BR/FG/VE/CO/EC/PE	VU
Podocnemis erythrocephala	BR/VE/CO	VU
Podocnemis expansa	BR/GU/VE/CO/EC/PE/BO/TR	CD
Podocnemis lewiana	CO	EN
Podocnemis sextuberculata	BR/CO/PE	VU
Podocnemis unifilis	BR/GU/FG/SU/VE/CO/EC/PE/BO	VU
Podocnemis vogli	VE/CO	
Rhinoclemmys annulata	CA/CO/EC	NT
Rhinoclemmys diademata	VE	
Rhinoclemmys melanosterna	CA/CO/EC	
Rhinoclemmys nasuta	CO/EC	NT
Rhinoclemmys punctularia	BR/GU/FG/SU/VE/TR	
Trachemys adiuthrix	BR	EN
Trachemys dorbignyi	BR/UR/AR	
Trachemys scripta callirostris	CO/VE	NT
Trachemys scripta chichiriviche	VE	NT
Trachemys scripta venusta	MX/CA/CO	NT
Chelydra serpentina acutirostris	CA/CO/EC	
Kinosternon dunni	CO	VU
Kinosternon leucostomum	MX/CA/CO/EC/PE?	
Kinosternon scorpioides	MX/CA/TR/BR/GU/FG/SU/VE/CO/EC/PE/BO/AR	
Geochelone carbonaria	CA/BR/GU/FG/SU/CO/VE/BO/PA/AR	
Geochelone chilensis	AR/PA	VU
Geochelone denticulata	BR/GU/FG/SU/CO/VE/EC/PE/BO/TR	VU
Geochelone nigra	GI	VU

Source: Information from references 27 and 28. AR, Argentina; BO, Bolivia; BR, Brazil; CA, Central America; CO, Colombia; EC, Ecuador; FG, French Guyana; GI, Galapagos Islands (Ecuador); GU, Guyana; MX, Mexico; PA, Paraguay; PE, Peru; SU, Suriname; TR, Trinidad; UR, Uruguay; VE, Venezuela. ?, unconfirmed distribution. CR, critically endangered; CD, conservation dependent; EN, endangered; NT, near threatened; VU, vulnerable.

Most species in the family Emydidae are opportunistic omnivores, eating plant material and animals (like leaves, fruit, aquatic plants, mollusks, and insects).[12,13,44,52] Exceptions include the herbivorous *Rhinoclemmys annulata*,[12] and the predominantly carnivorous young of *Trachemys scripta*,[12,13] and *Trachemys dorbignyi* (personal observation).

In the Testudinidae family, *Geochelone carbonaria* and *Geochelone denticulata* are opportunistic omnivores, eating plant and animal material (like fruit, leaves, flowers, snails, insects, and carrion).[12,45,52] *Geochelone chilensis* is apparently omnivorous; its diet includes fruit, shrubs, cacti, grasses, and probably carrion.[60] In captivity, all three of these species will take meat and fish.[52,60] *Geochelone nigra* is herbivorous, eating leaves, berries, cacti, and grasses.[12,59]

Kinosternidae species and *Chelydra serpentina* are omnivorous, eating algae, aquatic plants, worms, mollusks, insects, carrion, and diverse vertebrates, including fish, frogs, turtles, and aquatic birds.[12,13,44,52] Molina[35] analyzed the feeding behavior of 14 species of South American turtles and found that most of them located their prey by olfaction. An exception is the matamata turtle, *Chelus fimbriatus,* which is an ambush predator.[35,50,52] Other unique feeding behaviors include spear fishing[25,35,43,50] and consuming surface particulate matter by neustophagia.[54]

Thermoregulation and Thermal Preferences

Chelonians are ectotherms. Basking behaviors are key factors affecting thermoregulation. The most important behaviors performed by freshwater turtles and tortoises during basking activities are moving from water to land (and vice versa) and exposing different body parts to environmental heat. Tortoises move from sunny areas to shady ones (and vice versa), but they may also enter water. Environmental factors affecting basking behaviors include water and air temperatures, light intensity, and wind.[5,58]

Basking may be divided into atmospheric and aquatic basking. Atmospheric basking is important for thermoregulation, elimination of ectoparasites, assisting in the shedding of shell scutes, and for the synthesis of vitamin D_3.[1,5,9,44,58] Aquatic basking is important for thermoregulation.[9] The preferred body temperature of *Trachemys scripta* is 28–29°C, and the species is most active at 25–30°C.[58] Thermal preferences for most species of freshwater turtles and tortoises seem to fall between 23 and 33°C, and the critical thermal maximum is around 39 to 41°C for freshwater species and 43°C for tortoises.[1,13,58] Thermoregulation mechanisms, thermal preferences, and basking behaviors of most South American species are poorly known.

Reproductive Biology and Behavior

Breeding is usually seasonal in reptiles and young hatch during a period of favorable environmental conditions. Photoperiod, temperature, rainfall, and food availability are important environmental factors. Tropical reptiles breed more frequently than temperate ones, their breeding season is usually longer, and in some species reproductive activity is seen throughout the year.[10,11]

Little is known of the mating season and mating behavior of most South American turtles. The mating season varies according to the species.[38,39,40,41,52] For *Podocnemis expansa* mating depends on the water level fluctuation in each geographic locality.[52] Mating behavior may be divided into four phases, i.e., search for female, pursuit of female, precopulation, and copulation. The pattern may be simple, as in *Phrynops geoffroanus,*[38, 39] or elaborate, as in *Phrynops hilarii*[4] and *Trachemys dorbignyi.*[38]

Nesting behavior is more stereotyped than courtship and usually may be divided into five phases: nest site selection, nest excavation, egg laying, nest covering, and return to water.[37,38,40,41] Clutch size varies from 1 large egg in *Platemys platycephala* to more than 100 eggs in *Podocnemis expansa.* Some species may lay more than one clutch per season.[12,52] Eggs may be brittle shelled, as in *Podocnemis unifilis, Phrynops geoffroanus,* and *Rhinoclemmys punctularia,* parchment shelled, as in *Trachemys scripta* and *Trachemys dorbignyi,* or leathery shelled, as in *Podocnemis expansa.*[40] Incubation time is variable, depending on the species involved, temperature, and other environmental conditions, such as the water level in local rivers. It may be short, ranging from 45–60 days in *Podocnemis expansa,*[8,12,52] or lasting for more than 330 days, as in *Phrynops geoffroanus.*[36]

Management

SEXING, MARKING, AND MEASURING Sexual dimorphism is usually evident only in adults. Useful characteristics include tail length and plastron shape. In all species of turtles and tortoises, males have longer and thicker tails with cloacal openings more distally situated, when compared with those of females. The differences are extreme in *Kinosternon scorpioides* and *Podocnemis expansa.* Males of chelid, kinosternid, and, especially, testudinid species usually have a concave (or slightly concave) plastron. Color dimorphism may be seen in *Trachemys scripta* and *Trachemys dorbignyi,* with males showing an ontogenetic melanism.

Chelonians over 2 years of age and adults may be easily marked according to Cagle's method, which consists of making a notch in the marginal scutes of the carapace following a predetermined code.[7] Newborn and young less than 2 years of age have a less-resistant carapace, so hand drawings and black and white photographs of the pattern of shell markings are preferable for identifying each specimen. Photocopies of the plastron may also be useful for identification.[24] These techniques should be repeated yearly as the chelonian grows.

It is important to record the growth rate of young and adults. Carapace length and width, plastron length and width, and shell height should be measured according to Medem.[34] Weight should also be recorded.

HOUSING, ENVIRONMENTAL ENRICHMENT, AND DIET The minimum area of an exhibit and other specifications may be defined by national law, as in Brazil.[26] Chelonians may be kept in mixed groups, and, if so, the exhibit must include diverse environments. Diversity is important to improve the behavioral performance of turtles and tortoises. A simulating natural environment contributes to public education. An area exposed to the sun is important for basking, and an area planted with dense arboreal vegetation offers additional options for thermoregulation. Both types of area are important for the nest site selection activities of females of different species.

A substrate of soil covered with dead leaves is a good choice in the area planted with trees and/or shrubs. Bromeliads, philodendrons, and epiphytic plants may be used to aid in the natural environment simulation. *Trachemys dorbignyi* and *Phrynops geoffroanus* females will never nest in areas planted with dense arboreal vegetation. *Trachemys dorbignyi* females prefer to nest in sandy areas without vegetation, and *Phrynops geoffroanus* females prefer to nest in soil with sparse herbaceous vegetation. *Geochelone carbonaria* females are generalists and prefer soil without vegetation in shaded areas, soil with sparse herbaceous vegetation in open areas, and sandy beaches.

An artificial pond is a fundamental requirement for freshwater species, and even tortoises enjoy entering the water. Pond depth should depend on the species involved, but must be deep enough to allow the male to place himself over the female during copulation. The substrate must be smooth to avoid damaging the plastron, and the banks must allow egress from the water. Aquatic plants, such as *Eichhornia* and *Pistia,* may be placed in the pond. Partially submerged stones or logs offer atmospheric basking.

Air and water temperatures depend on the species and the season. Air temperature may be kept between 20 and 30°C. Higher temperatures (e.g., 35°C) should be avoided, and turtles must always have access to adequate microenvironments to thermoregulate, such as a pond of water or a shaded refuge. Lower temperatures (e.g., 18°C) may be tolerated by some southern species (*Trachemys dorbignyi* and *Phrynops hilarii*), but may be critical for northern species (*Podocnemis* spp., *Rhinoclemmys* spp., *Kinosternon* spp., *Geochelone carbonaria,* and *G. denticulata*). Northern species will do better if kept between approximately 24 and 30°C; southern species will do better if kept between approximately 20 and 26°C. Water temperature may usually be kept between 22 and 28°C. Pelomedusids (but less markedly, *Podocnemis unifilis*) are particularly sensitive to cold water. Some southern species, e.g., *Trachemys dorbignyi* and *Phrynops hilarii,* may tolerate lower temperatures (e.g., 15–18°C). Northern species will do better if water temperature is kept between approximately 24 and 28°C; southern species will do better if water temperature is kept between approximately 22 and 26°C. Obviously, water temperature is more important to freshwater species than to terrestrial ones, the opposite being true for air temperature.

Turtles and tortoises may be maintained in indoor exhibits, including aquariums and terrariums, but must be periodically exposed to natural unfiltered sunlight or artificial ultraviolet light (UV) of appropriate wavelength (UVB) to allow the endogenous synthesis of vitamin D_3.[2,18,23]

Naturally designed exhibits are best to provide environmental enrichment for turtles and tortoises. Edible vegetation planted inside the exhibit may stimulate foraging behavior. It is important to determine that plants are not toxic.[18,19] *Geochelone carbonaria* and *G. denticulata* may spend a great deal of time grazing and are particularly fond of *Paspalum notatum* grass.

Mixed exhibits are an interesting option for environmental education and enrichment. Many species of freshwater turtles may be kept together. At São Paulo Zoo (Brazil), the following species have been housed together: *Trachemys dorbignyi* and *Phrynops geoffroanus; Podocnemis expansa* and *P. unifilis; Trachemys scripta elegans, Phrynops hilarii,* and *P. expansa.* Because of the risk of hybridization, housing closely related species should be avoided (e.g., *Trachemys dorbignyi* and *T. scripta, Phrynops geoffroanus* and *P. hilarii, Geochelone carbonaria* and *G. denticulata*). Aggressive species such as *Chelydra serpentina* should not be housed with vulnerable species. At Belem Zoo (Para/Brazil) *Kinosternon scorpioides,* a somewhat aggressive species, is kept together with *Rhinoclemmys punctularia.*

Turtles may be kept in enclosures containing caimans (*Caiman* spp.), but problems may develop if the exhibit becomes overpopulated.

The following procedures are suggested when maintaining mixed exhibits: avoid overpopulation of any of the species involved, keep all the reptiles well fed, know and monitor the caimans' hierarchy.

Chelonians may be kept with mammals, as well. At São Paulo Zoo mixing the following species was successful: *Trachemys dorbignyi, Phrynops geoffroanus,* and *Myocastor coypus* (nutria); *Geochelone denticulata, G. gigantea,* and *Hystrix africaeaustralis* (porcupine); *Geochelone denticulata, Tupinambis merianae* (Tegu lizard), and *Myrmecophaga tridactyla* (giant anteater).

Animal matter usually offered in captivity consists of meat (e.g., beef), fish, and invertebrates. Meat must be supplemented with a powdered calcium source, such as steamed bone meal or calcium carbonate, and cannot be the base of the diet. A prolonged deficiency of calcium in the diet, as well as an unbalanced calcium/phosphorus ratio, may lead to metabolic bone disease, a syndrome that is extensively reviewed by Fowler.[17]

Fresh whole fish (including head, viscera, scales, and bones) are not calcium deficient. However, it is important to be sure that the spines and scales will not injury the turtle's digestive tract. An enzyme called thiaminase is found in some species of fish and becomes active after the death of the fish, causing hypovitaminosis B_1. Thawed frozen fish should be supplemented with thiamin.[2,18,19]

Freshwater turtles readily accept earthworms, mealworm larvae, and crickets. Mealworm larvae and crickets must be supplemented with calcium,[2,18,19] and mealworm larvae should be offered only sporadically because of its high level of fat[2] (see Table 3-1 in Frye[18]). Allen and Oftedal[2] discuss the nutrition of carnivorous reptiles, and Frye describes the rearing of some invertebrate prey species.[18,19]

Plant matter usually offered in captivity consists of fruit, leaves, flowers, and other vegetables. Fruit, readily taken by turtles and tortoises, includes banana, papaya, apple, mulberry, pumpkin, and tomatoes. Leaves include chicory, kale, cabbage, mulberry, hibiscus, *Elodea,* and lettuce. *Geochelone carbonaria* and *G. denticulata* are fond of the flowers of hibiscus and *Malvaviscus; G. nigra* readily accepts prickly pear (*Opuntia*). Carrots are also readily eaten by chelonians. Diets containing an imbalance in the calcium/phosphorus ratio, with an excess of phosphorus, must be supplemented with powdered calcium carbonate, bone meal, or a similar calcium-rich material (see examples in Table 7-6, in Fowler[17]). Turtles and tortoises usually accept pelleted commercial dog or cat chows, but these should not be offered in excess.[18,19] Baer discusses many aspects of the nutrition of herbivorous reptiles.[6] Tortoises must have access to fresh drinking water.

Visual and olfactory stimuli are important to stimulate normal foraging behavior. *Geochelone carbonaria* and *G. denticulata* are attracted by red, orange, and yellow colors. Frequency of feeding should depend on the temperature at which the chelonian is kept. During warm months they may be fed five times a week; during cold months they can be fed three times a week.

Special attention must be paid to the diets of the young of such species as *Trachemys scripta* and *Trachemys dorbignyi* that prefer animal matter. To ensure that they also eat plant matter, it is necessary to vary their daily diets, routinely feeding only vegetables on selected days. Another problem that needs special attention is the influence of group hierarchy on food access. Monitoring is necessary to ensure that every turtle in a particular group is feeding well. Hierarchy interference was observed in a group of young *Trachemys dorbignyi* maintained at São Paulo Zoo (Moraes and Molina, personal observation).

EGG COLLECTION, INCUBATION TECHNIQUES, AND HATCHLING CARE After selecting a proper nest site females of all chelonian species lay eggs, usually in cavities dug into the substrate. To achieve the best results, the eggs should be removed from the nests as soon as possible, measured, and artificially incubated. It is advisable to keep the eggs in the same position as they are found in the nest;[1,14,18] parchment-shelled and leathery-shelled eggs may be easily damaged when not handled with proper care. Records should be kept of the nesting process; such facts as female identification, female nesting behavior, nest site selected, nest measurements,[34] and weather conditions, are important to improve knowledge about South American chelonians.[40,41] The eggs should be placed in a plastic container and covered with 1 or 2 cm of moist vermiculite. A 1:1 ratio of vermiculite and tap water, by mass, has been suggested.[14,46] A closed container must be opened periodically to allow aeration; the vermiculite in an open container must be moistened periodically to compensate for evaporation. Egg containers should be placed inside a Styrofoam box supplied with ordinary lamps and thermostats.

At São Paulo Zoo chelonian eggs were incubated at temperatures between 25 to 30°C. *Phrynops geoffroanus, P. vanderhaegei, Podocnemis unifilis, Trachemys dorbignyi, Trachemys scripta, Kinosternon scorpioides, Geochelone carbonaria,* and *G. denticulata* have been successfully hatched. Temperature affects length of incubation,[1,14,42] and in many species determines the sex of the hatchlings.[14,15,29,46] The eggs must be regularly inspected and the increase in the chalky white band must be monitored.[14] Decomposing eggs should be immediately removed.

Newborns should be cleaned with tap water to wash away small flecks of vermiculite that may obstruct the eyes and nares. Sometimes newborns hatch prematurely with a persistent yolk sac. This seems more likely to happen with species that lay parchment-shelled eggs, such as *Trachemys dorbignyi.* Premature hatchlings should be maintained for 24–48 hours between two layers of highly moistened cotton, close to a heat source (at about 30°C). After this period the greater part of the yolk sac will have absorbed and the young are no longer at risk.

Young turtles and tortoises should be kept in individual plastic containers in a heated room (at about 26°C) for 1 or 2 months, during which time they should be measured and identified (by photographs, line drawings, or photocopies). Usually, normal hatchlings are fed for the first time at the end of the first week. They should be allowed to bask outdoors almost every day.

After 1 or 2 months, they may be transferred to small outdoor enclosures or to 1000-L tanks, where they will live in groups. Internal space may be divided into one-fourth sand and three-fourths water at a depth of 15 cm. Tanks of this size can house about 30 newborns of medium-sized species (e.g., *Trachemys dorbignyi* and *Phrynops geoffroanus*).

At 2 years of age, the tanks can house 10 to 20 young. Individuals older than 2 years of age should be transferred to larger tanks or exhibits. Group density affects growth and survival rates. In studying groups of 5, 10, and 30 young of *Trachemys dorbignyi* maintained in the same kinds of tanks, an inverse relationship between turtle densities and growth and survival rates was observed.

REFERENCES

1. Alderton, D. 1988. Turtles and Tortoises of the World. New York, Facts on File.
2. Allen, M.E.; and Oftedal, O.T. 1994. The nutrition of carnivorous reptiles. In J. B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 71–82.
3. Almeida, S.S.; Sá, P.G.S.; and Garcia, A. 1986. Vegetais utilizados como alimento por *Podocnemis* (Chelonia) na região do Baixo Rio Xingú (Brasil(Pará) [Vegetables used as food by *Podocnemis* in the lower Xingú river region]. Bol. Mus. Paraense E. Goeldi, Botânica 2:199–211.
4. Astort, E.D. 1984. Dimorfismo sexual secundario de *Phrynops* (*Phrynops*) *hilarii* (D. y B., 1835) y su conducta reproductora en cautiverio (Testudines-Chelidae) [*Phrynops hilarii* sexual dimorphism and reproductive behavior in captivity]. Revista del Museo Argentino de Ciencias Naturales "Bernardion rivadavia", Zoología 13(9):107–113.
5. Auth, D.L. 1975. Behavioral ecology of basking in the yellow-bellied turtle, *Chrysemys scripta scripta* (Schoepff). Bulletin of the Florida State Museum, Biological Science 20(1):1–45.
6. Baer, D.J. 1994. The nutrition of herbivorous reptiles. In J.B. Murphy, K. Adler, and J. T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 83–90.
7. Cagle, F.R. 1939. A system of marking turtles for future identification. Copeia 1939(3):170–173.
8. Cantarelli, V.H. 1997. The Amazon turtles—Conservation and management in Brazil. In J. Van Abbema, ed., Proceedings of Conservation, Restoration, and Management of Tortoises and Turtles—An International Conference. New York, New York Turtle and Tortoise Society, pp. 407–410.
9. Chessman, B.C. 1987. Atmospheric and aquatic basking of the Australian freshwater turtle *Emydura macquarii* (Gray) (Testudines: Chelidae). Herpetologica 43(3):301–306.
10. Chiszar, D.; Smith, H.M.; and Carpenter, C.C. 1994. An ethological approach to reproductive success in reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 147–173.
11. Davies, P.M.C. 1981. Anatomy and physiology. In J.E. Cooper and O.F. Jackson, eds., Diseases of the Reptilia, Vol. 1. London, Academic Press, pp. 9–73.
12. Ernst, C.H.; and Barbour, R.W. 1989. Turtles of the World. Washington, D.C., Smithsonian Institution Press.
13. Ernst, C.H.; Lovich, J.E.; and Barbour, R.W. 1994. Turtles of the United States and Canada. Washington, D.C., Smithsonian Institution Press.
14. Ewert, M.A. 1985. Embryology of turtles. In C. Gans, F. Billett, and P.F.A. Maderson, eds., Biology of the Reptilia, Vol. 14A. New York, John Wiley and Sons, pp. 75–268.
15. Ewert, M.A.; and Nelson, C.E. 1991. Sex determination in turtles: Diverse patterns and some possible adaptive values. Copeia 1991(1):50–69.
16. Fachin-Teran, A.; Vogt, R.C.; and Gomez, M.F.S. 1995. Food habits of an assemblage of five species of turtles in the Rio Guapore, Rondonia, Brazil. Journal of Herpetology 29:536–547.
17. Fowler, M.E. 1978. Metabolic bone disease. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 55–76.
18. Frye, F. L. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry. Malabar, Florida, Krieger Publishing.
19. Frye, F. L. 1991. A Practical Guide for Feeding Captive Reptiles. Malabar, Florida, Krieger Publishing.
20. Gaffney, E.S.; Hutchison, J.H.; Jenkins, F.A., Jr.; and Meeker, L.J. 1987. Modern turtle origins: The oldest known cryptodire. Science 237:289–291.
21. Gaffney, E.S.; and Kitching, J.W. 1994. The most ancient African turtle. Nature 369:55-58.
22. Gehrmann, W.H. 1987. Ultraviolet irradiances of various lamps used in animal husbandry. Zoo Biology 6(2):117–127.
23. Gehrmann, W.H. 1994. Light requirements of captive amphibians and reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 53–59.
24. Gibbons, J.W. 1990. Turtle studies at SREL: A research perspective. In J.W. Gibbons, ed., Life History and Ecology of the Slider Turtle. Washington, D.C., Smithsonian Institution Press, pp. 19–44.
25. Holmstrom, W.F. 1978. Preliminary observations on prey herding in the matamata turtle, *Chelus fimbriatus*

(Reptilia, Testudines, Chelidae). Journal of Herpetology 12(4):573–574.
26. IBAMA. 1989. Legislação sobre zoológicos [Legislation About Zoos]. Brasília, Instituto Brasileiro do Meio Ambiente e dos Recursos Naturais Renováveis.
27. IUCN. 1996. 1996 IUCN Red List of Threatened Animals. Gland, Switzerland, IUCN.
28. Iverson, J.B. 1992. A Revised Checklist with Distribution Maps of the Turtles of the World. Richmond, Indiana, Iverson.
29. Lance, V.A. Ed. 1994. Environmental sex determination in reptiles: Patterns and processes. Journal of Experimental Zoology 270(1).
30. Lee, M.S.Y. 1993. The origin of the turtle body plan: Bridging a famous morphological gap. Science 261:1716–1720.
31. Licata, L.; and Elguezabal, X. 1997. Management plan for the Giant Amazonian turtle, *Podocnemis expansa,* in De La Tortuga Arrau Wildlife Refuge, Orinoco river, Venezuela. In J. Van Abbema, ed., Proceedings of Conservation, Restoration, and Management of Tortoises and Turtles—An International Conference. New York, New York Turtle and Tortoise Society, pp. 171–173.
32. Lima, A.C.; Magnusson, W.E.; and Costa, V.L. 1997. Diet of the turtle *Phrynops rufipes* in Central Amazonia. Copeia 1997(1):216–219.
33. Marcovaldi, M.A.; and Laurent, A. 1996. A six-season study of marine turtle nesting at Praia do Forte, Bahia, Brazil, with implications for conservation and management. Chelonian Conservation and Biology 2(1):55–59.
34. Medem, F. 1976. Recomendaciones respecto a contar el escamado y tomar las dimensiones de nidos, huevos y ejemplares de los Crocodylia y Testudines [Recommendations respecting scale count, nests, eggs, and specimen measurements in Crocodilia and Testudines]. Lozania (20):1–17.
35. Molina, F.B. 1990. Observações sobre os hábitos e o comportamento alimentar de *Phrynops geoffroanus* (Schweigger, 1812) em cativeiro (Reptilia, Testudines, Chelidae) [Observations on the feeding habits and behavior of *Phrynops geoffroanus* in captivity]. Revista Brasileira de Zoologia 7(3):319–326.
36. Molina, F.B. 1991. Some observations on the biology and behavior of *Phrynops geoffroanus* (Schweigger, 1812) in captivity (Reptilia, Testudines, Chelidae). Grupo de Estudos Ecológicos, Série Documentos (3):35–37.
37. Molina, F.B. 1992. O comportamento reprodutivo de quelônios [Chelonians reproductive behavior]. Biotemas 5(2):61–70.
38. Molina, F.B. 1996. Biologia e comportamento reprodutivo de quelônios [Chelonians biology and reproductive behavior]. In K. Del-Claro, ed., Anais do 14° Encontro Anual de Etologia. Uberlândia, Sociedade Brasileira de Etologia and Universidade Federal de Uberlândia, pp. 211–221.
39. Molina, F.B. 1996. Mating behavior of captive Geoffroy's side-necked turtles, *Phrynops geoffroanus* (Testudines: Chelidae). Herpetological Natural History 4(2):155–160.
40. Molina, F.B. 1998. Comportamento e biologia reprodutiva dos cágados *Phrynops geoffroanus, Acanthochelys radiolata* e *Acanthochelys spixii* (Testudines, Chelidae) em cativeiro [Behavior and reproductive biology of captive *Phrynops geoffroanus, Acanthochelys radiolata* e *Acanthochelys spixii*]. Revista de Etologia (special number):25–40.
41. Molina, F.B.; and Gomes, N. 1998. Breeding and nesting behavior of the d'Orbigny's slider turtle *Trachemys dorbignyi* at São Paulo Zoo. International Zoo Yearbook 36:162–170.
42. Molina, F.B.; and Gomes, N. 1998. Observações sobre a incubação artificial dos ovos e o processo de eclosão em *Trachemys dorbignyi* (Reptilia, Testudines, Emydidae) [Observations on the artificial incubation and hatching process of *Trachemys dorbignyi*]. Revista Brasileira de Zoologia 15(1):135–143.
43. Molina, F.B.; Rocha, M.B.; and Lula, L.A.B.M. 1998. Observações sobre o comportamento alimentar e a dieta de *Phrynops hilarii* em cativeiro (Reptilia, Testudines, Chelidae) [Observations on the feeding behavior and diet of *Phrynops hilarii* in captivity]. Revista Brasileira de Zoologia 15(1):73–79.
44. Moll, E.O.; and Legler, J.M. 1971. The life history of a neotropical slider turtle, *Pseudemys scripta* (Schoepff), in Panama. Bulletin of the Los Angeles County Museum of Natural History and Science (11):1–102.
45. Moskovits, D.K.; and Bjorndal, K.A. 1990. Diet and food preferences of the tortoises *Geochelone carbonaria* and *G. denticulata* in northwestern Brazil. Herpetologica 46(2):207–218.
46. Packard, G.C.; and Phillips, J.A. 1994. The importance of the physical environment for the incubation of reptilian eggs. In J.B. Murphy, K. Adler, and J. T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 195–208.
47. Páez, V.P., and Bock, B.C. 1997. Nesting ecology of the Yellow-spotted river turtle in the Colombian Amazon. In J. Van Abbema, ed., Proceedings of Conservation, Restoration, and Management of Tortoises and Turtles—An International Conference. New York, New York Turtle and Tortoise Society, pp. 219–224.
48. Pérez-Emán, J.L.; and Paolillo O.A. 1997. Diet of the pelomedusid turtle *Peltocephalus dumerilianus* in the Venezuelan Amazon. Journal of Herpetology 31(2):173–179.
49. Pough, F.H.; Heiser, J.B.; and McFarland, W.N. 1996. Vertebrate Life, 4th ed. Prentice-Hall.
50. Pritchard, P.C.H. 1984. Piscivory in turtles, and evolution of the long-necked Chelidae. Symposium of the Zoological Society of London (52):87–110.
51. Pritchard, P.C.H. 1997. Turtles as a resource: Avoiding the "Tragedy of the Commons." In J. Van Abbema, ed., Proceedings of Conservation, Restoration, and Management of Tortoises and Turtles—An International Conference. New York, New York Turtle and Tortoise Society, pp. 444–445.

52. Pritchard, P.C.H.; and Trebbau, P. 1984. The Turtles of Venezuela. Oxford, Society for the Study of Amphibians and Reptiles.
53. Reisz, R.R.; and Laurin, M. 1991. *Owenetta* and the origin of turtles. Nature 349:324–326.
54. Rhodin, A.G.J.; Medem, F.; and Mittermeier, R.A. 1981. The occurrence of neustophagia among podocnemine turtles. British Journal of Herpetology 6:175–176.
55. Rougier, G.W.; de la Fuente, M.S.; and Arcucci, A.B. 1995. Late Triassic turtles from South America. Science 268:855–858.
56. Souza, F.L.; and Abe, A.S. 1995. Observations on feeding habits of *Hydromedusa maximiliani* (Testudines: Chelidae) in Southeastern Brazil. Chelonian Conservation and Biology 1(4):320–322.
57. Souza, F.L.; and Abe, A.S. 1999. Um sobrevivente em rios poluídos [A survivor in polluted rivers]. Ciência Hoje 25(147):59–61.
58. Spotila, J.R.; Foley, R.E.; and Standora, E.A. 1990. Thermoregulation and climate space of the slider turtle. In J.W. Gibbons, ed., Life History and Ecology of the Slider Turtle. Washington, D.C., Smithsonian Institution Press, pp. 288–298.
59. Swingland, I.R. 1989. *Geochelone elephantopus:* Galapagos giant tortoise. In I.R. Swingland and M.W. Klemens, eds., The Conservation Biology of Tortoises. Gland, Switzerland, IUCN—The World Conservation Union, pp. 24–28.
60. Walker, P. 1989. *Geochelone chilensis:* Chaco tortoise. In I.R. Swingland and M.W. Klemens, eds., The Conservation Biology of Tortoises. Gland, Switzerland, IUCN—The World Conservation Union, pp. 20(21.

CHELONIAN INFECTIOUS DISEASES AND GENERAL MEDICINE

Eliana Reiko Matushima

RESPIRATORY TRACT DISEASE

Inadequate temperature and humidity, suboptimal hygiene conditions over inadequate substrate, and vitamin A–deficient diet have been associated with respiratory tract disease.

The absence of a diaphragm in chelonians has functional significance, resulting in an inability to cough and expel secretions and exudate from the lungs. This anatomical restriction undoubtedly contributes to the progress of a respiratory tract infection to pyogranulomatous pneumonia.[2]

Numerous etiologic agents have been associated with respiratory disease in chelonians. Bacterial pathogens such as *Pasteurella* sp., *Streptococcus* spp., *Staphylococcus* negative coagulase; fungal agents such as *Sporotrichium* sp., *Cladosporium*, *Parcilomyces* sp., *Candida* sp., *Penicilium* sp., and *Aspergillus* sp.; viral agents such as herpesvirus and iridovirus; and *Mycoplasma* have been isolated, as well as metazoic parasites.

Clinical signs of respiratory disease include nasal and ocular discharges, dyspnea and open-mouth breathing, anorexia and weight loss, and lethargy. Aquatic and semiaquatic turtles often show inability to swim or float in proper position due to consolidation or collapse of pulmonary parenchyma.[2]

Diagnostic methods include radiographic examination and tracheal wash using sterile saline. Fluid should be submitted for culture and sensitivity, as well as cytology. Viral isolation may be done if indicated.[3-7]

Therapy for respiratory infections is identical to that employed in higher vertebrates. Based on culture and sensitivity results, a broad-spectrum bactericidal antibiotic should be administrated. Nebulization with antibiotic and mucolytic agents may aid in treatment.

GASTROINTESTINAL TRACT DISEASE

Turtles and tortoises commonly present with chronic hypophagia or anorexia. This clinical sign may result from numerous causes, including infectious or metabolic diseases, parasitism, or gastrointestinal obstruction. Conditions of improper husbandry, including low temperature, stress, inappropriate diet, and failure of the animal to adapt to captivity, all may result in anorexia.[2] Bacterial enteritis, including salmonellosis, may be a chronic condition associated with pneumonia and septicemia or may present acutely resulting in systemic shock and death.

Clinical signs include dehydration, anorexia, lethargy, and decreased body weight. It is common to find bacterial enteritis, amoebic enterohepatitis, chronic parasitism, foreign body ingestion, and obstruction or impaction.

Antibiotic treatment may be recommended based on culture results as well as systemic fluid and electrolyte replacement.

Amoebic enterohepatitis is characterized by clinical signs similar to those of gastroenteritis. The presence of *Entamoeba invadens* is confirmed by the presence of elongated uninuclear trophozoites or rounded quadrinucleated cysts in fecal samples. Treatment with metronidazole at recommended dosages is usually effective, but the patient may succumb to associated hepatitis.[2]

Impaction caused by sand or foreign body ingestion is commonly seen in captive chelonians. Diagnosis is made through radiograph examination and endoscopy, followed by surgical removal of foreign bodies. Impactions caused by sand ingestion may require surgical procedures, fiber ingestion, or rehydration therapy.

REPRODUCTIVE DISORDERS

Dystocia is the most common reproductive disorder encountered in chelonians. Causes of dystocia include poor environmental conditions leading to chronic debilitation, metabolic disorders, anatomic anomalies of the reproductive tract or eggs, and repeated copulation resulting in broken eggs.[2]

Common clinical signs include anorexia, lethargy, history of straining to pass eggs, and hemorrhagic discharge from the cloaca. Diagnosis is usually based on radiographic evidence of retained eggs.

Treatment should be based on radiological evaluation. If no evidence of obstructive dystocia is present, administration of calcium gluconate and 5% dextrose may stimulate oviposition. Oxytocin should be used with extreme caution and only in animals that do not respond to initial treatment. If radiographs reveal fractured eggs or anatomic abnormalities preventing oviposition, surgical intervention is recommended. Eggshell fracture, oviduct laceration, and contamination of the ceolomic cavity with yolk commonly result in yolk seroceolomitis. Surgical repair may include copious lavage of the ceolomic cavity with warm physiologic saline. Antibiotic therapy should be instituted. In chronic cases, prognosis for recovery is poor once fibrinous ceolomitis occurs.[2]

Male chelonians occasionally demonstrate penile prolapse as a result of trauma, infection, neurologic deficiency in the retractor apparatus or cloacal sphincter, or impaction of urate material in the cloaca. Simple cases of prolapse may be treated by cleansing, lubricating, and replacing the penis. A temporary cloacal purse-string closure may be indicated for retention. If the prolapse is accompanied by swelling of the penis, application of a hygroscopic fluid may reduce swelling and permit replacement. If the penis is traumatized or desiccated, amputation may be indicated.

METABOLIC AND NUTRITIONAL DISEASE

Dietary imbalances commonly cause disease in captive chelonians. Usually it results from improper mineral balance, vitamin deficiencies, and excessive protein levels in the diet.

Metabolic bone disease is one of most common nutritional disorders in captive chelonians. For a discussion of this disease, see Specific Nutritional Disorders.

Excessive dietary calcium and vitamin D_3 may be problematic when chelonians are fed commercial pet foods. Mineralization of smooth muscle and renal tissue is often a secondary necropsy finding in animals that have been maintained on dog, cat, or monkey food.

Vitamin A deficiency is most commonly observed in captive turtles maintained on a high-protein, vitamin A–deficient diet. Clinical signs include conjunctivitis, blepharitis, and swelling of the adnexae. In vitamin A deficiency, mucin production decreases, and the glandular structure of the eyes and respiratory passages undergo squamous metaplasia. Hyperkeratosis and squamous metaplasia of ocular, nasal, and pharyngeal mucosal epithelia result in secondary invasion of these surfaces by opportunistic pathogenic microorganisms. Administration of parenteral or oral vitamin A and correction of dietary deficiencies are recommended. Chronic cases of vitamin A deficiency usually require antibiotic therapy for pathogenic microorganisms that have secondarily invaded damaged mucous membranes.[2]

Hyperuricemia or gout occurs frequently. Diets containing excessive levels of animal protein, reduction of renal perfusion caused by dehydration or nephrosis, and renal tubular insult from nephrotoxic drugs may all result in visceral, articular or periarticular gout. Terrestrial chelonians fed concentrated sources of animal protein will develop a high serum concentration of uric acid. Iatrogenic hyperuricemia may result from the administration of aminoglycosides or sulfonamides at elevated dosages or in the presence of clinical dehydration.

Clinical signs of hyperuricemia include lameness, joint swelling, and elevated serum uric acid concentrations. Radiographs of affected areas may reveal radio-opaque lesions associated with urate crystal deposition. Therapy must include correction of the primary cause of hyperuricemia. Urate depositions can be removed aseptically from affected joints. Subsequent anti-inflammatory therapy has proven beneficial. Systemic therapy should be monitored by sequential measurements of serum uric acid concentrations.[2]

DISEASES OF THE SHELL AND SHELL REPAIR

Abnormalities of the shell are usually the result of trauma or improper environmental conditions, although several organisms have been isolated in association with shell disease. There are reports of shell lesions in captive chelonians caused by *Mucor* sp., *Proteus* sp., *Escherichia coli, Pseudomonas* sp., and *Baneckea chitinovora,* and even viral agents such as poxvirus.[1,9] Additionally, the detachment and loss of keratin scutes has been associated with elevated serum uric acid concentrations during renal failure. Treatment of shell disease typically involves debridement and topical antimicrobial therapy. After associated infection has been resolved, the defect can be repaired with epoxy resin.

A specific disease syndrome, septicemic cutaneous ulcerative disease, has been identified in aquatic turtles. It is characterized by cutaneous ulceration, anorexia, lethargy, and septicemia progressing to death as the ulcers deepen. Soft-shelled turtles of the family Trionychidae are most frequently affected, and the associated etiologic agent has been identified as a gram-negative bacteria. This disease has been treated with bactericidal antibiotics and supportive care.[8]

Traumatic shell injury is one of the most common clinical presentations of all types of chelonians. Whole-body radiographs should be assessed to determine the extent of injury to the bony shell and appendicular skeleton. If limb paralysis or paresis is present, prognosis for recovery is poor.

Areas of shell trauma should be debrided and devascularized fragments of bone removed. Lavage with sterile saline will greatly reduce associated infection, and antibiotic therapy should be initiated. In grossly contaminated fractures or old injuries, final shell repair is done after a healthy bed of granulation tissue has been established. Repair of the shell may require placement of wires to hold pieces in apposition, or layers of sterile fiberglass cloth impregnated with rapid polymerizing epoxy resin may be used to bridge defects. The periphery of the defect should be cleaned with ether or acetone, and the epoxy resin mixed and applied to the periphery of the defect, avoiding contact with the edge of the shell or soft tissues.[2]

Alternatively, shell defects may be repaired with hoof repair acrylic or dental acrylic. Dental acrylic is useful if the shell repair contacts soft tissue, because this material is nontoxic and does not generate heat in association with polymerization. Hoof acrylic is more difficult to manipulate than resin but is extremely strong and durable for shell repair of giant tortoises.

NEOPLASIC DISEASE

The most frequent neoplasic disease observed nowadays is fibropapilloma in sea turtles. It is a neoplasia that originates in the epithelium and is benign. It may be a papilloma or a fibropapilloma, depending on stromal and/or epithelial proliferation.

Papillomatous formations may be variable, with solid tissue projections varying in consistency from soft to firm and diameters from 0.5 to 10 cm. These formations may have vegetative and verrucous forms, with papillar aspects or a more regular surface. The color may vary from whitish to grayish or be several tones of pink. Some papillomatous formations, as a result of their high stroma vascularization and traumas suffered by the animals, present ulcers with discrete hemorrhage and may or may not be parasitized by small trematode larva. The papillomatous formations may be in pelvic or thoracic limbs, head, dorsal cervical area, eyelid, oral region, inguinal or pericloacal area. It is possible to find papilloma in all of the areas mentioned in a single animal. The number of papilloma found in a single sea turtle may vary from 2 to 100.

Through the use of polymerase chain reaction (PCR) amplification, an alphaherpesvirus, a retrovirus, and a papillomavirus have been identified in fibropapillomatous lesions of sea turtles.

REFERENCES

1. Garner, M.M.; Herrington, R.; Howerth, E.W.; Homer, B.L.; Nettles, V.F.; Isaza, R.; Shotts, E.B.; and Jacobson, E.R. 1997. Shell disease in river cooters (*Pseudemys concinna*) and yellow-bellied turtles (*Trachemys scripta*) in a Georgia (USA) lake. Journal of Wildlife Disease 33(1):78–86.
2. Mautino, M.; and Page, C.G. 1993. Biology and medicine of turtles and tortoise. Veterinary Clinics of North America: Small Animal Practice 23(6):1251–1271.
3. Orós, J.; Ramirez, A.S.; Poveda, J.B.; Rodriguez, J.L.; and Fernandez, A. 1996. Systemic mycosis caused by *Penicillium griseofulvum* in a Seychelles giant tortoise (*Megalochelys gigantea*). The Veterinary Record 37:295–296.
4. Pettan-Brewer, K.C.B.; Drew, M.L.; Ramsay, E.; Mohr, F.C.; and Lowenstein, L.J. 1996. Herpesvirus particles associated with oral and respiratory lesions in a California desert tortoise (*Gopherus agassizii*). Journal of Wildlife Diseases 32(3):521–526.
5. Jacobson, E.R.; Gaskin, J.M.; Shields, R.P.; and White, F.H. 1979. Mycotic pneumonia in mariculture-reared Green Sea Turtles. Journal of American Veterinary Medical Association 175(9):929–933.
6. Jacobson, E.R.; Gaskin, J.M.; Brown, M.B.; Harris, R.K.; Gardiner, C.H.; LaPointe, J.L.; Adams, H.P.; and Reggiardo, C. 1991. Chronic upper respiratory tract disease of free-ranging desert tortoises (*Xerobactes agasizzi*). Journal of Wildlife Diseases 27(2):296–316.
7. Westhouse, R.A.; Jacobson, E.R.; Harris, R.K.; Winter, K.R.; and Homer, B.L. 1996. Respiratory and pharyngoesophageal iridovirus in a gopher tortoise (*Geopherus polyphemus*). Journal of Wildlife Diseases 32(4):682–686.
8. Jacobson, E.R.; Calderwood, M.B.; and Clubb, S.L. 1980. Mucormycosis in hatchling Florida softshell turtles. Journal of American Veterinary Medical Association 177(9):835–837.
9. Orós, J.; Rodríguez, J.L.; Déniz, S.; Fernández, L.; and Fernández, A. 1998. Cutaneous poxvirus-like infection in a captive Hermann's tortoise (*Testudo hermanni*). The Veterinary Record 31:508–509.

CHELONIAN NONINFECTIOUS DISEASES

Margarita Mas

SPECIFIC NUTRITIONAL DISORDERS

Most nutritional problems arise during active growth. Malnutrition may be the major cause of mortality in juveniles and young adults. Deficiencies and over feeding generally appear in combination, for example a tortoise with a softened shell caused by an inadequate calcium/phosphorus ratio in the diet may also suffer generalized edema in its limbs as a result of renal failure caused by too-high ingestion of protein.

METABOLIC BONE DISEASE

By far, metabolic bone disease is the most common of nutritional disorders of chelonians, usually resulting from ignorance of the requirements for an adequate diet. Instead, animals may be fed a diet with an inadequate calcium level, a too-high level of phosphorus, an unsuitable level of vitamin D_3, and too much protein. Turtles that in freedom eat a great variety of prey receive only minced meat in captivity, and tortoises that usually eat many different plants are fed lettuce as the major food item.

These management errors lead to progressive distortion in the shape, size, and consistency of the shell. These distortions are first evidenced by the softening of the plastron and motor function difficulties. When decalcification becomes severe, there is smoothing and lordosis in the region above the pelvis, and often dyspnea also occurs as a consequence of the decrease of the pulmonary space and pain when touched (Fig. 3.1 and Fig. 3.2). Radiographically, a loss of bone density and spontaneous fractures may be seen.

Management is diet correction, improvement of the environment and administration of calcium and vitamin D_3.

VITAMIN A DEFICIENCY

Although vitamin A deficiency is found in both tortoises and turtles, it is more common in turtles. Vitamin A is fat soluble, and a deficiency leads to squamous metaplasia of glands around the eye and in the oral cavity, kidneys, and respiratory and digestive systems.

Clinical signs observed are palpebral edema and conjunctivitis, dyspnea or a respiratory illness, stomatitis, skin hyperkeratosis, and, in severe cases, renal failure, hepatic failure, and death.

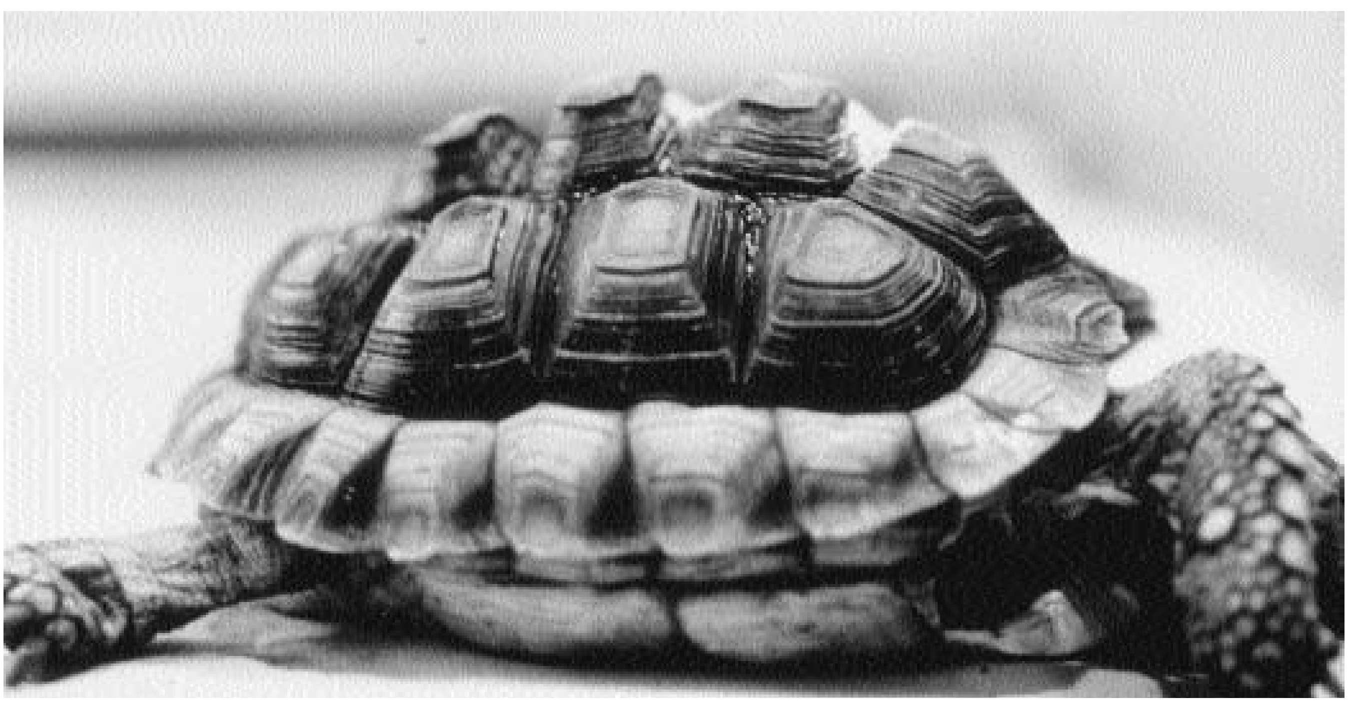


FIGURE 3.1. High levels of protein stimulate excessive growth of the carapace.

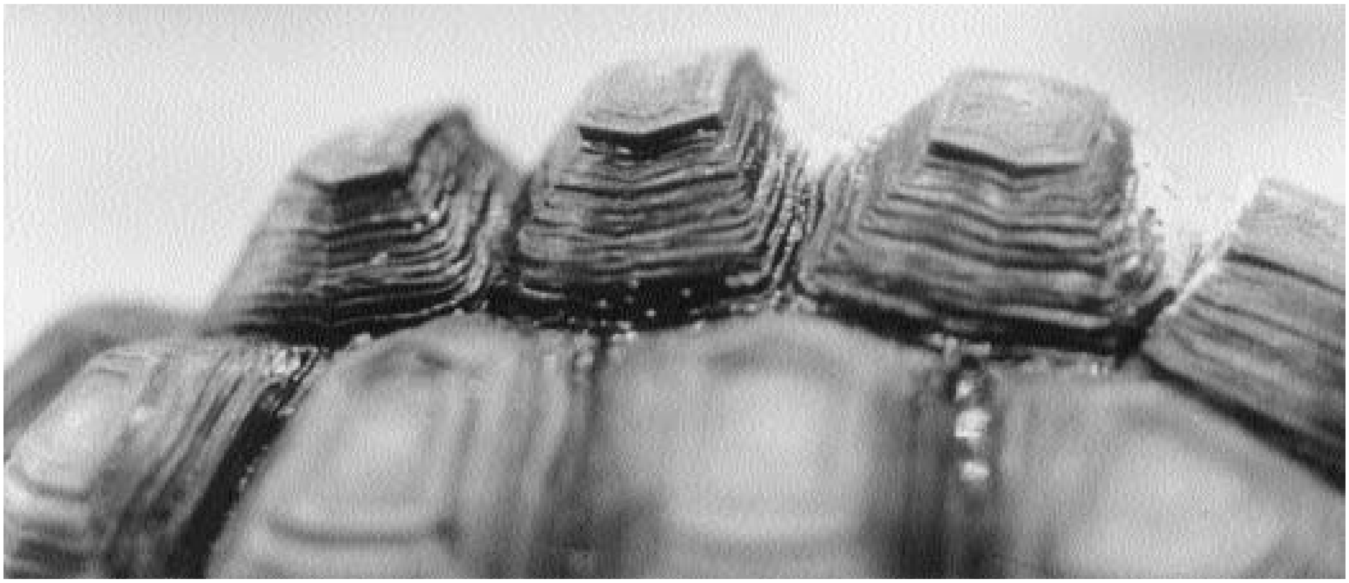


FIGURE 3.2. Nonreversible pyramidal shape of the carapace as a result of high-protein diet during growth.

Management

Management is vitamin and mineral supplementation, correction of the diet by adding natural sources of vitamin A, such as fruits and vegetables containing carotene, and treatment of the secondary infections. Excessive levels of vitamin A may cause toxicity, resulting in lesions similar to those caused by deficiency, such as skin thickening and sloughing with serious secondary effects.

Protein Excess

The main problem of protein excess occurs in herbivorous testudines when fed a high protein diet of animal origin. High levels of uric acid and urea accumulate in the blood, causing a precipitation of these elements into the tissues and joints, a process known as gout (visceral or articular). Also, formed crystals may precipitate in the renal tubules, blocking them (renal constipation), leading to renal failure. Affected tortoises show anorexia, edema and, finally, death.

High levels of protein in the diet of growing chelonians cause an abnormal development of the shell, with pyramidal distortion of the shields (Figures 3.1 and 3.2), melanistic coloration resulting from hyperplasia of the keratin coat and softening of the shell because of nutritional osteodystrophia and, as a consequence of renal complications, edema and death caused by renal failure.

Other diseases related to nutritional imbalances are steatitis, common in aquatic turtles that ingest high levels of fatty acids. This condition is characterized by obesity, accumulation and degeneration of fat deposits, and icterus because of hepatic lipidosis.

INTOXICATIONS

Most intoxications occur accidentally when certain poisons are incorporated into the environment or captive surroundings, either directly or indirectly. The use of disinfectants, such as alcohol or phenol derivates, or aerosol or liquid insecticides is a common cause of iatrogenic intoxication when they are used improperly as external antiparasitic agents.

Intoxications in private homes are frequent when owners paint the shells of pet turtles. Garden plants may also be hazardous (potato leaves, lobelia, lily of the valley, lupine, foxglove, rhododendron, delphinium, cyclamen, and certain mushrooms).

CONSTIPATION

Another frequent problem, especially in tortoises, is constipation, which may be caused primarily by the accumulation of dry matter, e.g., cage litter, small stones, or other materials in the intestine. In turtles, it is common to find stones or gravel ingested, because the animals are often fed in an area where these materials are available.

Secondary constipation is caused by dehydration, tumorous masses, a retained egg, or cystic calculi that compress the intestine by occupying the celomic space. Traumatisms and metabolic bone disease may lead to a paresis of the hind and fore limbs, with fecal retention as a result.

Management depends on the cause of the constipation. With primary constipation, animals should be hydrated and bathed with warm water (30°C) once or twice a day to stimulate defecation. Mild laxatives such as vaseline or milk of magnesia may be administered orally or by enema. In serious cases a celiotomy is necessary.

ANOREXIA

Anorexia is a common ailment of chelonians. If turtles fast for 3–4 weeks or fail to drink for 10 days, it is a sign of illness. Posthibernation anorexia is common, especially in malnourished animals or those subject to low temperatures during the winter. Anorexia also occurs in females with egg retention and animals infected with parasites or that suffer from metabolic illnesses. Anorexia may also be induced by factors related to the environment, such as below-optimal temperatures, inadequate habitats or terrariums, and the use of diets that do not encourage an interest in food. Although not related to pathologic causes, male anorexia is common during the breeding season.

Management should begin with correction of environmental factors, hydration, reestablishment of glucose levels, and decrease of uremia. Forced feeding using a gastric tube may be necessary.

ENDOPARASITES

Flagellate Protozoans

Many factors may be involved in fostering the presence of flagellates in turtles and tortoises, including inadequate management of the diet (low in fiber or extremely rich in sugar), high population density, incorporation of new individuals into a mixed group, poor sanitation, and high temperature and humidity. Some protozoans such as *Trichomona, Giardia,* or *Leptomonas* are considered to be nonpathogenic in the majority of reptiles, but a large number of one or more of these parasites combined with other infectious or parasitic agents may contribute to illnesses, including diarrhea, dehydration, anorexia, and emaciation.

Diagnosis is accomplished by observing the parasite or parasites in fresh fecal samples. The recommended treatment is administration of metronidazole and immediate correction of the predisposing factors.

Hexamita parva is a highly pathogenic flagellate of the urinary system, capable of producing acute or chronic nephritis that may lead to renal failure in either tortoises or turtles. Some tortoises, such as *Geochelone* spp., are particularly sensitive. Signs include anorexia, weight loss, polydipsia, and green-colored urine containing mucous filaments, excessive sediment, and blood remnants, and with a strong, penetrating odor.

Diagnosis may be accomplished by microscopic examination of fresh or manually removed urine. Treatment and improvement of environmental conditions should be immediate.

Other Ciliate Protozoans

Nyctotheurus kyphodes, N. teleacus, and *Balantidium* spp. have been found in species of the genus *Geochelone,* but they have not been proven to be agents of illness. It is possible that they are normal microflora and participants of the digestive process.

Coccidia

Although coccidial organisms are commonly found, signs must be present to diagnose disease.

MANAGEMENT Numerous drugs have been used in treatment. Trimethoprim sulfadiazine, in doses of 15–30 mg/kg every 48 hours for 10–14 days, works well.

Helminths

Nematodes are the most common parasites found in chelonians. The best known are roundworms, of which the ascarids (8–10 cm long, yellowish and white) and oxyurids (1.5–8 mm long, thin and whitish) are by far the most common.

Signs depend on the number of parasites present, but include anorexia, vomiting, diarrhea, or, in some cases, constipation caused by obstruction of the intestine.

THERAPY Fenbendazole in a dose of 50 mg/kg, repeated after 14 days, or oxfendazole, at 60 mg/kg in a single dose, is recommended. The largest expulsion will occur during the first 7–10 days, decreasing remarkably after the second dose of fenbendazole. It is advisable to conduct routine fecal examinations every 6 months.

EXTERNAL PARASITES

Fly strike and myiasis are common following lacerations or trauma. Ticks may also be found.

A pyrethrin solution can be used topically around the wound, but not in it. The most important action is thorough cleansing of the wound, eliminating larvae, and careful disinfection with povidone iodine or a dilute hydrogen peroxide solution, along with administration of local and systemic antibiotics. Amitraz (2 mL/L water) is used for tick control. Ivermectin is toxic for chelonians and should not be used.

HANDLING

Small tortoises may be managed easily by applying mild pressure to the head or tail area to cause the opposite end to be exposed. The head may be grasped immediately behind the occipital crest. A brief struggle may ensue, but relaxation will occur. In larger tortoises, the operator should sit, put the animal between his or her knees, and softly tap on the caudal shield with the fingers until the head appears. The head should then be grasped firmly with one hand, using the other to manipulate, probe, and examine the mouth.

When handling box turtles, which have kinetic plastral hinges, it is necessary to use scissors (Mayo) or another instrument as a lever to open the shell. The opening may be blocked with a sponge or one of the limbs may be held outside the shell to avoid closure of the trap door on the handlers fingers.

Terrapins and soft-shelled turtles (*Trionix* spp., *Phrynops* spp.) may bite or claw severely, especially those with long necks that can be extended backward or laterally and that have long sharp nails. Small individuals may be handled from the back by placing a finger in the inguinal fosse. For larger animals it is necessary to use gloves or to allow them to bite on an object to extend the head.

Special care should be taken with the snapping turtle (*Chelydra sepentina*) because the jaws are hooked and prominent, and they may bite suddenly, inflicting severe lacerations or crushing injuries. If the operator suspects a serious personal risk, especially with large or aggressive individuals, chemical immobilization is recommended to produce restraint sufficient for a thorough examination and/or therapy.

ANESTHESIA

Preanesthetic requirements are the same for all species of reptiles and needn't be reiterated here. It is important to obtain an accurate weight for calculating anesthetic agent dosage and fluid loss after surgery.

Preanesthetic Drugs

The use of atropine sulfate (0.01–0.04 mg/kg) is recommended for small turtles to avoid excessive secretions. Acepromazine maleate (0.1–0.5 mg/kg) may be used to reduce the amount of the induction agent required. Administration of the combination of zolazepam with tiletamine (4–6 mg/kg) helps diminish muscle rigidity produced by tiletamine and aids in induction for intubation, but it is not advisible as the only anesthetic drug.

Injectable Anesthetics

Injectable anesthetics are used, but inhalant agents are much preferred. When ketamine hydrogen chloride is used as the sole anesthetic, total analgesia is not achieved. It should not be used in patients with renal or hepatic pathologies or in dehydrated animals because of the slow metabolic and detoxification mechanisms of chelonians. In healthy tortoises, doses are 60–80 mg/kg. A dose for producing relaxation before endotracheal intubation is 20–40 mg/kg.

Inhalant Anesthetics

Although halothane is frequently used, the recommended agent is isoflurane. With the latter, induction and recovery are rapid. Both agents require the use of a precision vaporizer. The anesthetic level should be checked with a pulse oximeter, with the sensor attached to a flap of skin on the hind limb. The heart should be monitored with an ECG and reflexes should be tested (palpebral, corneal), as well as the pain response, Figure 3.3.

SURGERY

Common chelonian surgeries include limb amputation (Figure 3.4), penis amputation, and celiotomy (egg retention, stones, ingested foreign bodies). General surgical principles should be followed.

Cystotomy

Urolith formation is not uncommon in tortoises. It is generally related to nutritional disorders or long periods without water. Signs vary according to the size and number uroliths, but include depression, lethargy, constipation, and hind limb paresis. The majority are urates and they may be as large as 10 to 12 cm in diameter. They may be palpated during a physical examination and are easily diagnosed through radiography.

When uroliths are small, a lateral celiotomy may be performed by placing the tortoise on its right side with the hind leg withdrawn caudally. The celomic cavity is entered through an incision and division of the muscles until the bladder and its stones are detected. Special care should be taken with the bladder wall, which is generally thin. The incision in the bladder should be closed with absorbable sutures 4-0, in a two-layer inverting pattern.

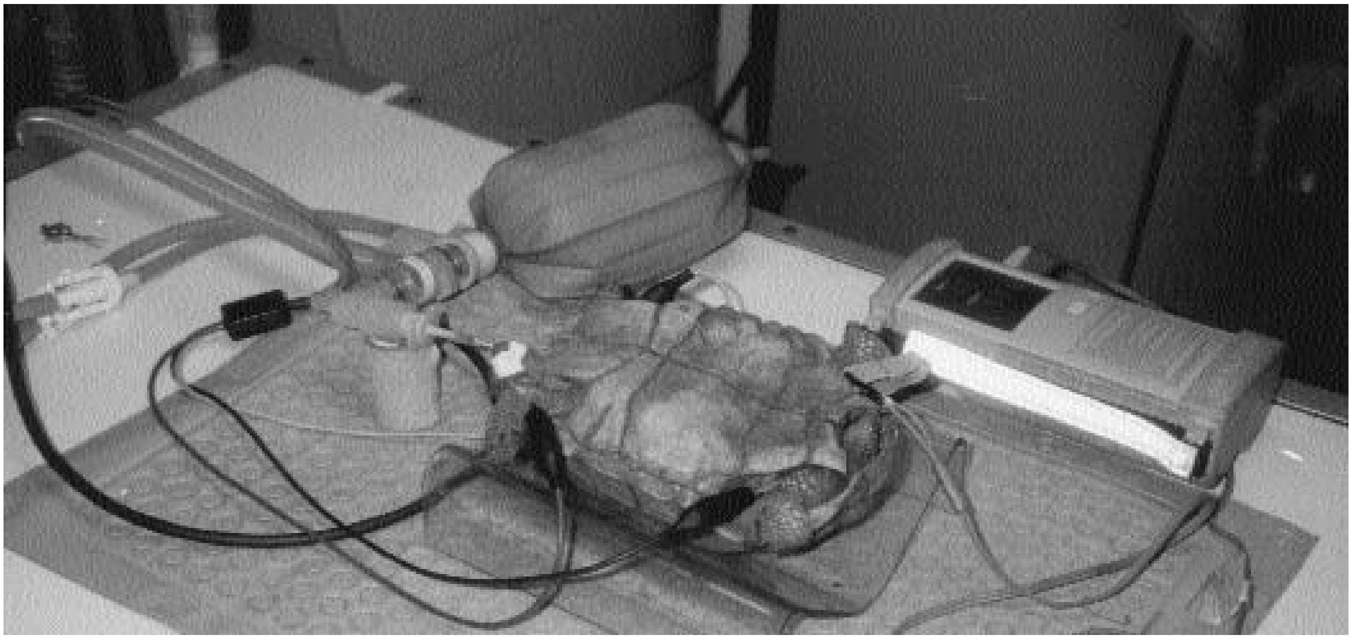


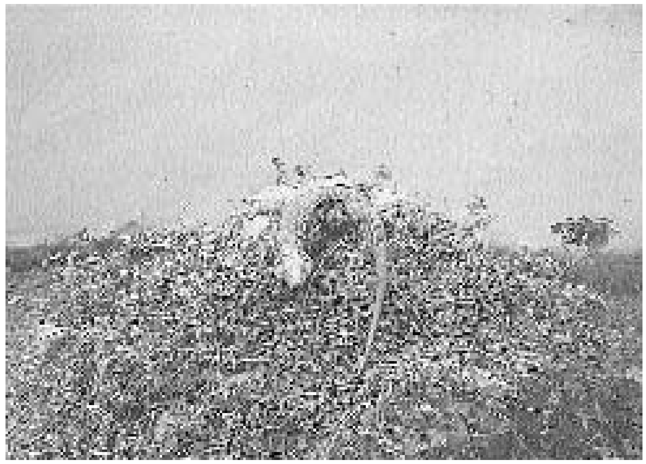
FIGURE 3.3. Anesthetic control with a sensor and an oximeter pulse connected with MPD, and three ECG leads to monitor heart rate. Body temperature is maintained through a heating pad.

FIGURE 3.4. Amputated rear leg. In chelonians it is advisable to amputate the entire leg to diminish skin abrasion.

The muscles should be closed with continuous sutures and the skin with everted sutures. Larger uroliths require a window to be opened through the plastron. The technique is described in many reptile medicine texts.

REFERENCES

1. Fowler, M.E. 1986. Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders.
2. Frye, L.F. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry. Krieger Publishing.
3. Highfield, A.C. 1996. Practical Encyclopedia of Keeping and Breeding Tortoises and Freshwater Turtles, 1st Ed. Ames, Iowa, Iowa State University Press.
4. Lewton, M.P.C.; and Cooper, J.E. 1994. Manual of Reptiles, 2nd Ed. England, British Small Animal Veterinary Association.
5. Mader, D.R. 1996. Reptile Medicine and Surgery. Philadelphia, W.B. Saunders.
6. McArthur, S. 1996. Veterinary Management of Tortoises and Turtles. Oxford, Blackwell Science.
7. Weigel, J.P. 1993. Textbook of Small Animal Surgery, Vol. 2, 2nd Ed. Philadelphia, W.B. Saunders.



4
Class Reptilia, Order Squamata, (Lizards): Iguanas, Tegus

Flavio de Barros Molina
Teresa L. Lightfoot

INTRODUCTION

The suborder Sauria, order Squamata, also includes the suborders Serpentes and Amphisbaenia. The Sauria first appeared during the Triassic Period.[10,12] Living lizards comprise about 3000 species[10,12] included in 25 families.[12] Ten families are represented in South America. According to the International Union for the Conservation of Nature (IUCN), six species of South American lizards are considered to be threatened (critically endangered and vulnerable).[9]

The objective of this chapter is to present information on how to manage and breed lizards in captivity, especially South American species. Excellent reviews on the anatomy, biology and ecology of lizards can be found in the references.[3,4,10,12,15]

BIOLOGY AND CAPTIVE MANAGEMENT
Flavio de Barros Molina

BIOLOGY

Lizards vary in body size from a few centimeters (small geckos such as *Coleodactylus* spp.) to about 3 m (*Varanus komodoensis*). They may be found on all continents, except Antarctica, from sea level to high elevations in mountains, and in habitats that vary from tropical rain forests to savannas and deserts. Most species are arboreal or terrestrial, but a few are fossorial. Species such as *Dracaena* spp. and *Crocodilurus lacertinus* are well adapted to swimming in rivers and lakes. *Amblyrhynchus cristatus,* from the Galapagos Islands, swims and dives in the ocean. There are diurnal and nocturnal species.

Lizards are ectotherms. Many species maintain body temperatures of 32–38°C during periods of activity, and some may reach 40°C.[10,12] During these periods they alternate basking behavior with foraging and other behaviors.[2,12] Climatic conditions obviously affect thermal behavior and body temperature, which are also affected by such internal factors as nutritional status, size, infection, and reproductive state.[12] Thermal behaviors may vary in a continuum gradient from thermal conformers to thermal regulators.[10]

Lizards may be herbivorous, insectivorous (80%),[12] carnivorous (*Crocodilurus lacertinus, Neusticurus* spp., and *Dracaena* spp.), or omnivorous (*Tupinambis* spp. and *Ameiva ameiva*).[3] Some lizards are primarily herbivorous (*Iguana iguana*), but may also take insects, bird eggs, and carrion.[3] *Amblyrhynchus cristatus* feeds only on marine algae.[10,12] Lizards may be classified as sit-and-wait predators, cruising foragers, or widely foraging predators.[12] Foraging modes are related to anatomical, physiological, ecological, and reproductive characteristics.[12]

Reproductive modes vary from oviparity to viviparity.[10,12] Clutch size is positively related to female size.[10,12] Temperature-dependent sex determination is known to occur.[14] Parthenogenesis has been reported for species in six families (Teiidae, Lacertidae, Gekkonidae, Chamaeleonidae, Agamidae, and Xantusiidae), but is probably even more widespread.[12] Parental care is discussed by Shine.[13]

CAPTIVE MANAGEMENT

Many species show sexual dimorphism related to size, form, and/or color. Males of *Tupinambis merianae* are bigger than females and have prominent, strong jaw muscles. Only males of *Basiliscus plumifrons* have prominent cranial, dorsal, and caudal crests. Males of *Tropidurus hispidus* have a black spot on the preanal plate and ventral portion of the thighs. Males of *Hoplocercus spinosus* have a black spot on the belly. Other methods for sexing lizards consist of looking for the presence of enlarged femoral or preanal pores and hemipenile bulges, all characteristics of males; manual eversion of hemipenes (a difficult technique); and sex probing (another difficult technique).[5]

Terrariums are satisfactory for housing small- to medium-sized lizards (e.g., *Mabuya* spp., *Tropidurus* spp.; and *Ameiva ameiva*) or young of larger species (e.g., *Tupinambis merianae* and *Iguana iguana*). Fiberglass terrariums with glass fronts are attractive to the public. Other types may be used outside the public area. Terrariums with wooden floors and sides, glass fronts, and screened covers are satisfactory. Those with wooden floors and screened sides, fronts, and tops have the advantage of being light, a useful characteristic when it is necessary to transfer the terrarium to an outside sunny area every morning. Aquariums with screen tops only may also be used when transfer to a sunny area is unnecessary.

Dimensions of the terrarium will depend on the number and size of specimens to be housed. De Vosjoli[5] suggests that the terrarium must have a length of at least four times the total length of the largest lizard and a width of at least one-and-one-half times that measurement. In many circumstances he recommends larger terrariums. He also recommends that the sum of the total lengths of all specimens to be housed must be less than three-fourths of the length of the terrarium. Many lizards may show territorial aggression and should be kept in large terrariums and at low densities. Adults of larger species are best housed in large outdoor (or indoor) enclosures.

Substrate should be selected according to the ecological characteristics of the species. Sand, gravel, soil, or a sand/soil mix are good options. A leaf litter may be simulated with dead leaves. Terrariums/enclosures must have rocks, logs, branches, plants, and shelters. Environmental diversification is important for lizards.[2] Semiaquatic species, such as *Crocodilurus lacertinus* and *Dracaena paraguayensis,* need a large water container or a small pond.

Dominant lizards may prevent others from having access to a basking site, so basking areas must be designed according to the number of lizards involved. Rocks or branches especially positioned under the sun are excellent basking sites. Indoor enclosures/terrariums must be supplied with heaters in order to create a thermal gradient. Again, rocks or branches specially positioned under the heat source are excellent basking sites. Most diurnal lizards must always have access to adequate microenvironments to thermoregulate. At São Paulo Zoo (Brazil) many species of South American lizards are maintained in indoor terrariums kept at a room temperature that varies from about 24 to 28°C during the daytime and from 22 to 26°C during the night. Relative humidity varies between approximately 60 to 80%. Early in the morning these terrariums are placed outside (on an almost daily basis), to allow the lizards to bask in the sun.

Lizards maintained in indoor enclosures/terrariums must be periodically exposed to natural unfiltered sunlight or artificial ultraviolet light of appropriate wavelength (UVB) to allow the endogenous synthesis of vitamin D_3.[1,7,8] Gehrmann[8] presents an interesting discussion about the light requirements of captive reptiles (including the detrimental effects of prolonged exposure to UVB from sunlamps).

Most lizard species are insectivorous and will readily accept mealworm larvae, crickets, and cockroaches. Insects have a deficient calcium/phosphorus ratio, and lizard diets must be supplemented with a calcium source[1,7] such as powdered calcium carbonate.[7] The proper calcium/phosphorus ratio in the diets of vertebrates (including reptiles) falls between ratios of 1:1 to 2:1.[6] Prolonged deficiency of calcium in the diet or an imbalance in the calcium/phosphorus ratio can lead to metabolic bone disease, a syndrome extensively reviewed by Fowler.[6] Mealworm larvae must be offered only sporadically because of its high fat content.[1] Frye[7] describes how to rear some invertebrate prey species.

A pinky mouse is a balanced item, which is accepted by many medium to large species. Some lizards, such as *Tupinambis merianae, Ameiva ameiva,* and *Crocodilu-*

rus lacertinus, will accept beef, which must be supplemented with a powdered calcium product, such as steamed bone meal or calcium carbonate. Fresh whole fish (including head, viscera, scales, and bones) are not calcium deficient; however, it is important ensure that the spines and scales will not injure the lizard's digestive tract. An enzyme called thiaminase is found in some species of fish and is activated after death. It may cause a vitamin B_1 deficiency[1,7] Thiaminase may also be found in thawed, frozen fish.[1,7] It is wise to supplement fish with thiamin.[1]

Plant matter, such as fruits and leaves, is part of the diet of several species (*Iguana iguana, Tupinambis merianae,* and *Ameiva ameiva*). Fruits readily taken include banana, papaya, and tomatoes. Leaves include chicory, kale, and cabbage. *Iguana iguana* is especially fond of bean sprouts. Diets containing an imbalance in the calcium/phosphorus ratio, with an excess of phosphorus, must be supplemented with powdered calcium carbonate, bone meal, or a similar calcium-rich material (see examples in Table 7-6 in reference 6).

Lizards are usually fed twice a week. This frequency may be changed according the temperature at which the lizards are maintained and the diet they are fed. A problem that needs special attention is the possible influence of group hierarchy on food access. It is important to ensure that every lizard in a particular group has access to food and is eating a sufficient quantity. Constant access to fresh water is essential.

Eggs to be artificially incubated must be removed from the terrarium/enclosure as soon as possible. After being measured, the eggs should be placed in a plastic container and covered with 1–2 cm of moist vermiculite. A 1:1 ratio of vermiculite and tap water, by mass, is suggested by different authors.[11,14] A closed container must be opened periodically for ventilation. The vermiculite in an open container must be moistened periodically to maintain humidity. The containers should be placed inside an incubator. A Styrofoam box supplied with common lamps and a thermostat works well. At São Paulo Zoo lizard eggs are incubated between approximately 26 to 30°C. Eggs of *Tropidurus hispidus, Tropidurus oreadicus, Polychrus acutirostris, Basiliscus plumifrons, Kentropyx calcarata,* and *Tupinambis merianae* have been successfully hatched. Viets[14] incubated eggs of many species between 21.5 and 35°C (most from 24 to 32°C). Proper methods for incubating reptile eggs are discussed by Packard and Phillips.[11] The eggs must be regularly inspected and rotten eggs removed.

Newborns should be cleaned with tap water to remove small flecks of vermiculite that may obstruct the eyes and nares. Hatchlings should be placed in terrariums and cared for in the same manner as adults.

REFERENCES

1. Allen, M.E.; and Oftedal, O.T. 1994. The nutrition of carnivorous reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 71–82.
2. Avery, R.A. 1994. The effects of temperature on captive amphibians and reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 47–51.
3. Avila Pires, T.C.S. 1995. Lizards of Amazonia (Reptilia: Squamata). Zoologishe Verhandelingen, 299:1–706.
4. Davies, P.M.C. 1981. Anatomy and physiology. In J.E. Cooper and O.F. Jackson, eds., Diseases of the Reptilia, Vol. 1. London, Academic Press, pp. 9–73.
5. de Vosjoli, P. 1994. The Lizard Keeper's Handbook. Lakeside, California, Advanced Vivarium Systems, Inc.
6. Fowler, M.E. 1978. Metabolic bone disease. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 55–76.
7. Frye, F.L. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry. Malabar, Florida, Krieger Publishing.
8. Gehrmann, W.H. 1994. Light requirements of captive amphibians and reptiles. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 53–59.
9. IUCN. 1996. 1996 IUCN Red List of Threatened Animals. Gland, Switzerland, IUCN.
10. Mattison, C. 1989. Lizards of the World. New York, Facts On File.
11. Packard, G.C.; and Phillips, J.A. 1994. The importance of the physical environment for the incubation of reptilian eggs. In J.B. Murphy, K. Adler, and J.T. Collins, eds., Captive Management and Conservation of Amphibians and Reptiles. Ithaca, New York, Society for the Study of Amphibians and Reptiles, pp. 195–208.
12. Pough, F.H.; Heiser, J.B.; and McFarland, W.N. 1996. Vertebrate Life, 4th ed. Upper Saddle River, New Jersey, Prentice-Hall.
13. Shine, R. 1988. Parental care in reptiles. In C. Gans and B.R. Huey, eds., Biology of the Reptilia, Vol. 16. New York, Liss, pp. 275–329.
14. Viets, B.E.; Ewert, M.A.; Talent, L.G.; and Nelson, C.E. 1994. Sex-determining mechanisms in Squamate Reptiles. Journal of Experimental Zoology 270:45–56.
15. Vitt, L.J.; and Pianka, E.R. Eds. 1994. Lizard Ecology: Historical and Experimental Perspectives. Princeton, New Jersey, Princeton University Press.

IGUANA MEDICINE AND SURGERY

Teresa L. Lightfoot

The green iguana (*Iguana iguana)* has a natural range from Mexico through Central and South America. It is arboreal, diurnal, mainly folivorous, and solitary except during breeding season.[11,2,4] These characteristics constitute many of the problems encountered in proper captive management of the green iguana and will be addressed briefly prior to discussion of the medical and surgical aspects of iguana care. These same characteristics make iguanas, though one of the most popular pet reptiles, one of the more difficult to properly maintain in captivity.

Iguanas are most comfortable in trees or shrubs. Keeping iguanas in aquariums or cages at low heights, especially if they are not adapted to human contact, may be stressful. Constructing an enclosure that allows the iguana to ascend branches vertically and rest at a height of over 6 feet is preferred. The rostral abrasion from constant attempts to escape from wire cages will often be prevented once the iguana is supplied with a means to attain a safe and secure height. This may be a difficult requirement for the average pet iguana owner to accept and accomplish.

Iguanas spend most of the day basking in full or partial sun. When they eat, they tend to eat rapidly and return to basking.[7] They are hind-gut fermenters, and require that their body temperatures be high, probably between 36 to 37°C (about 97–98°F) for effective fermentation.[12] Exposure to sunlight also allows sufficient vitamin D_3 production for proper calcium balance. In captivity, maintaining this temperature at a location in the enclosure that is psychologically comfortable for iguanas to occupy is important to their ability to digest food. In other words, it does no good for the temperature to be at 36°C (98°F) in a place in the cage where the iguana refuses to sit. Creating a temperature gradient throughout the cage so the iguana may regulate its body temperature is optimal. If it prefers a location where the temperature never exceeds 25.5°C (78°F), digestion will suffer.

When iguanas were first kept as pets it became apparent that an iceberg lettuce diet was insufficient for health and growth. It was then suggested that young iguanas consume large amounts of animal protein in the form of insects and possibly carrion. This "fact" has been carried through the literature, and many books found in pet stores still recommend that a young iguana's diet consist of a large portion of insects, dog or cat food, and live prey, such as pinky mice. When young iguanas are fed this diet, no apparent adverse effects are immediately noted. In fact, these iguanas often grow extremely rapidly and have a healthy appearance, leading owners, herpetoculturists, and veterinarians to assume that this diet is suitable. It is not until these animals reach the age of 3 to 6 years that the renal failure so often noted becomes rapidly apparent. No controlled studies have been done to definitely link an early high-protein diet with later renal failure, but the clinical incidence is extremely incriminating. The history of many adult iguanas with fatal renal failure includes moderate to high levels of animal protein either still being fed or having been fed for a considerable period during the animal's first years of life. More recent studies of the diets of young iguanas in the wild have shown that little if any animal protein is ingested by neonatal or juvenile green iguanas.[11,2] An occasional insect is devoured, but these may simply be inadvertent occupants of the foliage or fruit that the animal is actually consuming. Even fruits and vegetables with a high calorie content are consumed in small amounts—hence the term folivorous rather than herbivorous.[11,2,12,8]

The solitary nature of iguanas is also a problem. As juveniles they are often tolerant of one another, and the fact that they "stack" on top of each other in their enclosure is often interpreted by the owner as a sign that they enjoy each other's company. Actually, they generally tolerate and are indifferent to one another until one of them becomes sexually mature.[5] Even during this period of tolerance, their sharp claws may puncture the skin and cause abscesses in their cage mates. Owners are often shocked to find iguanas that have peacefully cohabited for years are suddenly fighting when one becomes sexually mature. They may inflict serious wounds within a matter of minutes. It is preferable to house multiple iguanas separately once they have reached the age of 2 or 3 years or reach a length of more than 28 cm.[5]

DISEASE SYNDROMES

Metabolic Bone Disease

Most practitioners who see even a few iguanas are familiar with metabolic bone disease (MBD). Lack of natural sunlight doesn't allow the production of vitamin D_3 necessary for calcium absorption. The provision of ultraviolet (UV) spectrum light (especially UVB) with artificial lights may be sufficient for some iguanas and is definitely recommended if an iguana must be housed indoors.[11] Several factors may contribute to the development of MBD, even when the owner is providing "proper" artificial lighting:

a) Several studies have shown that artificial lights cannot provide the amount and/or width of spectrum present in natural sunlight.[11]

b) Artificial lights may lose their UV spectrum while still emitting visible light. Artificial UV lights should be replaced at frequent intervals (6–9 months).
c) Iguanas in large enclosures, or those that roam free in the house for long periods of time, may not have enough contact with the light source to absorb sufficient UV rays. It is generally recommended that iguanas be within 18–24 inches of the light for effective absorption of the rays.[8]
d) The UV light provided by owners is often filtered through plastic or glass, which removes most of the useful spectrum.
e) The diet may be deficient in calcium or incorrect in the calcium-phosphorous balance.

A young iguana presenting with MBD will generally have one or more of the following clinical signs: (1) pathologic fractures of one or more long bones, with the accompanying soft tissue swelling that is classic of these fractures; (2) softening of the mandible and maxilla ("rubber jaw"); (3) paresis or paralysis of the rear legs; (4) inability to urinate and/or defecate (both 3 and 4 above being caused by pathologic spinal or pelvic fractures); (5) muscle fasciculations (especially toe twitching); (6) anorexia; and (7) eventual death.

Treatment begins with keeper, curator, or client education concerning husbandry and diet. Hospital treatment generally consists of calcium injections (Calphosan Solution; calcium glycerophosphate 5 mg/mL and calcium lactate 5 mg/mL, Glenwood, Inc., Tenafly, New Jersey, USA) at 1.0 mL/kg intramuscularly or calcium gluconate 10% solution, also dosed at 1 mL/kg intramuscularly, once daily until the patient is stable; then, oral supplementation may be implemented. Intravenous injections of calcium gluconate may be given to effect, generally at 1.0 mL/kg slowly. An immediate response is often seen in moribund animals and in those with seizures or severe fasciculations caused by calcium deficiency. Injectable vitamin D_3 may be administered at 200–1000 IU/kg once, along with supportive care, including subcutaneous fluids and tube feeding if anorexia has developed. Rarely do pathologic fractures need coaptation, because the body rapidly produces a fibrous callus that gives the bones great stability (and often gives the owners a false sense of security, thinking that these large appendages must indicate a well-fed and healthy iguana).

Once the acute calcium deficiency has been corrected with injectable calcium, a more rapid return of mineral density to the bones can be obtained with the use of calcitonin (Calcimar; Rhone-Poulene Rorer, Ft. Washington, Pennsylvania, USA) at 50 IU/kg once weekly for two injections.[11] Continued calcium supplementation is especially critical when calcitonin is administered to maintain serum calcium levels. Most iguanas like the sweet taste of calcium glubionate (Neo-Calglucon; Sandoz Pharmaceuticals, East Hanover, New York, USA) at 1.0 mL/kg twice a day, which the owners can give orally at home. The constant use of vitamin D_3 in the oral form, as is found in many commercial reptile mineral supplements, is controversial. The absorption and physiologic utilization of this vitamin given orally has not been well documented, and there may be a danger of vitamin D toxicity with subsequent renal damage.

Most young iguanas with MBD that are still eating respond well to therapy. Those that have become severely anorexic, emaciated, or depressed have a more guarded prognosis. The owners should be warned that during treatment, even when the husbandry has been corrected, more pathological fractures may occur before mineralization of the bone has been reestablished. The long bones tend to remodel well, but mandibular and maxillary deformities may persist for long periods and even worsen with time, because one jaw, usually the maxilla, may grow longer than the other (mandible). MBD may also cause progressive scoliosis of the spine and tail that may worsen with time, despite restoration of the calcium balance.

Abscesses

Abscesses in iguanas differ significantly in potential severity and required duration of treatment from the comparable syndrome in cats. Lacking proteolytic enzymes, the exudate in reptile abscesses is inspissated, and aggressive debridement of what is often a tenacious and vascular capsule must be performed. A single abscess, especially one with a known cause (i.e., cagemate trauma, rostral abrasion, bite wounds from being fed live prey), has a much better long-term prognosis than do multiple abscesses. When an iguana presents with abscesses on several digits, and typically in several other locations such as the rostrum, the knee or elbow joint, and along the tail, it is likely that the animal is septic. The literature states that there may be either a single coelomic cavity abscess or multiple granulomas of the viscera that are "seeding" these abscesses.[2] These internal lesions may not be obvious on gross examination, either during an exploratory procedure or at necropsy. In any case, multiple abscesses require a more guarded prognosis and a longer course of antibiotics than a single abscess. A culture and sensitivity (C&S) of the abscess lining prior to flushing is important, because these animals are septic and will often require a long-term (6–12 weeks) course of antibiotics. Besides the commonly encountered *Pseudomonas, Proteus, Klebsiella,* and assorted other species of gram-negative bacteria, and *Staphylococcus* spp., other potential pathogens include yeasts, fungi, acid-fast bacteria (both typical and atypical mycobacteria), and anaerobic bacteria.

Parenteral antibiotics should be selected by the C&S results. Though often enrofloxacin (Baytril) is efficacious as indicated by C&S, the volume required for administration is quite large (5–10 mg/kg every 24–48 hours). If long-term administration is needed, amikacin (5 mg/kg initial injection, then 2.5 mg/kg every 72 hours), along with a synergistic broad spectrum β-lactam antibiotic, such as piperacillin (100–200 mg/kg every 24–48 hours), Naxcell (ceftiofur; 4 mg/kg every 24 hours) or Fortaz (ceftazidime; 20 mg/kg every 72 hrs), may be more effective.

Most reptile antimicrobial dosages are empirical. Though the volume of the β-lactam may be quite high, there is a much lower incidence of tissue irritation than with Baytril. Also, preserving the quinolones for use when other antibiotics are ineffective is an effort that everyone should promote. Daily flushing of the abscess with a dilute chlorhexidine solution will both cleanse the area and prevent the typical premature closure of the skin that inevitably leads to recurrence of the abscess. Some work has been done with methylmethacrolate beads impregnated with antibiotics. The use of umbilical tape, saturated with an antibiotic and placed as a Penrose drain, may also help to prevent premature closure of the skin and also provide a steady release of antibiotic. The addition of Wydase (hyaluronidase) to the flushing fluid in reptile abscesses, as in rabbit abscesses and nasal flushes for sinusitis in birds, shows considerable promise.

Thermal Burns

Most thermal burns are caused by hot rocks with focal "hot spots", heating pads in the cage, or lights with which the dorsum of the animal can come in contact. Generally, the more severe burns are seen in one of two situations: (1) Iguanas kept in a cool (outdoor) environment that the owner osr keeper attempts to ameliorate by adding lights or heaters. The iguana then comes too close to the heat source as its body temperature becomes extremely low. The iguana's receptors and reaction times to heat are greatly slowed by suboptimal core body temperature. (2) Debilitated animals, whose illness prevents them from moving away from the heat—either because of decreased perception of heat or decreased ability to react to that perception.

Treatment will vary according to the severity and extent of the burns. Minor superficial burns may require only application of a cream, such as Silvadene or Bactoderm and maintaining the animal on clean paper towels or newspaper (if the ventrum is the area affected). Severe burns may necessitate extensive debridement (often in stages), bandaging, supportive care by tube feeding, fluids if needed, and parenteral antibiotics when the burns are deep and/or multiple.[11,6]

Parasitism

Iguanas will often present with one or more parasites of the gastrointestinal tract.

NEMATODES Nematodes are the most commonly encountered parasites on direct fecal or flotation examination. Ascarids, hookworms, strongyloids, and pinworms are the most frequently detected, all of which respond to fenbendazole (Panacur). Dosage varies, but generally is 25–50 mg/kg, repeated in 2–3 weeks. Doses of 100 mg/kg have often been recommended in publications, and it appears that the high safety factor enjoyed with this drug in mammals also applies to iguanas. Some species of snakes, however, may be adversely affected by these higher dosages.[9] All of these parasites have a direct life cycle, so reinfection is likely.

FLAGELLATES Much debate still occurs as to the pathogenicity of protozoal parasites in reptiles. Many clinicians consider low numbers of flagellated protozoans to be normal flora in an asymptomatic iguana.[1] Likewise, pinworms occur in iguanas with few if any clinical signs. For excellent coverage of reptile parasites, refer to Roger Klingenberg's *Understanding Reptile Parasites*.[9] Flagellated protozoans can be difficult to speciate. Often the determination of whether to treat for these protozoa is based empirically on the number and types seen on fecal smears and the consistency of the stool. A system for quantifying these organisms may be helpful. Generally, on a direct smear, they can be noted as rare, occasional, 1+, 2+, and up to 4+ as the highest concentration and then identified as to the number of different types of protozoa seen. Treatment with Flagyl at dosages between 25 mg/kg and 125–250 mg/kg is recommended. No treatment should be given unless there is significant overgrowth. Iguanas seem to be tolerant of Flagyl, despite the fact that the major bacteria in their hind-gut fermentation appear to be *Clostridium* spp.[12] Flagyl at a single dose of 75–100 mg/kg is administered, and the feces should be retested in 1–2 weeks to see if a repeat dose is needed. Often, with good hygiene, the numbers will have reduced dramatically, and no further treatment is required.

TAPEWORMS Cestodes usually require an intermediate host and are rarely seen in the folivorous iguana. They are much more common in water snakes, water monitors, and aquatic chelonians. When encountered, treatment is with Droncit at 5–8 mg/kg, orally or subcutaneously.

COCCIDIA Coccidia are not as common in iguanas as in other lizards, in which pathogenicity may range from none to severe. When encountered, treatment with either sulfadimethoxine (Albon) or sulfamethazine/trimethoprim (SMZ-TMP) is efficacious. It is essential to maintain hydration. Unlike monitors and bearded dragons, iguanas are usually not difficult to clear of coccidial infections. One recommended treatment regimen is Albon at 50 mg/kg every 24 hours for 3 days, off 3 days, then repeated for 3 days.[9]

ECTOPARASITES Both ticks and mites may be found on iguanas, although fortunately they are not nearly as common in green iguanas as they are in snakes. The use of Ivermectin in reptiles is controversial. Ivermectin has so revolutionized treatment of many endo- and ectoparasites in domestic and other exotic species that it was assumed by many that it would be similarly useful in reptiles. This has not proven to be true for the following reasons: (1) Ivermectin should never be used in chelonians, because it may cause central nervous system (CNS) symptoms and death, even at low dosages, in many species of turtles and tortoises; and (2) the efficacy of Ivermectin is not nearly that seen in mammals against either external parasites (mites and ticks) or nematodes. Generally, Ivermectin is reserved for filarial worm infection. Recently it has been recommended as a dilute spray for mite and tick infestation (0.5 mL, 5 mg of Ivomec/quart of water, used once, or repeated in 1–2 weeks).[9]

Thorough cleaning of the cage and substrate is essential to ensure effectiveness with any mite treatment. No-Pest Strips may cause toxicity, especially in underventilated and overstressed animals, but are still used by many herpetoculturists. The use of olive oil to coat a reptile and suffocate mites has been advocated as the least potentially toxic of all treatments.

Regardless of the method chosen to treat severe mite infestation, adverse reactions are common. Reptiles heavily infested with mites were either severely debilitated initially, or had become so as a result of the infestation. If they die during treatment for the mites, it may well be the underlying disease or diseases that are actually the cause of death.

SURGERY

Celiotomy

A large ventral midline vessel suspended from the linea alba by a short ligament is present from the umbilicus caudal to the pelvis. A ventral midline incision may be used if the vessel is either ligated or reflected, but this requires very delicate dissection. Generally, a paramedian approach for a celiotomy is preferred to avoid this vessel. The absence of a ventral midline incision also prevents the incision from being in direct and constant contact with the substrate during healing. Sutures are left in place for prolonged periods, often 4–8 weeks.

FOREIGN BODY INGESTION This is a common presentation in iguanas. Often it is discovered on screening radiographs, and it is not easy to determine whether the foreign body is a primary problem or an incidental finding. Dense metal objects (e.g., pennies, screws, and jewelry) are commonly ingested. Although on radiographs the object may appear to be in the stomach, most of these are actually in the cecum, which is positioned in the cranial portion of the abdomen. Removing foreign objects through the oral route may be attempted, but is often unsuccessful. The decision as to whether to surgically remove the object must be made in light of the overall picture, which should include a complete blood count (CBC), serum chemistries, heavy metal assays, and the presence of clinical gastrointestinal signs. Iguanas seem able to tolerate somewhat high levels of serum zinc (from ingestion of a galvanized screw for example), without becoming toxic.

GRAVID FEMALE IGUANAS Dystocias in iguanas in North America usually occur between November and June. Typically, an owner will report an iguana with an initial restlessness, irritability, or hyperactivity, often accompanied with or followed by decreased appetite, possible lethargy, and an enlarging abdomen. After the ova leave the ovary and move along the oviduct, they may be palpable through the abdominal wall. The question is when is dystocia present? There is no absolute answer, but some guidelines may help. Generally, if radiographs show that the female has ovulated and she is anorectic or has shown no interest in the nesting location provided for more than 2–3 weeks, intervention should be considered. Follicles still on the ovary may occupy the entire abdomen, both radiographically and on palpation. To determine that ovulation has occurred (i.e., that the follicles have left the ovaries) radiographic appearance of "stacking" of the eggs along the oviduct can be seen. Ideally, serum chemistries and a CBC will help determine whether the calcium level is adequate for egg laying and rule out any concurrent infection. Although oxytocin is not the most effective hormone to induce oviposition,[11] it is the most accessible; in my experience, it has about a 50–75% success rate when given at the proper time in the cycle to a female with a "calcium-primed" uterus and an acceptable substrate available for laying. If attempts at induction of egg laying are not successful and supportive care (fluids, tube feeding, and calcium) for another week or two still fails to induce oviposition, it may be necessary to perform an ovariohysterectomy. For details of this procedure, see references 10 and 11.

TAIL AMPUTATION Whether caused by infection or trauma, if the distal portion of an iguana's tail has become avascular, that section should be amputated. Generally, a "snapping" of the tail—like snapping a green bean—at the point of least resistance above the damaged tissue, will result in minimal if any bleeding and good regrowth. A single loose mattress suture may be placed in the tail to narrow the gap of exposed tissue, and a light antibiotic wrap may be applied for the first week or two. Once a slight scab has formed so fecal contamination is unlikely, the mattress suture and bandage should be removed to allow the tail maximal regrowth. If the tail skin is sutured with the skin edges opposed, there will be more difficulty in regrowth. A regrown tail never has the same pattern as the original, but some regenerated tails will obtain a length that is one-half to three-fourths of normal and serve the same functions as the original tail.

MISCELLANEOUS PRESENTATIONS

RENAL FAILURE As discussed earlier, this is a heartbreaking and often acute disease of large, strong, apparently healthy adult iguanas. What combination of excessive protein intake, dehydration, parenteral vitamin D_3 administration, visceral gout, and bacterial glomerulonephritis are involved in this syndrome is yet to be defined. Most of these iguanas present with a past history of rapid growth and strength and a significant animal protein intake. Often the kidneys, which should lie within the pelvic inlet, have become large, palpable, and painful masses hanging down within the abdominal cavity. Blood chemistries generally show an increased phosphorus level, anywhere from a mild increase of 7–8 mg/dL to 40 mg/dL, or greater. A lower-than-normal calcium level is often noted. Uric acid tends to remain normal, except in terminal cases.

Treatment at this stage may be frustrating. Many of these iguanas present acutely ill, with scleral injection (probably resulting from blood pressure changes associated with renal shutdown and hypertension) and pharyngeal edema, which may impede swallowing. Administration of parenteral fluid when renal function is severely compromised may exacerbate edema of pharyngeal and soft tissues. Aluminum hydroxide (Amphojel) administered orally may aid by binding to and lowering serum phosphorus. Diuresis is needed to treat renal failure. Unfortunately, loop diuretics don't work because reptiles lack a loop of Henle in their nephrons. Some success has resulted from administration of mannitol intravenously, but mannitol is expensive, and daily administration is cumbersome. Acetazolamide (Diamox), a carbonic anhydrase inhibitor, has been tried in two iguanas at my hospital. Both animals responded to therapy, but neither has received this treatment alone, nor has either iguana maintained normal renal function (as indicated by serum phosphorus levels) when therapy was discontinued. Whether the reptile nephron responds with increased HCO_3 excretion when this drug is administered is unknown. If an iguana presents with a low enough serum calcium to be exhibiting neuromuscular signs (fasciculations, seizures, cloacal prolapse), administration of injectable calcium intravenously or intramuscularly is indicated, though the danger exists that the injected calcium will bind with the excess phosphorus and cause soft tissue mineralization.

The severe swelling of the kidneys in cases where they are abdominally palpable has led some reptile veterinarians to try a bolus of a short-acting glucocorticoid, often with an initial dramatic decrease in renal swelling. The long-term benefit of this treatment is less certain (Ross Prezant, personal communication).

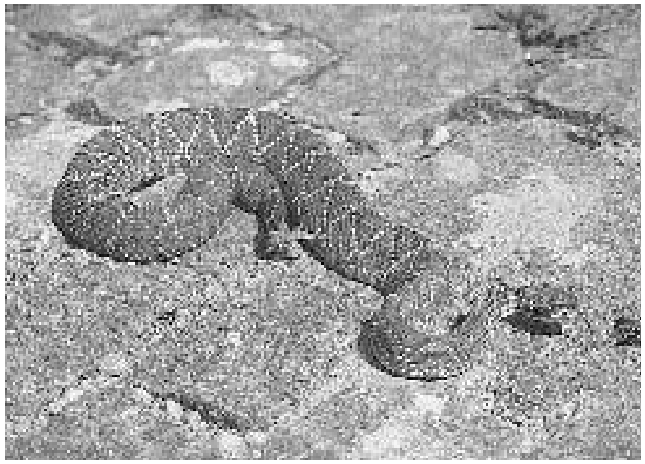
CBCs should *always* be run concurrently with serum chemistries, because active glomerulonephritis is a distinct possibility and treatment of the underlying infection may be critical. Electrolytes should be included in the chemistry panel to allow proper selection of fluids. Often, a renal biopsy will help with both treatment and prognosis.

CYSTIC CALCULI Large bladder stones are commonly encountered in iguanas as well as tortoises. Stones may be indicated by the appearance of blood in the urine, straining to urinate, or may be an incidental finding by the owner or examining veterinarian on palpation. Removed stones should be cultured, because a high number are positive for *Salmonella* or other gram-negative bacteria(Karen Rosenthal, personal communication, 1996).

SALMONELLA AND ZOONOTIC CONCERNS Many recent newspaper articles have been written concerning the danger of transmission of salmonellosis from reptiles to humans. Though a high percentage of reptiles may carry *Salmonella* bacteria, unless hygiene is poor or the animal is stressed, clinical salmonellosis may never develop, become contagious, or be detectable by culture. However, households with children and/or immune-compromised individuals should be warned of this increased risk of keeping reptiles.

REFERENCES

1. Barnard, S.M.; and Upton, S.J. 1994. A Veterinary Guide to the Parasites of Reptiles, Vol. 1. Protozoa. Malabar, Florida, Kriegar Publishing.
2. Barten, S.L. 1993. The medical care of iguanas and other common pet lizards. Veterinary Clinic of North America Small Animal Practice (Exotic Pet Medicine I) 23(6):1213–1250.
3. Beynon, P.H.; Lawton, M.P.C.; and Cooper, J.E. 1994. Manual of Reptiles. Ames, Iowa, Iowa State University Press, British Small Animal Veterinary Association.
4. Boyer, T.H. 1993.Iguana care. In A Practitioner's Guide to Reptilian Husbandry and Care. Lakewood, Colorado, AAHA, pp. 33–44.
5. Dugan, B. 1982. The mating behavior of the green iguana, *Iguana iguana*. In G.M. Burghardt and A.S. Rand, eds., Iguanas of the World, Their Behavior, Ecology, and Conservation. Park Ridge, New Jersey, Noyes Publications, pp. 320–341.
6. Frye, F.L.; and Townsend, W. 1993. Iguanas: A Guide to Their Biology and Captive Care. Malabar, Florida, Kreiger Publishing, pp. 85–92.
7. Iverson, J.B. 1982. Adaptations to herbivory in iguanine lizards. In G.M. Burgardtand A.S. Rand, eds., Iguanas of the World, Their Behavior, Ecology and Conservation. Park Ridge, New Jersey, Noyes Publications, pp. 60–76.
8. Johnson-Delaney, C.A. 1996. Exotic Companion Medicine Handbook for Veterinarians. Lake Worth, Florida, Wingers Publishing, p.5.
9. Klingenberg, R.J. 1993. Understanding Reptile Parasites: A Basic Manual for Herpetoculturists and Veterinarians. Lakeside, California, Advanced Vivarium Systems.
10. Lightfoot, T.L.; Bartlett, L.W.; Harrison, G.J.; Manarino, R.; and Griffin, C.W. 1999. Exotic Animal Surgery CD-ROM. Lake Worth, Florida, Wingers Publishing.
11. Mader, D.R. 1996. Reptile Medicine and Surgery. Philadelphia, W.B. Saunders.
12. McBee, R.H.; and McBee, V.H. 1982. The hindgut fermentation in the green iguana, *Iguana iguana*. In G.M. Burghardt and A.S. Rand, eds., Iguanas of the World, Their Behavior, Ecology and Conservation. Park Ridge, New Jersey, Noyes Publications, pp. 77–83.



5
Class Reptilia, Order Squamata (Ophidia): Snakes

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BIOLOGY AND KEEPING SOUTH AMERICAN SNAKES IN CAPTIVITY

Luiz Roberto Francisco

INTRODUCTION

Many reptile species are found in South America, of which snakes are an important component. A great variety of snake species occupy many various geographical regions, making special conditions necessary for the care of certain species in captivity. However, care of the most common species will be emphasized in this discussion.

BASIC HUSBANDRY

Snakes, like many other reptiles, are able to adapt to captivity if basic needs of feeding, sanitation, temperature, and wetness are met. It is important to understand environmental conditions in their native habitats in order to approximate them in captive enclosures.

Reptiles are ectothermal animals, which means they depend on environmental temperatures to regulate such functions as feeding, assimilating nutrients, and breeding. They need an external source of heating, which in nature is the sun. Body temperature is controlled by varying the amount of skin surface exposed to the sun. They are able to accelerate heat gain through peripheral vasodilatation and by retaining small quantities of metabolic heat. Heat gain is retarded by panting.

In captivity, the ideal temperature may be reached through the use of heaters or infrared light bulbs. A common mistake is made when the terrarium is uniformly heated, producing a greenhouse effect that may kill a snake from hyperthermia. Heat must be supplied in such a manner as to produce a gradient, so the snake is able to select optimal conditions.

Snakes also should be exposed to ultraviolet radiation, which is necessary to produce vitamin D_3 for bone development. Periodic exposure to the sun or ultraviolet light bulbs that emit the proper wave length of the spectrum is necessary.

Another factor that must be carefully considered in keeping snakes in captivity is humidity. Tropical species should be maintained at slightly less than 80% humidity, whereas species from deserts require less than 30% humidity. A correct humidity gradient is fundamental to proper skin shedding. Incomplete shedding may result in retention of eye caps. A management tool is to place the snake in a closed cloth bag that is then placed into a flat tray of warmed water. The snake should soak within the bag for about an hour, during which time it will attempt to escape, and by so doing will rub off the moistened skin.

An appropriate humidity inside the terrarium may be obtained by using plant water sprayers, water vats with aerators to bubble the water to increase evaporation, or through electrical water sprayers regulated with a timer to spray at selected times.

The cage should be cleaned regularly to remove wastes. The warm temperature and moist conditions foster bacterial growth and the development of mold.

Outdoor Enclosures

Captive snakes are best maintained in indoor terrariums, rather than in outdoor enclosures. Unfortunately, outdoor enclosures are common in warmer regions of the continent, particularly for anacondas (*Eunectes murinus*) and boas (*Boa constrictor* ssp.) which are often kept together. The rationalization for this custom is that these snakes are usually native to the region. This argument only considers temperature, without concern for other important factors, such as the significantly larger anacondas taking advantage of the boas. Large individuals may crush smaller ones of the same or different species or may not allow the smaller snakes to eat. Males may fight with each other when overcrowded.

Feeding in Captivity

Snakes are carnivorous so must necessarily be fed animals. Prey may be offered alive, but there are inherent dangers in doing so, because uneaten prey, if a rodent, may attack and injure the snake. Many species may be trained to accept dead prey. Frozen prey may be easily thawed in a microwave oven and given to the snake, but it must not be heated above 37°C. In captivity snakes usually are fed mice, rats, guinea pigs, rabbits, and poultry. Juvenile snakes consume the same types of prey as adults, only needing smaller specimens. The Boidae prefer rodents. Poultry may be offered to anacondas. The Viperidae eat rodents. The Colubridae have various species preferences. Some eat birds (*Spilotes pullatus*) and rodents, others such as *Waglerophis merremii* eat amphibians. *Hydrodinastes gigas,* which lives in marshy regions, feeds primarily on amphibians native to the area, although it may also hunt fish, small birds, and mammals. Some species prey mainly on fish (*Liophis miliaris*), and some eat other snakes (*Clelia clelia*).

When an individual refuses to eat, forced feeding may be required. Forced feeding is a last resort and must never be a usual practice, because it is stressful for the snake and may be dangerous for the handler if the snake is venomous.

SNAKE FAMILIES

South American snakes maintained in captivity are from four snake families: Colubridae, Boidae, Viperidae, and Elapidae.

Colubridae

About 75% of the world's snakes are in the family Colubridae and are regarded by many authors to be the most recently evolved snakes because they show no traces of rear limbs. As may be expected with this large group, the Colubridae fill many different habitats and vary widely in characteristics. One of the most interesting is the rat eater (*Spilotes pullatus*), widespread throughout the continent, but found mainly in eastern Brazil. It reaches 3 m in length, and has a flat-sided body. Because of its large size and attractive color (yellow and black), it is a popular exhibit species. Another large species is the black indigo snake (*Drymarchon corais*), which may grow to more than 2 m long. Many Colubridae species are kept in collections of zoological institutions and may be easily sustained with laboratory mice (Table 5.1).

Boidae

Species of Boidae are among the largest snakes in the world, some growing to be 10 m length, although most of the species are only a little more than 1 m long. They characteristically have pelvic girdle remnants and vestigial hind limbs in the form of spurlike extensions near the vent. They are found in tropical and subtropical regions.

Boidae require maintenance of optimal conditions in captivity. Because of its aggressiveness and large size,

TABLE 5.1. Species of Colubridae and conditions of keeping in captivity

Species	Feeding	Temperature	Humidity
Spilotes pullatus	Mice, birds, cavies	25–28°C	50–70%
Drymarchon corais	Mice, birds, cavies	27–28°C	65–70%
Hydrodinastes gigas	Toads, frogs and mice	25–28°C	70–80%
Boiruna maculata	Snakes	26–27°C	50–70%
Waglerophis merremii	Toads and frogs	25–27°C	70–80%
Mastigodryas bifossatus	Toads, frogs and mice	25–27°C	70–80%

TABLE 5.2. Species of Boidae and conditions of keeping in captivity

Species	Feeding	Temperature	Humidity
Boa constrictor constrictor	Cavies, birds	26–28°C	60–70%
Boa constrictor amarali	Cavies, birds	26–28°C	50–60%
Epicrates cenchria cenchria	Mice and rats	26–28°C	60–70%
Epicrates cenchria alvarezi	Mice	24–27°C	50–70%
Corallus caninus	Mice and birds	27–30°C	70–80%
Corallus hortulanus	Mice and birds	25–30°C	50–80%
Eunectes murinus	Rats, rabbits, and hens	27–30°C	70–80%

TABLE 5.3. Species of Viperidae and conditions of keeping in captivity

Species	Feeding	Temperature	Humidity
Bothrops jararaca	Mice	26–28°C	40–70%
Bothrops jararacussu	Mice and rats	26–28°C	60–70%
Bothrops alternatus	Mice and rats	26–28°C	60–70%
Bothrops cotiara	Mice	24–27°C	50–70%
Crotalus durissus terrificus	Mice and rats	27–30°C	≤ 10%
Lachesis muta	Mice and rats	25–30°C	60–80%

the anaconda requires a large cage. Handling an anaconda should only be performed by two or more people, and such handling should not be done when the snake is active (night). Feeding must be controlled to avoid obesity.

Among snakes kept in captivity, probably boas (*Boa constrictor* ssp.) are the most common. It is difficult to find a zoo in South America that does not exhibit boas. Many Boidae species are widespread throughout South America (Table 5.2).

Viperidae

Vipers are venomous. A distinctive characteristic is the presence of heat-sensing pits caudal to the nostrils, used to detect prey. Vipers may be crepuscular or nocturnal. They feed mainly on small mammals. They are represented by *Bothrops, Bothriechis, Bothriopsis, Lachesis, Porthidium,* and *Crotalus* genera in South America.

All species may be maintained in captivity if their specific requirements are met (Table 5.3). Handling must be done safely to avoid envenomation. Special care must be taken with species such as the Bushmaster (*Lachesis muta*), which is large, fast, and extremely aggressive, as well as having powerful venom. Its safe handling requires observance of all possible precautions.

Elapidae

South American species of Elapidae include the sea snake, *Pelamis platurus,* and the true coral snakes of the genus *Micrurus* (Table 5.4).

TABLE 5.4. Species of Elapidae and conditions of keeping in captivity

Species	Feeding	Temperature	Humidity
Micrurus frontalis	Mice and snakes	24–26°C	40–60%
Micrurus corallinus	Snakes	24–26°C	40–70%

Coral snakes inhabit desert areas and bushy jungles. They have beautiful color patterns (red, black, and yellow), and may be mistaken for nonvenomous colubrids with similar color patterns. In some cases, it requires a dental examination to identify the species. Intensive melanism is not uncommon, which is another factor that leads to a mistaken species identification. Coral snakes have a powerful neurotoxic venom and are extremely dangerous to humans, although most species are not highly aggressive. Coral snakes are not good exhibit snakes because they choose to burrow or hide all the time.

REFERENCES

1. Breen, J.F. 1974. Encyclopedia of Reptiles and Amphibians. Neptune City, New Jersey, T.F.H. Publications. p.6.
2. Campbel, J.A.; and Lamar, W.W. 1989. The Venomous Reptiles of Latin America. Ithaca, New York, Cornell University, p. 425.
3. Francisco, L.R. 1997. Répteis do Brasil: Manutenção em Cativeiro. São José dos Pinhais, Gráfica e Editora Amaro, p. 208.

4. Frye, F.L. 1991. Reptile Care: An Atlas of Diseases and Treatments. Neptune City, New Jersey, T.F.H. Publications, p. 637.
5. Geus, A. 1992. The Proper Care of Snakes. Neptune City, New Jersey, T.F.H. Publications, p. 256.
6. Grantsau, R. 1991. As Cobras Venenosas do Brasil. São Bernardo de Campo, Bandeirante, p. 121.
7. Mader, D.R. 1996. Reptile Medicine and Surgery, Philadelphia, W.B. Saunders, p. 512.
8. Mehrtens, J.M. 1987. Living Snakes of the World. New York, Sterling Publishing, p. 480.
9. Obst, F.J.; Richter, K.; and Jacobe, U. 1988. The Completely Illustrated Atlas of Reptiles and Amphibians for the Terrarium. Leipzig, T.F.H. Publications, p. 831.

OPHIDIA—RESTRAINT, ANESTHESIA, MEDICINE

Kathleen Fernandes Grego

RESTRAINT OF SNAKES

Physical Restraint

Several techniques for handling and restraining snakes safely have been published.[1,5,6,18,23,26]

Poisonous snakes should be handled with extreme caution, only by experienced personnel, and never by one person alone. The head of an aggressive snake may be gently pinned with a hook just behind the head to facilitate initial handling, and its head should be held securely, laterally, with the thumb and forefinger, holding the posterior part of the quadrate bone. The free hand should grasp the snake's body. Lutz's snare is a good alternative for handling and examining aggressive and poisonous snakes without danger for the handler or harm to the snake. More than one person is required for adequate restraint of large constrictors.

Plastic tubes of various sizes make excellent tools for handling many species of poisonous snakes.[5,23] The use of tongs, hooks, portable squeeze boxes, plastic tubes, and Lutz's snares, along with appropriate chemical restraint, is preferred when treating poisonous species. Physical restraint permits examination, administration of medication, and even minor surgery.

Chemical Restraint

Injectable and inhalant agents have been used clinically with a wide margin of safety, making it possible to perform a variety of surgical procedures on reptiles. It must be remembered that metabolic rates are temperature dependent in ectothermic animals and that the absorption and excretion of anesthetics are prolonged in comparison with mammals; thus, supplemental heat should be provided.

Anesthetic induction, maintenance, and recovery should be performed within the preferred temperature range for a given species. If the range is not known, a temperature of 26–32°C is adequate.[2] Fasting reptiles for 24–48 hours before an anesthetic is administered is indicated to prevent putrefaction of ingesta as a result of decreased gastrointestinal transit time. In snakes, a large food item in the stomach may also compress the lungs.

The choice of agent for chemical restraint in reptiles will depend upon the purpose. Some agents are more appropriate for short or nonpainful procedures such as physical examination, transportation, or diagnostic sampling, and others are indicated for general surgical anesthesia.

INJECTABLE ANESTHETICS Reptiles have a relatively slow metabolism, and the effects of injectable agents are usually prolonged with induction time requiring hours and total recovery as long as several days. Injectable anesthetics require little equipment, but once given, the effects and depth of anesthesia cannot be controlled. The effects of injectable anesthetic agents that are excreted unchanged by the kidney are diminished when injected into the caudal half of the snake's body, because of the renal portal system.

- **Ketamine—**The response to ketamine is dose dependent, but also varies with the species and the individual. Induction occurs in 10–30 minutes, and recovery requires from 24 to 96 hours. Even with a surgical level of anesthesia, some animals will exhibit serpentine movements.
- **Tiletamine plus zolazepam—**The action of tiletamine is similar to that of ketamine, but it is two to three times more potent, which lowers the volume needed. Because of the rapid onset of effect, tiletamine plus zolazepam may be most useful as a tranquilizing agent or an induction agent for maintenance with inhalant anesthetics.
- **Xylazine hydrochloride—**Xylazine has been used in conjunction with ketamine hydrochloride to produce an acceptable level of surgical anesthesia. The induction time is about 30 minutes.

INHALANT ANESTHETICS Inhalant anesthesia has become the standard of practice for reptiles. The recovery is usually quick once the anesthetic gas is discontinued, and an accurate patient weight is not critical. Endotracheal intubation and assisted ventilation are recommended, but care must be taken to avoid rupture of the lung when positive pressure ventilation is applied.

Whichever anesthetizing method is used, balanced electrolyte solutions may be administered at a rate of 5 mL/kg/h intravenously (IV), intracoelomically (ICe), or subcutaneously (SC), when procedures take more than 1 hour. Recovery should occur in a warm environment. Doxapram at 5 mg/kg IV may be used to stimulate respiration.

CARBON DIOXIDE ANESTHESIA Inhalation of pure carbon dioxide CO_2 is currently being used as an anesthetic for snakes because of its efficiency and low cost. It is used for milking (obtaining venom) poisonous snakes and to accomplish minor nonpainful procedures. The induction time ranges from 4 to 10 minutes after the snake has inspired CO_2. Short-term anesthesia with CO_2 is safe and not traumatic for pit vipers.[28] CO_2 has also been used safely in snakes of the Boidae and Colubridae families.

SURGERY

Basic knowledge of the anatomy and physiology of the particular species is important before undertaking surgery. Antibiotic therapy is indicated only when infection is already present. The anesthetized snake should be firmly strapped to the table with adhesive tape. The surgical approach will depend upon the organ system involved. Povidone iodine and chlorhexidine are disinfectants recommended to sanitize the skin.

The incision into the snake's body must be done at the junction of the lateral and ventral scales; an incision at this site is easier to keep clean because it is not in direct contact with the substrate during the postoperative period.[4] Longitudinal incisions heal faster than transverse incisions. Chromic catgut is contraindicated for use in snakes, and synthetic absorbable materials must be removed from the skin 3–4 weeks after surgery. Suture removal must be delayed until the subsequent ecdysis because the activity of the dermis and epidermis during ecdysis seems to promote healing. An everted suture pattern for skin closure avoids the opposition of scales that may delay wound healing.

Celiotomy

Laparotomy or celiotomy in snakes provides access to all the coelomic organs and is indicated for several conditions, such as removal of retained eggs or young from the oviducts, gastrotomy, investigation of diseases of the abdomen, organ biopsies, and surgery. Closure is generally done in two layers: one in the body wall (simple continuous) and the second in the skin (everted pattern).

Cesarean Section

Sometimes a gravid snake has difficulty or is unable to lay her eggs or deliver young. If not treated promptly, dystocia may lead to oviduct prolapse, peritonitis, and death of the female. X-rays may confirm dystocia. If massage fails to move the egg or fetus down the oviduct, a celiotomy must be performed. Once the coelomic cavity has been entered, the oviducts are incised, their contents removed, and the incision closed with absorbable materials in an inverted pattern in at least two layers.

Gastrointestinal Procedures

Gastrointestinal surgery is indicated for removal of foreign bodies, resolution of intussusception, or investigation of abdominal distention.

Neoplasia

Snakes may develop various types of tumors in all the major body systems.[27] There is an increased incidence of neoplasia in immunologically compromised reptiles in general. Unfortunately, neoplastic conditions are usually diagnosed at necropsy. Surgical excision is the most common method to remove a neoplastic lesion.

Ophthalmologic Procedures

Dysecdysis usually causes retained spectacles, and when these are removed without care, the underlying cornea may be traumatized leading to keratitis and panophthalmitis. In such a case, the only useful technique is complete removal of the spectacle and underlying globe by enucleation.

Intestinal Prolapse

Eversion or prolapse of the terminal section of the intestinal tract through the cloaca may occur in snakes, usually caused by bacteria, protozoa, or infestation with helminths. The prolapsed tissue becomes desiccated and loses vascular supply, resulting in edema and necrosis. Minor protusions can often be reduced after the exposed tissues have been cleansed and lubricated. After simple replacement, the anal vent should be partially closed with a purse-string suture. A more extensive prolapse requires more aggressive surgical therapy. The cause of the original prolapse should be determined and remedied.

Repair of Severe Rat Bites

The bites of rats and mice often produce deep injuries in snakes. Some of these lesions must be surgically repaired, primarily when viscera has prolapsed through the intercostal musculature. Live rodents should never be left unattended in a snake's cage.

DIAGNOSTIC PROCEDURES

Physical Examination

Physical examination includes examination of the eyes, nostrils, mucous membranes, and oral cavity. The unrestrained snake should first be observed to evaluate its locomotor ability. To perform a physical examination, the snake should be restrained in the manner explained earlier.

The oral cavity may be opened for examination with a variety of rubber-coated spatulas. The next step is to make an overall assessment of the animal's health, palpating the entire snake for foreign bodies, abscesses, abnormal swellings, or trauma. The vent must be inspected for prolapse and diarrhea and the skin for ectoparasites and trauma. The heart is an important landmark in locating other coelomic structures. The lungs are located cranial to the heart in most viperids and elapids and caudal to the heart in boids and colubrids.

Abnormal body movements may indicate neurological disorders. A healthy snake should have good muscle tone and strength, appear alert and active, according to normal behavior patterns for the taxon, and actively flick its tongue. Snakes that gasp for breath, wheeze, bubble when exhaling, or have a nasal discharge may have a respiratory infection. The stethoscope is not as valuable a tool in snakes as it is in mammals because there are fewer narrow spaces in the respiratory tree to produce sounds as air rushes in and out. The best way to evaluate pulmonary and cardiac function is by a Doppler flow detector.

Laboratory Investigations

Sometimes, evaluation of a snake's health requires the collection of appropriate biological samples for laboratory investigations. The most commonly collected samples are blood for hematological, biochemical, and serological evaluation; biopsies for histopathology; scrapings, washings, and exudates for microbiological and cytologic evaluations; and fecal samples for parasitologic investigations.

- **Blood collection—**Clinical hematology and physiological chemistry are useful as adjuncts to a thorough physical examination. Unfortunately, few studies have been made to determine normal blood values of South American native snakes. Blood may be collected from snakes by venipuncture of the ventral tail vein or the palatine vein, or by cardiocentesis. The venipuncture site should be prepared by thorough cleansing and application of appropriate surgical antiseptics. Cardiocentesis should be limited to snakes weighing more than 300 g. The desirability of employing anticoagulants is determined by the circumstances and by the laboratory tests to be conducted.
- **Biopsies—**Biopsies are often necessary to diagnose disease in reptiles. Multiple samples should be obtained for histopathology, electron microscopy, cytology, and microbiology.
- **Microbiology—**Swabs, aspirates, and biopsy specimens may be collected and submitted for microbial isolation. It is important to collect samples of blood for cultures from reptiles suspected of being septic.
- **Fecal examinations and colonic washes—**Fecal samples or colonic washes may be evaluated by direct wet mount to ascertain the presence of protozoa and helminth ova; by sedimentation after centrifugation for the presence of protozoa and trematode ova; and by flotation, for identification of nematode ova. A few articles with photomicrographs of gastrointestinal parasites and helminth ova of reptiles have been published and should be used as guides for proper identification.[8]

Diagnostic Imaging

Unfortunately, diagnostic imaging is vastly underused in reptiles because of the lack of clear guidelines for its use in reptilian medicine and the diagnostic procedures of choice for each organ system.

- **Radiology—**The snake should be anesthetized and taped to a padded board. Radiographic examination is indicated when fractures or intestinal obstruction are suspected. Radiographic contrast procedures are often necessary to document intestinal obstructions, foreign bodies, respiratory diseases, or pregnancy.[4,20] X-rays may damage developing embryonic organs.
- **Ultrasonography—**The examination techniques are not complex but rely on the use of high-resolution equipment. For snakes, 7.5-MHz transducers with small footprints are recommended. Ultrasonography has been effective in documenting hepatic disease, reproductive status, coelomic fluid, pregnancy, and cardiac activity. The most basic requirement in interpreting ultrasound imaging is a knowledge of normal anatomy.

DISEASES AND THERAPEUTICS

Infectious Diseases

Infectious diseases are almost always secondary to immunosuppression in reptiles, and this is often associated with the stress of captive management and husbandry problems.

BACTERIAL INFECTIONS

- **Stomatitis or "mouth rot"**—Snakes with infectious stomatitis show swelling of the infected tissues, and the mucous membrane may exhibit areas of petechiation to ulcerative lesions with caseous material. Trauma and rostral abrasions may provide an opportunity for bacteria present in the normal microbial flora of snakes, such as *Pseudomonas* sp., *Klebsiella* sp., *Edwardsiella* sp., *Streptococcus* sp., *Escherichia coli,* or *Citrobacter diversus,* to enter the body. Not only must the infection be treated, but the problem that predisposed the snake to infection must be resolved. The oral lesions should be debrided and cleaned every 48 hours with 10% hydrogen peroxide or povidone-iodine solution. Severe cases must be treated with aminoglycoside antibiotics such as amikacin or gentamicin.
- **Pneumonia**—The commensal microflora may become agents of pneumonia when the animal has become susceptible as a result of chronic illness, poor nutrition, or adverse environmental factors (light, humidity, or temperature). The most common causes of bacterial pneumonia in snakes are aerobic gram-negative bacteria, such as *Aeromonas, Pseudomonas, Salmonella, Arizona, Klebsiella, and Proteus* spp. The signs are dyspnea, open-mouth breathing, and head and neck extension. Transtracheal washes are indicated for diagnosis and selection of the appropriate antibiotic.
- **Abscesses**—Reptilian abscesses are caseated and hard and are usually caused by gram-negative bacteria. Abscesses are best treated surgically, and the entire lesion with its capsule should be removed. Subspectacular abscesses occur as an ascending infection from the oral cavity or from septicemia. Treatment involves removal of a wedge of ventral spectacle, flushing of the subspectacular space with a balanced saline solution, and application of ophthalmic ointment.

VIRAL INFECTIONS Minimal information is available concerning viral diseases in reptiles. This lack of knowledge is because only a few researchers and practitioners have had an interest in investigating viral diseases. The two most important viral diseases are paramyxoviral infection and inclusion body disease.

- **Paramyxoviral infection**—This disease is most common in viperid snakes and may be transmitted by contaminated secretions from the respiratory tract.[12] The clinical signs are nasal discharge, open-mouth breathing, accumulation of caseous debris in the oral cavity, and respiratory sounds. Occasionally a snake may exhibit signs of central nervous system disease. There is no specific treatment except administration of antibiotics for secondary bacterial infections.
- **Inclusion body disease of boid snakes**—See the discussion later in this chapter.

Metabolic and Nutritional Diseases

Nutritional diseases rarely occur in snakes because they feed on whole vertebrate prey, which provides them with all essential nutrients. Well-nourished vertebrate prey may be considered to be a "complete and balanced" diet.

- **Hypothiaminosis**—Water snakes, such as *Liophys* sp. and *Hydrodinastis* sp., that feed on fish may develop thiamin deficiency if fed thawed frozen fish. Thiamin is depleted through the action of the enzyme thiaminase.[7] Clinical signs are generally nonspecific. Affected snakes are unable to accurately strike prey. Frozen fish should be supplemented with thiamin. Avoid offering fatty fish.
- **Overfeeding**—Captive snakes may become obese if fed too often, because they do not expend energy in obtaining food. Obesity predisposes animals to diseases such as steatitis and may cause infertility. Accumulation of enormous volumes of adipose tissue in celomic, subcutaneous, and intramuscular sites, and/or parenchymatous organs may lead to malfunctioning. Snakes of the Boidae and Viperidae families should be fed once every 4 weeks, whereas water snakes should be fed every week.
- **Gout**—Snakes excrete ammonium acid urate, sodium urate, or uric acid. When the renal threshold is exceeded, urates are deposited in other parts of the body. Dehydration is probably the primary factor in predisposing snakes to gout. Chronic diseases such as renal disease, hypertension, and starvation may affect uric acid excretion. Misuse of nephrotoxic antibiotics, such as the aminoglycosides, may cause tubular nephrosis and predispose reptiles to hyperuricemia. Treatment of gout in reptiles is uncertain.

Parasitic Diseases

ECTOPARASITES

- **Ticks**—Ticks of the genus *Ixodes* have been reported in snakes and may damage the host's skin and transmit blood parasites and viruses.
- **Mites**—The most important mite of snakes is *Ophyonissus natricis,* which is frequently seen in the area of the eyes, between scales, and around the cloaca. Mite infestation can cause debilitation through blood loss and predispose the animal to stomatitis, pneumonia, and septicemia resulting from the transmission of the gram-negative bacteria *Aeromonas*

hydrophila that also causes hemorrhagic septicemia in snakes. Spraying the animal with Fipronil (Frontline Spray; Rhodia Mérieux) is a good treatment against mites.

ENDOPARASITES

Protozoa

- **Amoebiasis—***Entamoeba invadens* causes high morbidity and mortality in snakes. It is contracted by ingestion of infected cysts that are passed in the stools of other reptiles (direct life cycle). Clinical signs are anorexia, dehydration, wasting, diarrhea with mucus and blood in malodorous feces. A direct smear of fresh feces will reveal the cysts. Lesions seen at necropsy include gastritis, enteritis, colitis, ulceration, and necrosis of the gastrointestinal mucosa. Two amoebicides that are reported to be safe and effective are metronidazole and dimetridazole.
- **Coccidiosis—**Coccidiosis affects the epithelial surfaces of the intestinal and biliary systems. Organisms most frequently reported are *Eimeria, Isospora,* and *Caryospora* spp. *Sarcocystis* spp. are not particularly pathogenic. The life cycle of coccidia is direct. Cellular destruction of the epithelial cells of the intestines, biliary system, or kidneys may cause fibrosis, ulcers, and septicemia. Clinical signs are anorexia and diarrhea. Diagnosis of coccidiosis can be made from direct fecal smears or with flotation techniques. Control and prevention of coccidiosis in a captive colony of reptiles requires good hygiene and isolation procedures. Sulfonamide antibiotics should be effective.
- **Cryptosporidiosis—**The source of the infection is unknown, but may be sporulated oocysts shed by other reptiles or from small mammals used in the diet. The parasite causes postprandial regurgitation and chronic hypertrophic gastritis in snakes. The clinical course may continue for an extended period with persistent regurgitation, weight loss, and midbody swelling. Diagnosis of cryptosporidiosis is established by finding oocysts in stained fecal smears. There is no satisfactory treatment for cryptosporidiosis, but supportive care has had good results.

Helminths

- **Trematodes—**Digenetic trematodes are the most important group in snakes. The majority of these trematodes, although commonly seen in snakes, cause little pathology and are likely to be pathogenic only in aberrant sites or when they occur in large numbers. Examination of the oral cavity will generally aid in making a diagnosis, together with routine fecal examinations.
- **Cestodes—**All reptilian tapeworms require an intermediate host. *Ophiotaenia* sp is the most common tapeworm. Diagnosis is based on adult segments in the feces or, more commonly, the demonstration of eggs in fecal smears or flotations. Usually tapeworms are only mildly pathogenic.
- **Nematodes—**The clinical signs of nematode infection include anorexia, anemia, regurgitation, signs of obstruction, and wasting disease. Diagnosis is accomplished by fecal flotation.
- **Ascarids—**The majority of these nematodes occur in the cranial end in the gastric mucosa, causing granulomatous gastritis. It is believed that they feed on the ingesta rather than on the host's tissue. The adult parasite occurs in the esophagus, stomach, or small intestine. Heavy adult ascarid infestation can cause gastrointestinal perforation or obstruction in snakes. Regurgitation of half-digested food may be an indication of ascarid infection in snakes. The best known species of ascarids are *Ophidascaris* and *Polydelphis* spp.
- **Lungworms—***Rhabdias* spp. are parasites of the lungs, but may also be found in the coelom and the pericardial sac. Many snakes harboring lungworms show no clinical signs and have a minimal inflammatory response; however, the parasites have been described as causing severe pneumonia.
- **Strongyloid nematodes—**Strongyloids may be found anywhere in the alimentary tract. They feed mainly on the blood of their host. Infestation has been associated with hemorrhagic ulceration, caseous enteritis, and gastrointestinal obstruction. The life cycle is direct. Heavy parasite loads may cause anorexia, debility, and death. The most important genera is *Kalicephalus*.
- **Pentastomids—**These parasites live exclusively as internal parasites. The adult worms occur in the lungs of snakes. The most common genera are *Porocephalus* sp. and *Armillifer* sp. Pentastomid infections cause little inflammatory response, but in some instances, there may be tissue destruction of the host. Most infections with pentastomids are asymptomatic.

QUARANTINE

All new arrivals to collection of snakes should be physically examined, dewormed, and bathed with an ectoparasiticidic solution. They should be housed in a separate room, inspected daily, and routine fecal examinations should be performed. A quarantine period of 1–2 months is recommended. To avoid stress during quarantine, the animal should be maintained at its preferred optimum temperature and humidity, which may be difficult if other snakes with different temperature/humidity requirements are undergoing quarantine at the same time.

Snakes that are ill should also be isolated while being treated. Necropsies should be performed on all snakes that die.

Cages should be checked daily for feces, and the entire enclosure should be cleaned and disinfected between patients. The best disinfectants are 4% sodium hypochlorite and quaternary ammonia compounds.

THERAPEUTICS

Reptilian formularies are available in editions dealing with reptiles or exotic animals.[8,11,15,21]

REFERENCES

1. Almandarz, E. 1978. Physical restraint of reptiles. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 130–132.
2. Bennett, R.A. 1991. A review of anesthesia and chemical restraint in reptiles. Journal of Zoo Wildlife Medicine 22(3):282–303.
3. Boyer, T.H. 1992. Clinical anesthesia of reptiles. Bulletin of the Association of Reptile and Amphibian Veterinarians 2(2):10.
4. Cooper, J.E. 1981. Diseases of the Reptilia, Vol. 2. London, Academic Press.
5. Fowler, M.E. 1978. Restraint and Handling of Wild and Domestic Animals. Ames, Iowa, Iowa State University Press.
6. Freed, P.S.; and Freed, M.G. 1983. An additional restraint technique for venomous snakes. Herpetology Review 14(4):114.
7. Frye, F.L. 1986. Feeding and nutritional diseases. In M.E Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 139–151.
8. Frye, F.L. 1991. Biomedical and Surgical Aspects of Captive Reptile Husbandry. Malabar, Florida, Krieger Publishing.
9. Frye, F.L. 1991. Viral diseases. In F.L Frye, ed., Biomedical and Surgical Aspects of Captive Reptile Husbandry, 2nd Ed. Malabar, Florida, Krieger Company, pp. 137–138.
10. Hill, M.; Wagner, R.A.; and Yu, V.L. 1990. A prospective study of upper airway flora in healthy boid snakes and snakes with pneumonia. Journal of Zoo and Wildlife Medicine 21(3):318–325.
11. Jacobson, E.R. 1987. Reptiles. Exotic pet medicine. Veterinary Clinics North America Small Animal Practice 17(5):1203.
12. Jacobson, E.R. 1993. Viral diseases of reptiles. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 153–159.
13. Jakob, W.; and Wesemeier, H.H. 1995. Intestinal inflammation associated with flagellates in snakes. Journal of Comparative Pathology 112:417–421.
14. Jenkins, J.R. 1996. Diagnostic and clinical techniques. In D.R. Mader, ed., Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 266–271.
15. Johnson, E.R. 1987. Reptiles. Exotic pet medicine. Veterinary Clinics North America Small Animal Practice 17(5):1203.
16. Johnson, J.H. 1991. Anesthesia, analgesia and euthanasia of reptiles and amphibians. In Proceedings of the American Association of Zoo Veterinarians, Calgary, Canada, pp. 132–138.
17. Jorge, M.T.; Mendonça, J.S.; and Ribeiro, L.A. 1990. Flora bacteriana da cavidade oral, presas e veneno de Bothrops: Possível fonte de infecção no local da picada. Revista do Instituto de Medicina Tropical, São Paulo 32:610.
18. King, M.B. 1984. Noose tube: A lightweight, sturdy restraining apparatus for field and laboratory use. Herpetology Review 15(4):109
19. Mader, D.R. 1996. Parasitology. In D.R. Mader, ed., Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 185–203.
20. Mader, D.R. Ed. 1996. Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, p. 148.
21. Mader, D.R. 1991. Antibiotic therapy. In F.L. Frye, ed., Biomedical and Surgical Aspects of Captive Reptile Husbandry, 2nd Ed. Malabar, Florida, Krieger Publishing, pp. 661–634.
22. Millichamp, N.J. 1988. Surgical techniques in reptiles. In E.R. Jacobson and G.V. Kollias, eds., Exotic Animals: Contemporary Issues in Small Animal Practice. New York, Churchill Livingstone, pp. 49–69.
23. Murphy, J.B. 1971. A method for immobilizing snakes at Dallas Zoo. International Zoo Yearbook 11:233.
24. Murray, M.J. 1996. Pneumonia and normal respiratory function. In D.R. Mader, ed., Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 396–405.
25. Paulino, R.C. 1988. Investigação visando aspectos da biologia de espécie de *Poracephalus* Humboldt, 1811 (Pentastomida, Paracephalidae) que ocorre na área geográfica brasileira em *Bothrops jararaca* e sobre sua situação taxonômica. Instituto de Ciências Biomédicas da Universidade de São Paulo, São Paulo.
26. Quinn, H.; and Jones, J.P. 1974. Squeeze box technique for measuring snakes. Herpetology Review 5(2):35.
27. Ramsay, E.C.; Munsun, L.; Lowenstein, L.; and Fowler, M.E. 1996. A retrospective study of neoplasia in a collection of captive snakes. Journal of Zoo Wildlife Medicine 27(1):28–34.
28. Wang, T.; Fernandes, W.; and Abe, A.S. 1993. Blood pH and O_2 homeostasis upon CO_2 anesthesia in the rattlesnake (*Crotalus durissus*). The Snake 25:21–26.

INCLUSION BODY DISEASE OF SNAKES

Margarita Mas

Inclusion body disease (IBD) is a disease described in the 1970s, first in pythons (*Python molurus bivittatus*) and later in boas (*Boa constrictor*). Worldwide, it is one of the most important health problems of snakes in captivity.

Currently, a retrovirus is thought to be the etiology. The transmission route appears to be through secretions from infected animals. Keepers may carry the virus from one animal to another during routine care or the cleaning of enclosures. Intrauterine transmission may occur in viviparus or oviparus species. It may also be transmitted during copulation. A further possibility is that the snake mite *Ophionyssus natricis* may be a vector.

One of the worst aspects of this disease is that the virus may remain latent for a long period of time, without producing signs. Probably under conditions of stress, it becomes active and overt disease appears.

Although the illness in both pythons and boas is usually terminal, each group has different signs. Boas have a nervous system dysfunction, characterized by postural abnormalities ("star gazing") and loss of strength for constriction. As the disease progresses, the signs that appear include weight loss, regurgitation, anorexia, dysecdysis, and a predisposition to bacterial infections (stomatitis, pneumonia). Pythons exhibit aberrant behavior, such as hyperreflexia, disorientation, and loss of motor coordination, but no regurgitation.

Because most animals die or are euthanized, the final diagnosis is made through histopathologic analysis. Using hematoxilin-eosin staining, intracytoplasmic inclusion bodies may be observed as circles in the epithelial cells of the pancreas, kidney, and liver, ranging in size up to 10 microns in diameter. However, the absence of these circles cannot exclude the disease. Early in the course of the disease, leucocytosis may be pronounced, possibly as an immune response to the virus. Inclusion bodies may also be observed in red blood cells in 95% of the confirmed cases of IBD. Histopathologic evaluation should be done by trained personnel, in order to avoid confusion with parasites or staining artifacts. A test that may be used in the live animal is tissue biopsy to look for inclusion bodies in the pancreas, liver, kidney, or esophagus.

It is important to realize that a suspect animal with negative results of blood tests or biopsies should not be considered free of the disease. The animal should be subject to a strict quarantine for months with repeated examinations.

There is no specific effective treatment for this disease. Symptomatic therapy and control of secondary infections have been suggested, but because the disease is highly contagious and fatal, euthanasia is usually recommended.

Prevention necessitates strict quarantine, for at least 3 months in pythons and 6 months in boas. Mites must be eliminated and strict cleaning and disinfection of cages and material used for ill animals should be required.

In 1996, four cases of IBD were confirmed at Buenos Aires Zoo. A *Boa constrictor occidentalis* that had belonged to an illegal trader had been confiscated and placed in the Buenos Aires Zoo collection. Three other boas contracted the disease. Symptoms observed included regurgitation, weight loss, and weakness in constriction. The snakes developed secondary infections (stomatitis, pneumonia, enteritis, and neurologic alterations; see Figures 5.1 and 5.2). Intensive treatment failed.

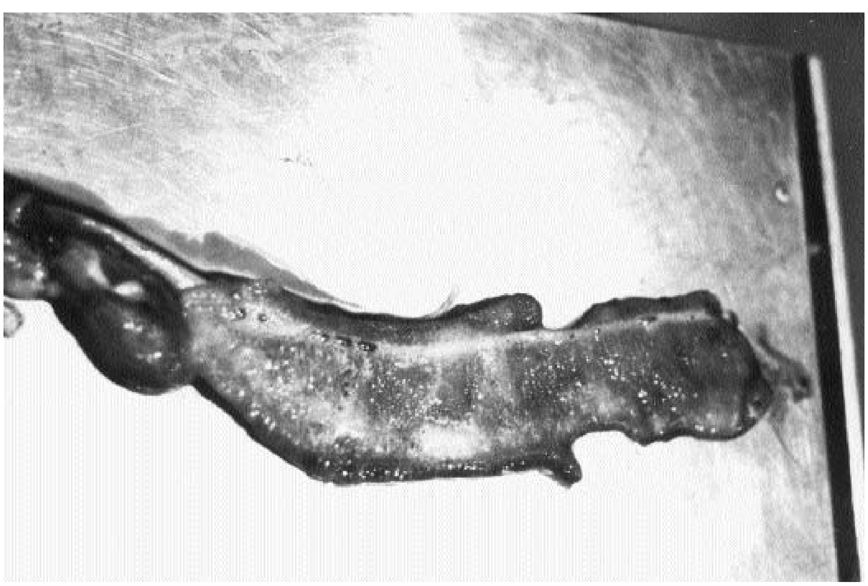


FIGURE 5.1. Inclusion body disease in *Boa constrictor occidentalis,* with secondary lung infection caused by *Proteus mirabilis.* (Courtesy Dr.Marcela Liliana Diaz, V.M.)

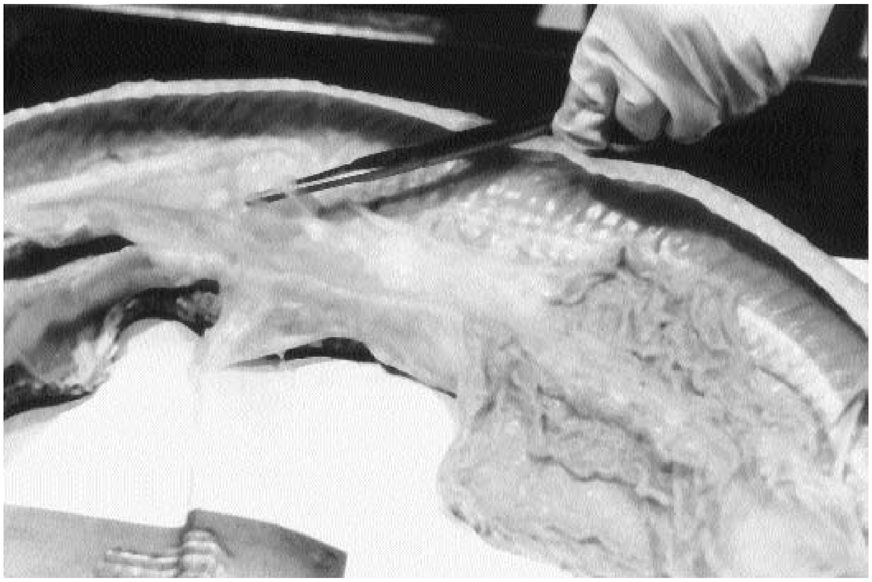
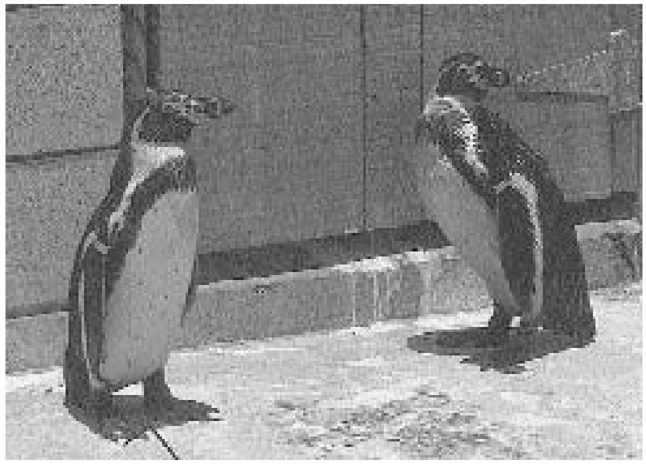


FIGURE 5.2. *Boa constrictor occidentalis* affected by inclusion body disease, with severe inflammation and production of mucus in stomach and small intestine. (Courtesy Dr.Marcela Liliana Diaz, V.M.)

REFERENCES

1. Frye, L.F. 1991. Biomedical and surgical aspects of captive reptile husbandry. Malabar, Florida, Krieger Publishing.
2. Mader, D.R. Ed. 1996. Reptile Medicine and Surgery. Philadelphia, W.B.Saunders, pp. 406–411.
3. Mader, D.R. 1996. Inclusion body disease virus. Reptiles Magazine 11:89.
4. Schumacher, J.; Jacobson, E.R.; Homer B.L.; and Gaskin, J.M. 1994. Inclusion body disease in boid snakes. Journalof Zoo and Wildlife Medicine 25(4):511–524.
5. Vosjoli, P. 1998. The Boa Constrictor Manual. California, AVS, pp. 64–70.

Birds: Class Aves



6 Order Sphenisciformes (Penguins)

Gene S. Fowler
Murray E. Fowler

BIOLOGY, CAPTIVE MANAGEMENT, AND MEDICINE

INTRODUCTION

Penguins are the most marine of all the seabirds, possessing a suite of adaptations that allows them to spend much of their lives living *in* (as opposed to *on*) the ocean. All are highly mobile swimmers, having traded flight in air for flight underwater, and many are pelagic predators, spending months at a time at sea without returning to land. However, like all birds, they must return to land to lay and incubate eggs, which they do typically in large colonies. This combination of pelagic predation and colonial nesting, sometimes far from feeding grounds, has led to some of the more interesting adaptations of penguins. They are among the fastest of vertebrate swimmers, and the order contains the deepest divers of all birds, those that can voluntarily fast for the longest period, and the only avian species that incubate their eggs on the tops of their feet (one of the latter is the only animal species that breeds in Antarctica during winter). Penguins are also extraordinarily attractive to humans, both in the wild and in zoos and aquaria. Worldwide, between 500,000 and 1,000,000 visitors per year travel to penguin colonies in the wild, and countless others observe them in zoos and aquariums on every continent, where they are among the most popular of visitor attractions.

BIOLOGY[1,8]

Taxonomy

Penguins belong to the order Sphenisciformes, which contains a single family, the Spheniscidae. The family contains 17 species, in six genera.[4] Ten of these species, in four genera, occur in South America or its offshore islands (Table 6.1), though only seven of these actually breed in the same area. Penguins are most closely related to the tubenoses (order Procellariiformes) and loons (order Gaviiformes), but fossil evidence indicates that they diverged more than 65 million years ago.

Distribution

Penguins are found only in the Southern Hemisphere, and only two species breed north of 30°S latitude. The family is generally thought by most people to be associated with the Antarctic continent, but this is a misperception; the

TABLE 6.1. Penguins inhabiting South America, including the Falkland Islands and offshore islands

Scientific Name	Common Name (English)	Common Name (Español)	Common Name (Portuguese)	Distribution
Spheniscus humboldti	Humboldt's penguin or Peruvian penguin	Pinguino de Humboldt	Pingüim	On the Pacific Coast of South America from 10^{o} to 45^{o}S.
Spheniscus magellanicus	Magellan's penguin	Pingüino Patagónico	Pingüim, Naufragado	Both coasts, 30^{o}S on Pacific and 40^{o}S on Atlantic, to Tierra del Fuego, Falkland Islands
Spheniscus mendiculus	Galapagos penguin	Pingüino de Galapagos	Pingüim	Only in the Galapagos archipelago
Aptenodytes forsteri	Emperor penguin	Pingüino emperador	Pingüim	Rare visitor to Tierra del Fuego and the continent, breed only in Antarctic
Aptenodytes patagonica	King penguin	Pingüino rey	Pingüim	Tierra del Fuego, Falkland Islands, and sub-Antarctic Islands
Pygoscelis antarctica	Chinstrap penguin	Pingüino de barbijo	Pingüim	Occasional visitor to Tierra del Fuego and the continent
Pygoscelis adeliae	Adelie penguin	Pingüino de adelia	Pingüim	Occasional visitor to Tierra del Fuego and the continent
Pygoscelis papua	Gentoo penguin	Pingüino pico rojo	Pingüim	Tierra del Fuego, Falkland Islands, and sub-Antarctic Islands
Eudyptes chrysolophus	Macaroni penguin	Pingüino frente dorado	Pingüim	Tierra del Fuego, Falkland Islands, and sub-Antarctic Islands
Eudyptes chrsocome	Rockhopper penguin	Pingüino penacho amarillo	Pingüim	Tierra del Fuego, Falkland Islands, and sub-Antarctic Islands

majority of species do not occur in Antarctica. The geographic origin of the family is uncertain, but modern species diversity is centered around New Zealand, which also contains the two most primitive genera (and the only two not found in South America). South America possesses by far the widest latitudinal range of species distributions, with penguins being found from Tierra del Fuego and sub-Antarctic islands (approximately 60°S), to the Galapagos Islands, on the equator. Of the four South American genera, only one (*Spheniscus*) breeds on the continent proper; all others breed only on offshore islands.

Habitat

All penguins live in two very different kinds of habitat, the feeding habitat, which is the ocean, and the breeding habitat, which is on land. Conditions in these two habitats are markedly different, and adaptations that benefit the animal in one habitat may pose limitations in the other. For example, the insulation required for survival in cold water predisposes desert-breeding species to hyperthermia when active on land.

Water temperature is a critical parameter for foraging habitat, and penguins live and forage in waters that range from –2°C to 23°C, though each species is restricted to only a portion of this temperature range.[9] All penguin species are dependent on cool to cold waters for their prey and even small amounts of variation in water temperature can have profound effects on populations. The warmer waters brought about by El Niño events in South America are closely associated with lack of forage species, reproductive failure (often by starvation of chicks), and sometimes starvation of adults.

Breeding habitats in South America are quite variable, ranging from rocky caves on barren desert shoreline, to tussock grass on rain-swept offshore islands in the far south. All, however, are very close to the shorelines; it is rare for South American penguins to travel more than 1 km inland. For the three *Spheniscus* species, soil type and/or guano depth are important habitat parameters, especially in the northern portion of their range.[9] These species nest in burrows that they dig where they find appropriate soils. Thermal cover is important at the desert colonies found in the northern portion of these species' ranges, but where soils are not suitable for burrows, nests are often placed under bushes or rock piles, or in caves. In a few cases, pairs will nest in the open, but such nests are not often successful. In the southern portion of the continent, nests may also be placed in burrows or under bushes, but nesting in the open becomes much more common. Species that nest in the more southerly offshore islands commonly nest in the open, either in tussock grass habitats or on rocky outcrops. The Magellanic penguin, the only *Spheniscus* species found in the far south, still preferentially digs burrows on the offshore islands, where soils are suitable.

Anatomy

Penguins are highly specialized and perhaps the most morphologically unique of all avian species, and most of their uniqueness is related to their life in the sea. Though descended from flying species, the high density of seawater has relieved them of the evolutionary pressure for weight-saving adaptations, at the same time causing selection for swimming adaptations. The 17 penguin species are strikingly uniform in overall morphology and coloration; body size is the most variable characteristic among species.[4]

All penguins have streamlined, fusiform bodies, short necks and tails, and short legs set far back on the body so that they stand upright on land. Wings have been modified into flat, bony flippers in which mobility is limited in all joints but the shoulder. The feet are three-toed and webbed, with an elevated, largely vestigial hallux. Bills are covered with horny plates, and vary considerably among species in shape. Fish-eating species tend to have longer and thinner bills, whereas krill-eating species have shorter and stouter bills. Species with less specialized food habits have intermediate bill form. All species have a white breast and belly, and a blue, dark gray, or (most commonly) black back. All other colors are restricted to the head except for the colored throat feathers of *Aptenodytes*. The other feather colors are restricted to yellow and orange, but there may be pink areas of bare skin on the face and reddish bills in different species.

The feathers are uniform in shape all over the body (although feathers on the flippers are shorter than body contour feathers), and apteria are lacking except during incubation, when a bare brood patch forms on the abdomen. Feathers are short and stiff, lanceolate, and slightly curved in shape, with a flattened shaft and downy aftershafts that form a dense down layer. They overlap extensively and form a waterproof covering, trapping a layer of insulating air below. Thermal insulation is also increased by the presence of a subcutaneous fat layer, with fat layers being thicker in the more southerly species.

There are also circulatory specializations that aid in thermoregulation. Blood flow to and from extremities is countercurrent, and *retia mirabilia* are also present to increase heat exchange. Blood flow to the extremities can be decreased or increased by vasoconstriction and anastomoses that shunt arterial blood back to veins. These adaptations help penguins to both conserve heat (in water and during winter on land) and to shed heat (on land in warm environments). Unlike flying birds, both jugular veins are present.

Internal anatomy is not strikingly different from other predatory birds, except that air sacs are lacking. There is no crop or proventriculus, and ingested prey moves directly to the glandular stomach. The gizzard is weakly muscular; stones have sometimes been found in the gizzard, but their function is unclear. As in flying birds, females normally possess only a single active (left) ovary, but testes are paired. There is no intromittent organ, and copulation occurs by cloacal contact. The kidneys excrete uric acid, and supraorbital glands are present and highly developed for salt excretion.

The overall musculoskeletal anatomy is similar to that of flying birds, with a highly developed carina of the sternum and strong breast muscles that are rich in red muscle fibers. Leg muscles are also well developed for upright walking on land. A major difference from flying birds is that bones are not pneumatic, but are solid and dense. Wing bones are flattened and the alula is lacking, but otherwise resemble those of flying birds. The lower leg bones differ substantially from those of flying birds, with a short, broad, tripartite tibiotarsus that is a diagnostic character of the family.

Molt

All penguins have a single annual molt, usually after breeding (Galapagos and king penguins typically molt before breeding).[9] Unlike most birds, where feathers are dropped sequentially and new feathers then grow in, old feathers are pushed out by new-forming papillae, and all feathers are dropped simultaneously. During this period, birds lose their impermeability to water and remain on land for 2–4 weeks, fasting. The process is energy demanding, and most birds precede the molt with a foraging period at sea in which they increase body weight by 50–70%. This additional weight is in the form of fat stores, which are lost as the molt proceeds. Migratory species typically leave the colony for the winter as soon as the molt is completed.

Longevity

Penguins appear to be long-lived birds, with high annual survival rates once adulthood has been reached.[9] However, precise estimates of longevity require detailed long-term studies on marked individuals, and there are no published studies reporting longevity for any South American penguin; very few data are available for penguins in other areas. Available data report individuals surviving and breeding until approximately 20 years of age in the wild in three species. Shorter-term studies of annual survival rates suggest a mean life span of similar duration for several species, but also suggest that some individuals may live as long as 25–50 years, depending on the species. High adult survival is balanced by low survival of chicks and juveniles. Longevity appears to be somewhat higher in captivity, and there are records of animals captured as adults in the wild living as long as 35 years.

Sex Determination

Penguins are sexually monochromatic, and distinguishing the sexes is difficult.[9] However, in many species males are slightly larger than females, but there is overlap in size distributions, and overall size is not a reliable criterion for sexing individuals or members of pairs. Bill size is also commonly sexually dimorphic (especially in *Spheniscus*), with males having larger, stouter bills, and can be used to distinguish sexes, but only after bill measurements have been collected from a sample of animals of known sex. Females can be reliably recognized during egg laying by their large cloacal diameter, but this criterion is only useful for a short period during the year. The only universally reliable methods are laparoscopic surgery (difficult given the body size of most species), and genetic differentiation. The latter is the method currently used most commonly in captive populations.

Social Structure

All South American penguins are highly gregarious, social species, both on land and at sea.[4] Little is known about social structure at sea, except that animals travel and forage in groups. On land, penguins also aggregate into colonies for breeding purposes, and even when resting on beaches animals tend to join groups—single, isolated individuals are not commonly observed in the wild. Social groups are probably not related to family structure, as fledglings and juveniles are generally not tolerated in groups of adults. When chicks first fledge, leave their nest, and go to sea, they do so on their own, and are not assisted by parents. In fact, they are usually the recipients of active aggression from adults resting on beaches. However, in the few studies detailed enough to show recruitment patterns, there is a high degree of philopatry, and most animals return to breed in the same portion of the colony in which they were hatched. But, it is unlikely that they are assisted by or even recognized by their parents (most species do not return to breed until they are 4–6 years of age), so that, although animals nesting near each other may be genetically more closely related than more distant animals, there is no true familial structure to colonies. In those species studied in detail, breeding adults have a strong tendency to return to the same nest year after year, and so neighbor relationships are mostly stable.[9]

Diets and Feeding Behavior

Food habits vary considerably among genera and species, although all are active pelagic predators. The *Spheniscus* penguins feed primarily on pelagic schooling fish and squid, which they attack in groups.[10] The black and white facial pattern of this genus is thought to aid in group foraging by causing startle responses in prey fishes that make them more susceptible to capture by other penguins in the same group. The *Eudyptes* and *Pygoscelis* penguins feed on both crustaceans (primarily krill) and on fishes, with the former genus more specialized on krill; both genera feed occasionally on cephalopods.[9] However, given that all available data are from the sub-Antarctic and Antarctic parts of their ranges and that the proportion of fish in the diet is higher in the north, it may be that populations nesting in South America are more piscivorous than the available data would suggest. All species in the above three genera are relatively shallow divers and rarely forage below 50 m depth. The *Aptenodytes* penguins are largely piscivorous, but feed on bottom-dwelling myctophid fishes (rather than schooling pelagic fishes) and may be less social in foraging than the other species.[9] They are also much deeper divers, commonly exceeding 200 m; emperor penguins have been recorded at depths exceeding 450 m.

Predators

On land, humans have historically been by far the most important predators of adult penguins, which were heavily exploited for oil and protein during the previous two centuries. Although harvest levels have been greatly reduced during this century, both eggs and adults continue to be consumed in some parts of South America, especially on offshore islands (eggs) and within the range of the Humboldt penguin (adults). Most offshore colonies lack native terrestrial predators entirely, although introduced species such as rats (*Rattus norvegicus* and *R. rattus*) and cats (*Felis domesticus*) may pose problems for eggs and chicks (see Conservation below). On the continent, the fact that penguin colonies are only occupied during the breeding season means that potential predators of adults cannot maintain populations above levels that can be sustained on other prey species available during the nonbreeding season, when penguins are at sea. Terrestrial predators such as foxes (*Dusicyon* sp.) and felids (*Felis* sp.) and introduced species do take advantage of the availability of eggs and chicks, as do avian predators that breed in the same environments, especially gulls (*Larus* sp.) and skuas (*Stercorarius* sp.). However, predation on eggs and chicks does not appear to limit penguin populations.

Adult South American penguins do appear to face important predators at sea, although there are few published studies and evidence is mostly anecdotal. Sharks, sea lions (*Otaria* sp.), and orcas (*Orcinus orca*) all probably prey on adult birds throughout South America and the offshore islands. Leopard seals (*Hydrurga leptonyx*) are important predators of penguins in the far south, but are rarely found north of the sub-Antarctic islands. The high annual survival rates of adult penguins suggest that predators are not important limiting factors in penguin population dynamics in South America.

Reproduction in Wild

Courtship and Mating

In most cases, male penguins arrive at breeding colonies before females do and begin territorial and mate-attraction displays that tend to be similar among species. Fidelity to nest sites is high in all but *Aptenodytes* species (which do not build nests), ranging from 70–95%. Males engage in vocal and visual displays before and during female arrival. Vocal displays are raucous, noisy, and acoustically complex and are used in both aggressive and courtship contexts. The “ecstatic” display is a common male courtship display, in which they hold their bill vertically, extend their flippers, and call. Females are also vocal after their arrival (though less so than males), and begin to engage in “mutual” displays that are much like ecstatic displays, but in which both sexes participate. “Bowing” displays are also common between the sexes during courtship. Copulation is often preceded by mutual bowing displays, and by one or both sexes circling the nest. Males often vibrate their flippers against the flank of the female just before copulation. Copulation begins when the female assumes a prone position, the male stands on her back and moves posteriorly to bring cloacae into contact. Copulation is frequent in most species before egg laying, but is uncommon thereafter.

All penguin species appear to be socially monogamous during the breeding season, meaning that each individual has only a single mate, with which it typically shares parental duties.[9] Individuals may or may not keep the same mate between years; long-term pair bonds (“mating for life”) are quite variable among species. *Aptenodytes* penguins typically do not retain mates from previous years, but in all of the other South American penguins that have been studied, mate retention is the norm, with 50–90% of pairs reuniting each year. In many cases, failure to reunite with a previous mate is preceded by reproductive failure, and pairs that reunite often have higher reproductive success than newly formed pairs. There have been no published studies of extra-pair fertilization, but extra-pair copulations have been observed in the wild, and it is likely that the rate of genetic monogamy is less than 100%.

Nesting

Most penguin species do not construct elaborate nests.[4] Even in the burrowing *Spheniscus* species, the nest itself is of simple construction. In burrows, under bushes, and when the members of this genus nest in the open in shallow depressions, the nest is usually lined with only a few twigs and penguin feathers (the latter probably plucked from the breast of the incubating parents as the brood patch forms). The burrows themselves are typically simple straight tubes, usually 0.5–1.0 m in depth, though occasionally deeper. The *Pygoscelis* and *Eudyptes* penguins typically build nests that are piles of small stones, often lined with small amounts of grass and/or feathers. Suitable stones can be important resources that allow nests to be elevated above flooding under wet conditions in level colonies. *Aptenodytes* is unique among penguins in building no nests at all. In both king and emperor penguins, eggs are incubated on the tops of the feet.

Incubation

Clutch size in penguins is invariant; normal clutches consist of two eggs in all species except *Aptenodytes,* both of which lay only a single egg.[4] Young females of other species may occasionally lay only a single egg, but experienced adults lay two. There are reports of three-egg clutches, but it has not been conclusively established that such clutches have been laid by a single female; "egg adoption" has been observed, and females have been observed to be driven out of the nest by another female after laying an egg. Eggs within a clutch are typically similar in size, except in *Eudyptes,* where the first egg is substantially smaller than the second.[9] The laying interval between eggs is relatively long, ranging from 3–6 days, with 4 days being the most common interval. Penguin eggs are quite small relative to the size of the female, ranging from 2–5% of female body mass, but have a relatively high yolk content. Eggs are spheroid, ovoid, or pyriform, depending on species, but all are pale and have unspotted shells, ranging from white to greenish- or bluish-white.

In all species except emperor penguins, both sexes share in incubation.[9] In emperor penguins, only males incubate. The duration of incubation turns is highly variable among species and between populations of the same species. In some species (e.g., gentoo and some populations of Magellanic penguins), parents alternate incubation turns on a daily basis. In others (*Eudyptes* penguins and other populations of Magellanic penguins) incubation turns may last 1–2 weeks. Change of incubation turns is often accompanied by intense vocalizations and mutual displays. The duration of incubation is positively correlated with body size; the medium-sized penguins such as *Eudyptes, Pygoscelis,* and *Spheniscus* have incubation durations of 35–40 days, whereas in *Aptenodytes* incubation lasts 55–65 days.

Chick Rearing

Care of chicks is biparental in all species of penguin.[9] Chicks are hatched in a semialtricial state, with a complete downy plumage, but eyes are closed and mobility is limited. Newly hatched chicks are also unable to thermoregulate and must be brooded by one parent for several weeks, while the other parent forages. Feeding of chicks is by regurgitation of stomach contents. When chicks are able to thermoregulate, they are left alone at the nest site and both parents forage, returning only to deliver food. At this point, chicks may aggregate into groups called creches, although this is most common in cold climates and is less common in more northerly colonies. When chicks have reached adult size and acquired mature feathers, usually in a juvenile plumage that is distinct from that of adults, they are left alone at the nest site and make their way to the sea without any adult assistance. Postfledgling mortality appears to be high in most species, resulting in low recruitment rates into breeding populations.[9] At the end of their first year, juveniles molt out of their distinctive plumage and become difficult to distinguish from older birds. However, in all South American penguins, breeding does not commence in the second year and is further delayed for up to several years. Average age at first breeding is about 5 years for females and 6 years for males.

Conservation

Although they live and breed in remote and isolated areas, penguins have not been immune to human-caused conservation problems, and, indeed, only the southernmost insular and Antarctic populations are currently thought to be at low risk of extinction.[2] South American species of special concern include the Galapagos penguin, considered Endangered under both U.S. law and by the International Union for the Conservation of Nature (IUCN), and the Humboldt penguin, listed on Appendix I of the Convention for International Trade in Endangered Species (CITES) and considered Vulnerable by the IUCN. In addition, populations of penguins nesting on the Falklands/Malvinas Islands have undergone drastic decreases in recent years, for reasons that are not fully understood. Worldwide, 10 of 17 penguin species are considered to be Endangered or Vulnerable under IUCN criteria.

The amphibious life of penguins predisposes them to a number of conservation problems.[9] The great amount of time they spend on the sea surface exposes them to petroleum, plastics, and other pollutants at their highest marine concentrations. They spend the breeding season in nearshore waters that are most heavily impacted by human activities, and they depend for forage on species that are also exploited for human food. They have low reproductive rates and delayed maturity, so that potential population growth rates are low, and they lack the ability to recover rapidly from catastrophes such as oil spills. Their tendency to aggregate in large colonies and their apparent approachability make them attractive for tourism, but disturbance may interfere with breeding. And, the South American species may be especially susceptible to global climate change, because El Niño events have their greatest impact in the southeastern Pacific Ocean and are predicted to become more frequent.

The Galapagos penguin is considered to be the most endangered of all penguin species. Recent estimates suggest that there may be as few as 800–1500 individuals in the population. This northernmost of all penguins is especially susceptible to changes in water temperature, and El Niño events have resulted in substantial adult mortality and population declines. In addition, recent increases in fishing activity have resulted in increased mortality. Galapagos penguins are also at risk from introduced non-native species (especially rats and cats).

Humboldt penguins face a number of risk factors. Total population size is low (estimates suggest no more than 15,000–20,000 animals), and the animals are spread thinly along the Pacific coast. Adults are illegally exploited for food and for fish bait, and nesting habitat has been damaged by excessive guano harvest, leaving the birds no place to dig burrows. This species is among the most wary of penguins, and they respond in panic to the presence of humans, occasionally injuring and even killing themselves. Commercial fishing activities result in direct mortality in lines and nets, and reduced prey populations decrease the prey base for survival and breeding. Humboldt penguins are also susceptible to changes in nearshore currents during El Niño events, although adult mortality does not appear to be as high as for Galapagos penguins.

Recent estimates suggest that rockhopper penguin populations (and perhaps those of other species) nesting in the Falkland/Malvinas Islands have declined as much as 70–90% in the last decade, although these populations have not been studied in detail. This decline corresponds closely with increased commercial fishing in the South Atlantic, following the Falkland War. No direct linkage has been established, but it is likely that the decline is related to changes in prey density caused by fish harvesting. In addition, there are plans to develop oil fields on the adjacent continental shelf, leading to a potential increase in oil fouling, which can kill penguins directly and interfere with breeding even in lightly oiled animals.

Magellanic penguins are not currently considered to be at risk, but some populations are declining for reasons similar to those of the Falklands/Malvinas. In addition, as many as 40,000 penguins may be killed per year by oil fouling due to chronic spillage on the Atlantic coast. The remaining South American species nest farther south and are considered to be at lower risk.

MANAGEMENT IN CAPTIVITY

Housing

Penguins are highly gregarious and are colonial nesters. The facilities provided depend on whether or not breeding is anticipated and on the heat tolerance of the species to be exhibited. Facilities vary from small fenced-in enclosures containing a small pond for swimming to elaborate air-conditioned, fully enclosed buildings with water filtration and filtered air.

SPACE REQUIREMENTS The minimum amount of dry space required may be calculated on the basis of the formula: $SA = (1.5\ h)^2 \times N/2$, where SA = surface area (square meters), h = the average height (meters)of the species, and N = number of birds to be exhibited. The dry area must allow the birds to dry completely, including the feet, to avoid bumble foot.[7] The water area should be two to three times the land area, and the water should be at least 1 m deep.

Water Quality

Penguins may be maintained in freshwater, but sodium chloride (0.15 g/kg daily) should be added to the diet to prevent functional loss of the salt glands. Water quality (filtration and purification) for penguins should be the same as for exhibiting marine mammals or fish. Space does not allow a detailed discussion. Special provisions must be made to remove any fish oil left in the water from uneaten fish or penguin feces. Surface skimmers perform this task adequately. Oils disrupt feather structure, which in turn may affect buoyancy and thermoregulation. Oils also affect filtration systems that use sand or diatomaceous earth, necessitating a weekly change of water.

Environment

Tropical species (Galapagos penguin) must be protected from cold weather in temperate climates.[7] *Spheniscus* penguins must be protected from cold temperatures that freeze water surfaces and prolonged exposure to temperatures over 30°C (86°F), unless they have access to shade, aerial showers, and adequately cooled water for swimming.[7] Polar species are usually housed in refrigerated buildings where air temperatures do not exceed 9°C (48°F) for a prolonged period. Optimally, ice temperatures should be below freezing to prevent the birds having to stand in water. Penguins maintained in self-contained buildings require adequate filtered ventilation to remove odors and ammonia and to prevent the introduction of fungal and bacterial pathogens.

Feeding

Because penguins vary in the type of food consumed in the wild, it follows that the diet of different species should be tailored to the requirements of the species. Perhaps most important is that a variety of food items be included in the diet. The nutritional value of an individual species of fish or squid may vary with the method of processing, length of time in storage, and the basic nutrient composition of the species. Feeding a variety of fish or squid helps to balance any nutrient deficiency present in a single species and avoids habituation to a single food source should it become impossible to obtain the habituated food fish.

A number of species feed primarily on krill (*Euphausia* spp) in the wild, but in captivity this is not available; thus, small fish are offered, including mackerel (family Scrombidae), herring (*Clupea* spp), smelt (Osmeridae, genera *Osmerus* spp, *Mallotus* spp, *Hypomesus* spp, *Taleichthys* sp), and sprat (*Sprattus* spp). Birds should be individually fed to ensure adequate intake. The first fish offered should contain any supplement or medication to be administered.

Supplements usually include vitamin A, vitamin D, and B-complex (including 25 mg of thiamine to compensate for possible thiamine destruction by thiaminase activity in stored fish). Many zoos offer a buffered salt tablet, providing 0.45 g sodium chloride daily, to maintain a functional salt gland.[3]

Recent penguin nutritional studies have shown significant differences between nutrient levels of vitamin E, vitamin D, and vitamin A in comparison with terrestrial birds. Thyroid activity and corticosteroid levels are dramatically altered during the molting process, which may include a 40-day fasting period and a loss of over 50% of the body weight.[6] Obviously, adequate food must be provided before the molt to allow the bird to accumulate ample body condition.

Reproduction in Captivity

A number of species of penguins have been propagated in captivity. Appropriate environmental and social considerations for the species must be provided. Refer to the foregoing reproduction section. Selected reproductive data on South American penguins is included in Table 6.2. Breeding seasons are reversed in the Northern Hemisphere.

Artificial incubation and hand rearing is possible, but is not commonly practiced in zoos. Penguin chicks grow rapidly. Parents must be provided with adequate food or the chicks may become malnourished. Metabolic bone disease will be a problem only if immature fish are used to feed the parents. Sexual maturing is not reached for 2–4 years.

TABLE 6.2. Reproductive data of South American penguins

Scientific Name	Breeding Season	Breeding Location	Incubation Period
Spheniscus humboldti	Poorly known, Sept.–May? 2 distinct peaks	Pacific Coast, 5°–42°S	41 days
Spheniscus magellanicus	Sept.–Feb.	Atlantic Coast, 42°–55°S, Pacific Coast, 29°–55°S, Falkland Islands	40–45 days
Spheniscus mendiculus	Year-round	Galapagos Islands, 0°	35–40 days
Aptenodytes forsteri	March–Sept.	Antarctic fast ice	65 days
Aptenodytes patagonica	Extended, 14–16 months per clutch, begins Nov.	Tierra del Fuego, sub-Antarctic islands	52–55 days
Pygoscelis antarctica	Sept.–Feb.	Sub-Antarctic islands, Antarctic Peninsula	34–37 days
Pygoscelis adeliae	Sept.–Feb.	Sub-Antarctic islands, Antarctic coast	35–38 days
Pygoscelis papua	Sept.–Feb.	Falklands, sub-Antarctic islands, Antarctic Peninsula	35–38 days
Eudyptes chrysolophus	Oct.–Feb.	Tierra del Fuego, Falklands, sub-Antarctic islands	35–36 days
Eudyptes chrsocome	Oct.–Feb.	Tierra del Fuego, Falklands, sub-Antarctic islands	32–38 days

RESTRAINT, ANESTHESIA, SURGERY

Restraint and Handling

Penguins are strong for their size and have sharp beaks. Both the beak and the flippers are potent weapons and their immobilization is essential to successful handling. Small- to medium-sized penguins are easily restrained physically, but procedures must be carried out quickly to avoid overheating while struggling. Restrict the penguin to a dry area without access to a pool. Use an appropriately sized hoop net for the initial capture, or small species may be grasped with both hands from behind. Grasp the base of the head firmly with a gloveless hand so that tactile discrimination is maximal. The legs may then be grasped and a light stretch applied. If wing movement must be controlled, another person is required for the procedure.

Large penguins may be captured by placing an appropriately sized plastic garbage can, with the bottom cut out, over the top of the bird. The bird's movements are restricted without undue trauma, yet many procedures may be carried out. It may be desirable to hood the penguin to avoid being pecked. Chemical restraint is rarely employed in the handling of penguins.

Holding a penguin for blood collection is accomplished as follows. The holder handles the bird by grasping the back of the neck at the base of the skull, and while kneeling, places the bird between his/her knees so that the shoulders and flippers are immobilized. It is useful to cover the birds' head with a hood of soft, heavy, dark cloth. The penguin, at this point, is in a semiprone position and is resting most of its weight on its own feet and belly. Holding the bird with its feet off the ground, or in a standing position without adequate immobilization of the shoulders and flippers, results in increased struggling. When the bird is adequately immobilized, its neck is then stretched forward and rotated slightly along the thigh of the holder to expose the right side of the neck, so that the jugular may be palpated. The intent is to get the neck as straight as possible. If the neck is contorted, or if the animal is struggling, the position of the jugular vein may change relative to the neck muscles, and it may be quite hard to find.

For sampling sites in the extremities, physical restraint is similar, except that body of the animal is held in a more upright position, with the animal resting its weight mostly on its feet. A foot or a flipper may then be grasped by the sampler and stretched out to find the relevant vein. Attempting to collect a blood sample without adequate physical restraint is hazardous to both the handler and the animal.

Anesthesia

Inhalation anesthesia using isoflurane is the safest and most effective method of anesthesia. Induction and recovery are rapid. Halothane has also been employed but is less desirable. Diving birds, such as the penguins, are able to hold their breath for a considerable time, thus mask induction may be prolonged. It is better to quickly open the mouth, insert an endotracheal tube, and compress the rebreathing bag to force inhalation.

Surgery

Surgical procedures are carried out as in other avian species. Penguins do not have apterylae so it is necessary to pluck feathers to gain access to the skin for the initial incision. Pluck only those feathers directly on the incision line. Use sterile aluminum foil to cover the compressed feathers adjacent to the incision line, and then use masking tape or duct tape to adhere the tape to the skin. Avoid placement of duct tape directly on the feathers, as the adhesive will damage the feather structure when the tape is pulled off. Once the incision site is adequately draped with aluminum foil, the skin may be cleansed and disinfected. Then sterile cloth drapes may be used to provide a sterile surgical field. Subcuticular sutures should be used to close the skin to avoid the bird's ability to grasp a suture and remove it.

DISEASES

Infectious Diseases

FUNGAL DISEASES The most important fungal disease is aspergillosis (see Table 6.3). However, other fungal genera may be isolated from infections. The stress associated with capture and subsequent captivity may render penguins susceptible to *Aspergillus fumigatus* infection. The organism is found wherever organic material is found. The key to management of aspergillosis is prevention. Success in maintaining polar species may require that the birds be kept in a filtered air environment. Aspergillus vaccines and other therapies have not been found to be effective in dealing with aspergillosis.

BACTERIAL DISEASES Penguins are susceptible to many of the opportunistic pathogens common to all species of birds (see Table 6.3). Penguins have no unique bacterial diseases.

Infectious pododermatitis (bumblefoot) is ultimately a bacterial infection, but predisposing factors include an inappropriate surface in the enclosure, and an abnormally sedentary lifestyle with the bird standing for long

TABLE 6.3. Infectious and parasitic diseases—Sphenisciformes

Disease (English)	Disease (Español)	Disease (Portuguese)	Etiology	Signs	Diagnosis	Management
Tuberculosis	Tuberculosis	Tuberculose	*Mycobacterium avium/ M. intracellulare*	Emaciation, diarrhea	Culture, histopathology	Difficult, sanitation, quarantine
Salmonellosis	Salmonelosis	Salmonelose	*Salmonella* spp.	Diarrhea, septicemia	Culture	Sanitation, antibiotics
Colibacillosis	Colibacilosis	Colibacilose	*Escherichia coli*	Septicemia	Culture	Sanitation, antibiotics
Infectious pododermatitis (Bumblefoot)			*Staphylococcus aureus, Pseudomonas* spp., *Klebsiella* spp., *E. coli*	Foot pad calluses and ulcerations, swollen digits and pads, lameness	Clinical signs, radiography	Provide suitable substrate, provide dry surfaces so the feet can dry completely
Velogenic viscerotropic Newcastle disease	Enfermedad de Newcastle	Doença da Newcastle	Paramyxovirus	Central nervous system effects, tremors, paresis, paralysis	History, signs, viral culture, serology	Quarantine, sanitation
Aspergillosis	Aspergilosis	Aspergilose	*Aspergillus fumigatus*	Dyspnea, exercise intolerance	Culture, direct smear	Minimize stress
Malaria			*Plasmodium* spp.	Weakness, anemia	Direct smear	Mosquito control

periods with the weight focused on one portion of the foot. This compromises dermal perfusion and ultimately results in an avascular necrosis and ulceration of the dermis, allowing invasion of the subdermal tissue with epithelial microorganisms. Contributing factors include local trauma, poor sanitation, and nutritional deficiency (hypovitaminosis A and E).[5]

There are three to five types of the disease. Using one raptor classification, type 1 is a callus formation (epithelial hypertrophy), with no pain, heat, or swelling present. Type 2 is characterized by subacute to chronic infection of the dermis and subdermis of the pad(s), which is warm and painful to the touch. The penguin may be reluctant to place weight on the affected limb. In type 3, the chronic, purulent infection spreads to produce cellulitis, tendosynovitis, osteomyelitis, suppurative arthritis, and deep-seated abscesses. Avian exudate is caseated because of lack of lysozymes so exudate cannot be withdrawn through a needle. In one well-managed institution, 64% of the Adelie penguins had one or more types of bumblefoot.[5]

Prevention is the key to management of bumblefoot. More research is necessary to establish the most suitable enclosure surface for penguins. Once the tissue has become devitalized and infected, heroic therapy may or may not be successful. The more advanced type 2 infections may require surgical debridement plus administration of local and systemic broad spectrum antibiotics. Long-term protection of the foot is necessary in most cases. This is difficult to provide in a penguin, but one institution has fashioned a penguin bootie from a diver's neoprene wetsuit bootie. The bootie is held in place on the foot by means of two Velcro strips, one over the toes and the other fixing the metatarsus to the heel. The wetsuit bootie is cut off to allow the toenails to extend beyond the bootie.[5] The bootie allows the bird to swim and may be employed for 2–3 months, but the foot should be evaluated and the bootie changed and cleansed every 3–7 days. A more detailed discussion of this disorder is found in the chapter dealing with raptors.

VIRAL DISEASES Only recently have viral organisms been isolated from free-ranging penguins. The importance of these viruses in producing disease is unknown.[6] Viral diseases are not common in captive collections, but Newcastle disease has been reported. Cutaneous pox lesions of the eyelids have been observed in black-footed penguins.[7]

PARASITIC DISEASES Penguins may harbor biting lice *Austrogoniodes watersoni,* fleas *Parapsyllus australiscus, Theromyozon rude,* and leeches *Placobdella ornata.* Various trematodes, cestodes, and nematodes infest penguins, but clinical parasitism is not a major problem. Avian anthelmintics are appropriate for penguins.

The most important parasitic disease of captive penguins in the Northern Hemisphere is avian malaria. *Plasmodium relictum, P. elongatum,* and other *Plasmodium* spp are relatively nonpathogenic parasites of North American birds. Penguins have little or no innate resistance and may succumb to infection with these parasites when transmitted from carrier birds to susceptible penguins by mosquitoes.

The clinical signs of malaria include anorexia, depression, weakness, convulsions, and sudden death. Gross lesions include hydropericardium, epicardial petechia, enlarged, soft friable spleen, swollen and pale liver, ascites, and pulmonary edema. Histopathology is characterized by reticuloendothelial cell hyperplasia and the presence of intracytoplasmic schizonts in those cells.

Antemortem diagnosis is hampered by the exoerythrocytic nature of plasmodia in penguins rather than intraerythrocytic trophozoites found in other species. There is a low peripheral blood parasitemia in penguins. Lymphocytosis causes a marked leukocytosis. Young penguins are most susceptible to malaria. Older birds rarely exhibit clinical signs of malaria, particularly birds that have survived infection.

Treatment should be instituted as soon as malaria is diagnosed. Oral administration of 0.3 mg/kg primaquine phosphate daily for 10 days, combined with an initial dose of 10 mg/kg chloroquine phosphate, followed by additional doses of 5 mg/kg chloroquine at 6, 10, and 24 hours.[7]

Noninfectious Diseases

Nonifectious diseases of penguins include trauma, frostbite, dehydration, ingestion of foreign bodies, oil contamination of the feathers, and poisoning. Poisonings may occur from accumulation of heavy metals or pesticides in the food fishes, and the ingestion of oil following an oil spill. Disinfectants and cleaning agents used in excessive concentrations and with incomplete rinsing may pose a risk. The signs of toxicity in penguins are not pathognomonic, but may include anorexia, diarrhea, depression, trembling, muscle spasms, and convulsions.

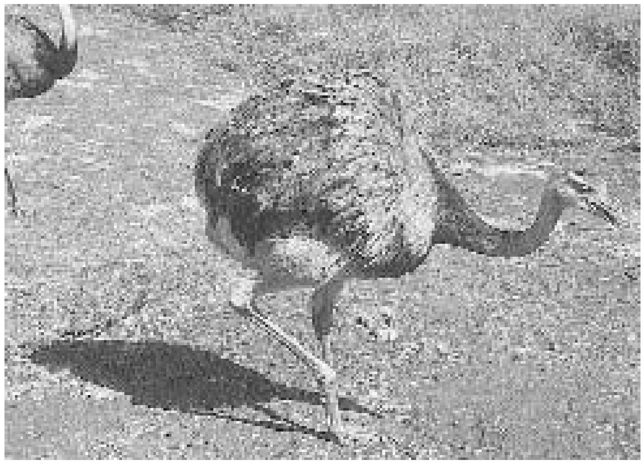
Preventive Medicine

Minimizing stress and providing proper nutrition are the keys to healthy penguins. An annual physical examination including, baseline clinical hematology and serum chemistry should be conducted to provide the clinician with a comparison should a penguin require examination and treatment. Fecal flotations are necessary to monitor the parasite burden, and if necessary anthelmintic agents may be administered orally in the food fish. No specific immunizations are routinely administered to penguins.

Prophylactic therapy for the prevention of malaria may be necessary during the mosquito season for penguins that are housed outdoors. Oral administration of primaquine phosphate at 1.25 mg/kg daily is the medication of choice. The prophylactic administration of antibiotics and antifungal medication is not warranted.

REFERENCES

1. Dann, P.; Norman, I.; and Reilly, P. Eds. 1995. Penguins—Ecology and Management. Chipping Norton, New South Wales, Australia, Surrey Beaty and Sons.
2. Ellis, S.; Croxall, J.P.; and Cooper, J. Eds. 1998. Penguin Conservation Assessment and Management Plan Report. Gland, Switzerland, IUCN/SSC, Conservation Breeding Specialist Group.
3. Fowler, M.E. 1978. Penguins, cranes, storks and flamingos. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 1st Ed., Philadelphia, W.B. Saunders, pp. 153–164.
4. Martinez, I. 1992. Order Sphenisciformes. In J. del Hoyo, A. Elliott, and J. Sargatal, eds., Handbook of the Birds of the World, Vol. 1. Barcelona, Spain, Lynx Edicions, pp. 140–161.
5. Reidarson, T.H.; McBain, J.; and Burch, L. 1999. A novel approach to the treatment of bumblefoot in penguins. Journal of Avian Medicine and Surgery 13(2):124–127.
6. Stoskopf, M.K. 1993. Penguin and alcid medicine. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 3rd Ed. Philadelphia, W.B. Saunders, pp. 189–194.
7. Stoskopf, M.E.; and Kennedy-Stoskopf, S. 1986. Aquatic birds (Spenisciformes, Gaviiformes, Podicepediformes, Procellariiformes, Pelecaniformes and Charadriiformes). In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 293–313.
8. Williams, A.J.; Cooper, J.; and Newton, I.P. 1985. Penguins of the World: A Bibliography. Cambridge, British Antarctic Survey, Natural Environment Research Council.
9. Williams, T.D. 1995. The Penguins, Spheniscidae. Oxford, Oxford University Press.
10. Wilson, R.; and Wilson, M-P.T. 1990. Foraging ecology of breeding *Spheniscus* penguins. In L.S. Davis and J.T. Darby, eds., Penguin Biology. New York, Academic Press, pp. 181–206.
11. Woehler, E.J. 1993. The Distribution and Abundance of Antarctic and Subantarctic Penguins. Cambridge, Scientific Committee on Antarctic Research.



7
Order Rheiformes (Rheas)

Alejandro Scataglini
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BIOLOGY OF THE SOUTH AMERICAN RATITES

Alejandro Scataglini and Marcela V. Torti

TAXONOMY

The family Rheidae is endemic to the neotropical zoogeographical region and is among the oldest avian families of South America.

The rheas (*Rhea americana* and *Pterocnemia pennata*) are related to the other living families of large flightless birds such as the ostrich (*Struthio camelus,* Struthionidae) of Africa, the emu (*Dromaius novaehollandiae,* Dromaiidae) of Australia and New Guinea, the cassowary (*Casuarius* sp.) of Australia and New Zealand, and the small Kiwi (*Apteryx* sp., Apterygidae) of New Zealand.

Although multiple classification schemes exist, the following classification system is used in this text: order, Rheiformes and family, Rheidae.

Two species of rheas have been recognized, in two genera. In each species, many subspecies have been identified.

GENUS *RHEA* (BRISSON, 1760), *RHEA AMERICANA* (LINNÉ, 1758): GREATER RHEA, COMMON NANDU, AMERICAN OSTRICH

Description

Length is 1270–1400 mm. Weight is 20–25 kg. Sexes are similar, but males are somewhat larger and darker than females. General color is gray or grayish brown above, whitish below; the crown, nape, base of neck, and upper back usually are dark brown or black, and the neck is sometimes extensively a whitish color. Entirely white individuals are common. Irises are brown; bill and legs are yellowish brown. Subspecies are supposedly separated by the extent of black on the neck, color of the interscapular region, and to a lesser extent, size (Figure 7.1).

Distribution

Rheas are found in grassy plains and open brush, locally to an altitude of 2000 m in eastern and central Brazil, the Bolivian Chaco, Paraguay, Uruguay, and southward in Argentina to Rio Negro.

Habitat

Rheas are typically found in steppes, savannas, and open, high grass plains (Argentine pampas, Brazilian campos, and cerrado). They are also found in groves

FIGURE 7.1. Common rhea, *Rhea americana.* (Courtesy of M. Dubrowsky.)

and forests, such as open chaco woodland, and are normally in areas with at least some tall vegetation, tending to avoid open short grassland. For breeding, rheas prefer to be near a river, lake, or marsh.

Feed and Feeding

Rheas are omnivorous. Diet includes leaves (even thistles), seeds, roots, and fruits. They also eat insects, especially grasshoppers, and small vertebrates, such as lizards, frogs, small birds, and some snakes; sometimes they catch flies that have gathered around carrion. When feeding, they tend to move continuously. They ingest pebbles to help crush food in the ventriculus.

General Habits

Rheas exhibit a gregarious but loosely cohesive social structure, which is a weak, male-dominant social hierarchy during the nonbreeding winter season. Except between the most dominant males, individual distances are maintained with little aggression.[3] Flocks of 15–40 birds have been seen.[2]

With the first warm days of late winter and early spring in the Southern Hemisphere, the large flocks of greater rheas disperse. The adult males are the first to leave as aggression increases.[2]

During the breeding season, the social structure consists of three categories: single males, small groups of females (2–15) with one or two males, and large flocks of yearlings (up to 40) together with some nonbreeding adults.[2] Hanford and Mares[13] detected another category, groups formed of single females.

Breeding takes place from August to January, depending on the region and climatic conditions. Males are simultaneously polygynous, females serially polyandrous.

Courtship display begins when the dominant male manages to assemble a group of 2–12 females. The male zigzags around the group of females, sounding the typical call, the courtship "boom," with his neck erect and inflated and his wings elevated. Finally, the male stands beside the females with his neck slightly lowered in a U shape and with the feathers of the neck and head bristled.

Copulation is initiated by the female. She solicits the male by sitting until he moves toward her and mounts. The female lays flat on the ground as the male grasps the feathers on the back of her neck with his bill. Copulation lasts from 40 to 140 seconds. In one flock of seven females, the average copulation continued for 2 minutes.[1] The length of time between five copulations was 48, 60, 70, and 180 minutes.[3] Females begin laying eggs approximately 25 days after copulation.[8]

The nest is a shallow depression, usually sheltered in vegetation. Eggs are golden yellow when laid, but soon fade to dull white. In size, the eggs average 132 (90 mm and weigh 600 g. Usually, 13–30 eggs(occasionally as many as 80) are laid per nest, by as many as 12 different females. Incubation of the eggs and caring for the chicks are carried out exclusively by males. The incubation period is 35–40 days. Chicks are grey with dark stripes. They grow rapidly and are almost half-grown by the age of 3 months, and reach sexual maturity at 12–20 months.

Conservation

Greater rheas are not globally threatened, but some populations have declined markedly and are now considered threatened. Decline is partly the result of hunting for meat and partly the result of the colossal export of skins, with over 50,000 traded in 1980, most apparently originating from Paraguay, with Japan and the United States as leading consumers. The race *Rhea americana albescens* is listed in Convention on International Trade in Endangered Species of Wild Fauna and Flora II (CITES II) and all other races in CITES III for Uruguay. The entire species should be listed on CITES II to avoid confusion and curb excessive levels of trade. In addition, the species has been included in the Red Data Book of Argentina (Libro Rojo de Mimíferos y Aves Amenazados de la Argentina) under the category LRpv (low risk, potentially vulnerable).[11]

Nowadays, the conversion of large fields from a traditional system of cattle pasturage to one of intensive cultivation has generated several problems in the wild populations of greater rheas in Argentina, Brazil, Paraguay, and Uruguay.

In Argentina, a total of 897 rheas have been registered in captivity.[14]

GENUS *PTEROCNEMIA* (G.R. GRAY, 1871), *PTEROCNEMIA PENNATA* (ORBIGNY, 1834): DARWIN'S RHEA, LESSER RHEA, CHOIQUE, SURI

Description

Darwin's rhea is 925–1000 mm long and weighs 15–25 kg. Sexes are similar. In the adult, feathering covers the thighs and top of the tarsi (in front). The female is generally duller and has fewer and smaller white spots on the back than the male. Juveniles are brown, without white spotting. The typical adult plumage develops gradually and is completed in the third or fourth year. Some subspecies are grey, with reduced areas of white spots and fewer frontal scutes on the tarsus.

Distribution

The distribution area of the lesser rhea is more restricted than that of the greater rhea. It is found in southern Peru, Bolivia, Chile, and Argentina. Another population exists in southern Chile and Argentina. In 1936, a small population was introduced into northern Tierra del Fuego, where it now is well established.

Three subspecies have been recognized: *P. pennata pennata* is found in southern Chile, from southern Aysén to the Strait of Magellan and in the Patagonian lowlands of Argentina to southern Mendoza province. *P. pennata garleppi* is found in southern Peru, southwestern Bolivia, and northwestern Argentina. *P. pennata tarapacensis* is found in the Puna zone of Chile and in the Atacama desert.

Habitat

Northern subspecies live in the desertlike salt puna, pumice flats, upland bogs, and tola (*Lepidophyllum*) heath in the altiplano, at altitudes of 3500–4500 m. In the south, the race *P. pennata pennata* occupies the shrub steppe and grassland of the flood plains, from sea level up to 2000 m. They usually breed in upland areas of bunch grass.

Food and Feeding

Darwin's rheas are omnivorous, with feeding habits similar to those of the greater rhea. They frequently associate with grazing South American camelids.

Breeding

Breeding takes place from September to January in northern populations, but begins in July in Río Negro, Argentina, and in November in the extreme south. Males are simultaneously polygynous and females are serially polyandrous. The nest is a scrape, lined with dry grass or twigs. Eggs are yellowish olive-green when laid, but fade to buff, averaging 127 × 87 mm in size (*P. pennata pennata*). Normally 10–30, but sometimes as few as 6 or as many as 50 eggs are laid per nest, by several different females. Incubation and chick care are carried out exclusively by the male. The incubation period is 40 days; chicks are greyish brown with blackish stripes and fully feathered tarsi. They reach sexual maturity at 24–36 months.

General Habits

Rheas are sedentary. The southern populations generally move into the uplands for breeding. Normally, they form groups of 5–30 birds.

Conservation

Although not globally threatened, populations of Darwin's rhea have declined markedly and are now considered threatened. The Puna rhea (*P. pennata tarapacensis*/*P. pennata garleppi*) is endangered as a result of intensive hunting pressure, which masks the possible effects of habitat alteration. Their presence in Tierra del Fuego is still fairly common, perhaps because of the inaccessibility and bleak conditions that prevail in much of its range. The upland breeding habit may be a result of agricultural development and hunting.

The Red Data Book of Argentina includes *P. pennata pennata* and *P. pennata garleppi* in the category LRpv (low risk, potentially vulnerable).[11]

Argentina has a total of 493 lesser rheas (*P. pennata pennata*) registered in captivity.[14]

REFERENCES

1. Brito, P. de M. 1949. Observaçóes sábre o comportamento e a reproducao da ema, *Rhea americana americana* (Linnaeus, 1758) em cautiveiro. Boletim do Museu Nacional, Nova Serie Zoologia N. 89:1–9.
2. Bruning, D.F. 1994. Social structure and reproductive behavior of the greater rheas. Living Bird 13:251–293.

3. Bruning, D.F.; and Dolensek, E.P. 1986. Ratites (Struthioniformes, Casuariiformes, Rheiformes, Tinamiformes and Apterygiformes). In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 277–297.
4. Carbajo Garcia, E.; Castello Fontova, F.; Castello Llobet, J.A.; Gurri Lloveras, A.; Marin, M.; Mesiá Garcia, J.; Sales, J.; and Sarasquetta, D.V. 1997. Cría de Avestruces, Emúes y ñandúes. Barcelona, Spain, Real Escuela de Avicultura, p. 421.
5. Cajal, J.L. 1994. Programa Nacional de Conservación y Manejo del ñandú *(Rhea americana*) en la República Argentina. Buenos Aires, Subsecretaría de Recursos Naturales de la Nación, Secretaría de la CITES y FUCEMA.
6. Cracraft, J. 1974. Phylogeny and evolution of ratite birds. Ibis 116:494–521.
7. Cracraft, J. 1981. Towards a phylogenetic classification of the recent birds of the world (class Aves). Auk 98:681–714.
8. Dani, S. 1993. A ema (*Rhea americana*): Biologia, manejo e conservaçao. In Coleçao Manejo da Vida Silvestre, No. 1. Belo Horizonte, Minas Gerais, Brazil, Fundacao AçANGAÚ, p. 136.
9. del Hoyo, J.; Elliot, A.; and Sartagal, J. Eds. 1992. Handbook of the Birds of the World, Vol. 1. Barcelona, Spain, Lynx Edicions.
10. Gallaway, B.J.; Patton, J.C.; Coldwell, K.; and Sealey, W. 1995. Ratite genetics. In C. Elrod and H. Wilnorn, eds., Ratite Encyclopedia. San Antonio, Texas, Ratite Records, pp. 63–77.
11. García Fernández, J.J.; Ojeda, R.A.; Fraga, R.M.; Díaz, G.B.; and Baigún, R.J. Comp. 1997. Libro Rojo de Mamíferos y Aves Amenazados de la Argentina. Buenos Aires, FUCEMA, p. 221.
12. Guittin, P. 1985. Les Struthioniformes en Parc Zoologique, Reproduction, Croissance, Elevage. Doctoral Thesis, Scienses, Universite Paris VII.
13. Handford, P.; and Mares, M.A. 1985. The mating systems of ratites and tinamous: An evolutionary perspective. Biological Journal of the Linnean Society 25:87–92.
14. Scataglini A.D.; and Torti, M.V. 1997. Registro Nacional de Aves Corredoras en Cautiverio, No. 2. Buenos Aires, Jardín Zoologico de la Ciudad de Buenos Aires, pp. 1–32.
15. Sibley C.G.; and Ahlquist, J.E. 1981. The phylogeny and classification of the ratite birds as indicated by DNA-DNA hybridization: Evolution today. In Proceedings Second International Congress of Systematics and Evolution of Birds. Pennsylvania. pp. 301–335.
16. Sibley C.G.; and Ahlquist, J.E. 1983. The phylogeny and classification of the ratite birds as indicated by DNA-DNA hybridization. Current Ornithology 1:245–292.
17. Sibley C.G.; and Ahlquist, J.E. 1990. Phylogeny and Classification of Birds: A Study in Molecular Evolution. New Haven, Connecticut, Yale University Press, pp. 72–88.
18. Sibley C.G.; and Monroe, E.L. 1990. Distribution and Taxonomy of Birds of the World. New Haven, Connecticut, Yale University Press.

HELMINTH PARASITES

Isaú Gouveia Arantes

The parasite-host relationship under natural conditions seems to be such that such infections are either well tolerated by their hosts or may not cause nosological problems. Inherent factors due to confinement (stress, nutritional problems, and inadequate management) seem to favor the parasites, culminating in severe diseases caused by organisms that were supposed to be less pathogenic in field conditions. This fact has been continually observed in ratite farms from Brazil (*Rhea americana* and *Rynchotus rufescens*) with helminthiasis outbreaks leading to high chick mortality rates. (See Table 7.1.)

NEMATODIASIS OF THE GASTROINTESTINAL TRACT OF RHEA

Sicariosis

Affecting mainly young birds of 1–5 months old, the Habronematinae nematodes (Nematoda: Habronematoidea) *Sicarius uncinipenis, Sicarius waltoni,* and *Odontospirura zschokkei,* are found at the mucosa and submucosa over the muscular area of the stomach and proventriculus. As parasites migrate through mucosa and submucosa, a traumatic irritating action causes hemorrhages and erosions that are easily seen. Larval forms penetrate the submucosa of the organ. The microscopic changes that are due to their presence at the submucosa of the ventriculus are listed as follows: proliferative inflammatory reaction with prevailing mononuclear cells and/or

TABLE 7.1. Helminth parasites of rheas

Helminth	Target Organ
Nematoda	
Sicarius uncinipenis	Stomach muscles
Sicarius waltoni	Stomach muscles
Odontospirura zschokkei	Stomach glands
Ascaridia orthocerca	Small intestine
Deletrocephalus dimidiatus	Large intestine
Deletrocephalus cesarpintoi	Large intestine
Paradeletrocephalus minor	Large intestine
Syngamus trachea	Trachea
Dicheilonema rheae	Abdominal air sacs
Acanthocephala	
Prosthorhynchus rheae	Small intestine
Cestoda	
Houttuynia struthionis	Small intestine
Chapmania tauricolis	Small intestine

fibroblasts; presence of numerous viable mastocytes, and moderate hyperemia, as well as mucous membranes covered by a thickened gelatinous material intermingled with loose cells. The mucosa is covered with a colloidal membrane (glycoprotein membrane) that is darkened and rugous. Sometimes it is found perforated at several locations on its surface (Figure 7.2). Severe infections—300 to 500 nematodes—in young chicks (20–45 days old) may cause stomach muscle debilitation, thus detaching partially the colloidal membrane that exposes the mucous membrane that is congested, hemorrhaged, and ulcerated and covered by a reddish, thick mucus. Infected animals may show clinical signs of anorexia, anemia, apathy, fetid diarrhea, emaciation, cachexia, and death. Young chicks assaulted by severe helminthiasis may have an inflammatory process in the muscles of the stomach correlated with loss of appetite as well as disturbances in digestion. The continuous presence of such signs may explain the poor development of affected rheas.

Diagnosis is possible through ova detection in feces either by floating or centrifugation techniques. Habronematinae ovum is characterized by its elliptical shape (45 μm × 25 μm), thick egg shell, presence of embryo, and two polar formations (Figure 7.3).

FIGURE 7.2. *Sicarius uncinipenis* in the ventriculus of Rhea.

Ascaridiosis

Roundworms are the most common parasite observed in confined birds with access to soil, as well as Strongyloidea nematodes, which constitute the main two groups of ratite helminthes. Moderate infections may cause food malabsorption, emaciation, anorexia, apathy, anemia, debility, and diarrhea. Severe infections may lead to intussusception, intestinal obstruction, and death. Diagnosis may be performed by detecting ova in feces through the flotation method. Ascaridia ova have a thick eggshell and a single cell.

Rheiforms Deletrocephaliasis

Deletrocephalus dimidiatus, Deletrocephalus cesarpintoi, and *Paradelotrocephalus minor* represent an ancient group of helminth parasites that affect ratites. Such disease affects young birds and may cause diarrhea, debility, and high mortality rates in flocks. These species apparently fed on blood and may be related to the anemic

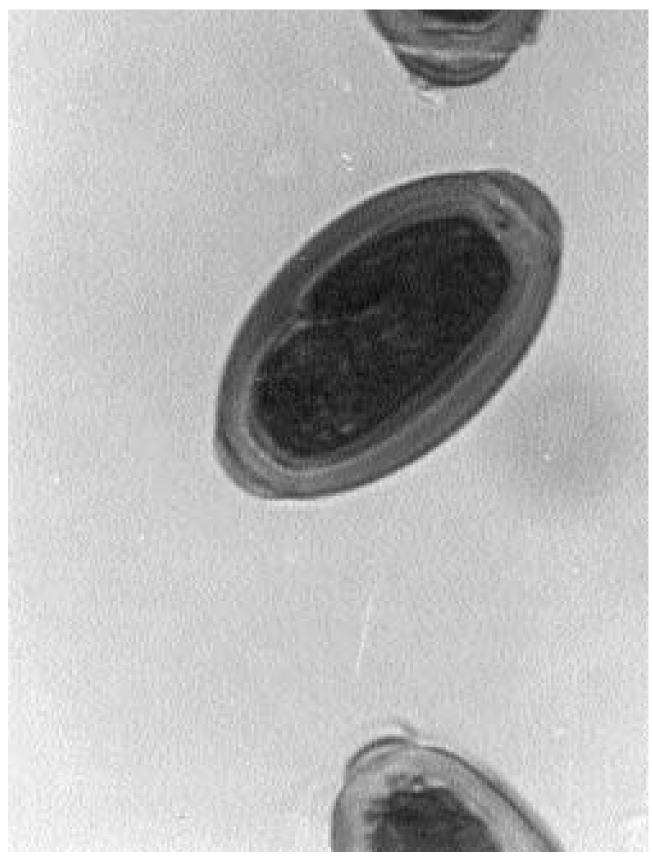

FIGURE 7.3. Eggs of *S. uncinipenis*.

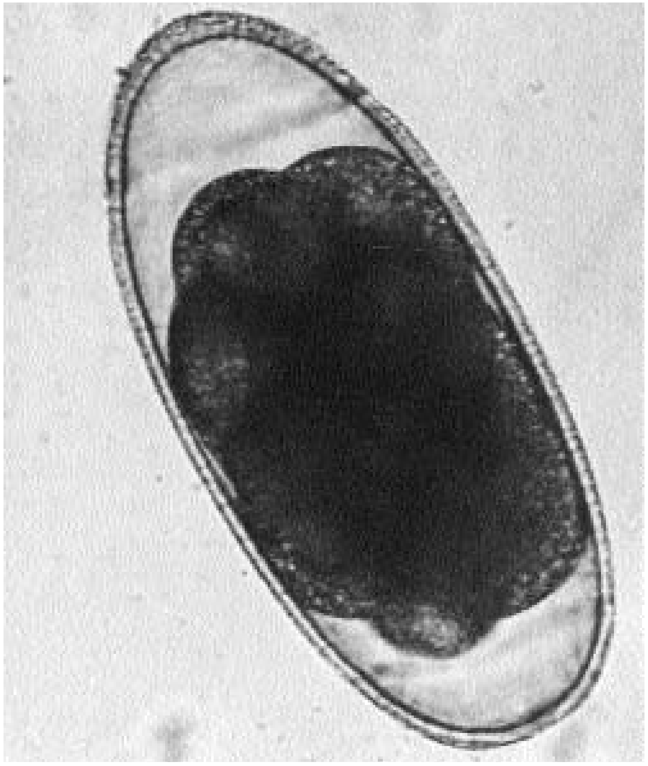

FIGURE 7.4. Eggs of *Deletrocephalus dimidiatus*.

syndrome in rheas when major infections occur. Infections ranging from 500 to 1000 parasites have not induced lesions or visible clinical signs.

Fecal examination using the flotation method may be used as a diagnostic procedure, which reveals characteristic Strongilidae ova: big egg (125–158 μm × 65–75 μm), few big cells, and the presence of an outer and apparently scaly cuticle (Figure 7.4).

The treatment directed against rhea helminthiasis are the following anthelmintics:

- Oral route—Fenbendazole at doses of 15–25 mg/kg body weight or 30–60 parts per million (ppm) mixed in food for 4–5 days; flubendazole, 30–60 ppm in food for 7 days; mebendazole, 15 mg/kg.
- Subcutaneous route—Ivermectin at doses of 0.2 mg/kg.

RESPIRATORY TRACT NEMATODIASIS OF RHEAS

Syngamiasis

Syngamiasis is caused by *Syngamus trachea*. Occasionally, captive young rheas (1–2 months old) may be infected with high numbers of parasites. Host infection takes place through ingestion of free-infecting larvae or even inside the egg, as well as swallowing paratenic hosts such as earthworms, snails, and slugs. One week after ingestion those nematodes may be found in the trachea, growing too quickly and thus obstructing the lumen. Male nematodes are found firmly attached to the trachea mucosa where a nodule is produced by cartilage proliferation. The clinical signs include respiratory distress, apathy, debility, head shaking, and rales, which occur when parasites come to the trachea.

Diagnosis is made through fecal examination using flotation techniques. Eggs pertaining to the genus *Syngamus* are big and double capped. At postmortem examination an inflammatory process may be found at the trachea, showing a lot of thick, reddish mucus, as well as the red-colored parasites, usually found in couples in a Y shape.

Treatment may be carried out with administration of fenbendazole, 25 mg/kg; mebendazole, at a dose of 15 mg/kg or 60 ppm in the food for 6 days or even 120 ppm in the food for 3 days.

Dicheilonemiasis

This disease is caused by *Dicheilonema rheae,* the biggest nematode of South America, which may reach 50–100 cm in length and which is observed frequently in the air sacs of captured birds. There is no information available concerning its pathogenicity. This giant filaria seems to induce no clinical signs, because diseased birds may run at high speeds seemingly fully healthy.

Diagnosis is possible through embryonating egg detection in the feces by centrifugation methods. Dicheilonema eggs are long elliptical shaped (60 μm × 44 μm) with a thick shell and are embryonate when laid (Figure 7.5).

FIGURE 7.5. Eggs of *Dicheilonema rheae*.

DIGESTIVE TRACT CESTODIASIS OF RHEAS

Cestodiasis may be caused by *Houttuynia struthionis* and by *Chapmania tauricollis,* which are found in the small intestines of their hosts promoting digestive disturbances as well as gradual emaciation and high mortality rates for young birds. Diagnosis is easily made by finding proglottids in the feces or by fecal examination for characteristic eggs (Figure 7.6 and 7.7), using flotation techniques. The treatment includes praziquantel, 7.5 mg/kg and fenbendazole, 15–25 mg/kg.

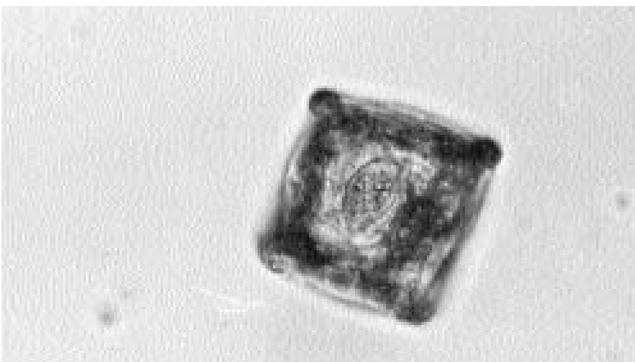

FIGURE 7.6. Eggs of cestoda.

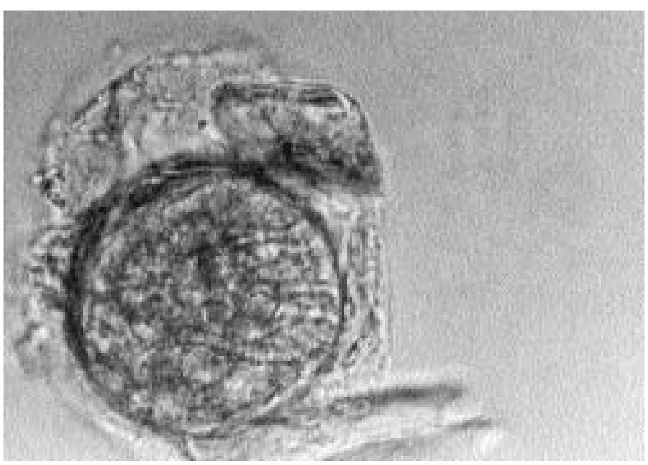

FIGURE 7.7. Eggs of cestoda.

REFERENCES

1. Bruning, D.F.; and Dolensek, E.P. 1986. Ratites (Struthioniformes, Casuariiformes, Rheiformes, Tinamiformes and Apterygiformes). In Fowler, M.E., ed., Zoo and Wild Animal Medicine. 1st Ed., Philadelphia, W.B. Saunders, p. 285–286.
2. Chabaud, A.G. 1986. No. 3 keys to the genera Spirurida, part 2. Spiruroidea, Habronematoidea and Acurioidea. In R.C. Anderson, , A.G. Chabaud, S. Willmott, and S. Cih, eds., Keys to the Nematode Parasites of Vertebrates. Wallingford, U.K., CAB International, pp. 29–58.
3. Craig, T.M.; and Diamond, P.L. 1996. Parasites of Ratites. In T.N. Tully, Jr., and, S.M. Shane, eds., Ratite. Management Medicine and Surgery, 1st Ed. Malabar, Florida, Krieger Publishing, pp. 115–126.
4. Euzeby, J. 1961. El Parasitismo en Patologia Aviar. 1st Ed. Zaragoza, Spain, Acribia.
5. Euzeby, J. 1961. Les Maladies Vermineuses des Animaux Domestiques et Leurs Incidences sur la Pathologie Humaine, Vol. 1, 1st Ed. Paris, Vigot Frères.
6. Ewing, M.L.; Yonson, M.E.; Page, R.K.; Brown, T.P.; and Davidson, W.R. 1995. *Deletrocephalus dimidiatus* infestation in an adult rhea (*Pterocnemia pennata*). Avian Diseases 39:441–443.
7. Freitas, J.F.T.; and Lent, H. 1947. "Spiruroidea" parasitos de "Rheiformes" (Nematoda). Memorias do Instituto Oswaldo Cruz 45(4):743–760.
8. Greiner, E.C. 1997. Parasitology. In R.B. Altman, ed., Avian Medicine and Surgery, 1st Ed., Philadelphia, W.B. Saunders , pp. 332–349.
9. Greiner, E.C.; and Ritchie, B.W. 1994. Parasites. In B.W. Ritchie, G.J. Harrison, and , L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 1007–1029.
10. Jensen, J.M. 1993. Infection and parasitic diseases of Ratites. In M.E. Fowler, ed., Zoo and Wild Animal Medicine: Current Therapy, 3rd Ed. Philadelphia, W.B. Saunders, pp. 200–203.
11. Stewart, J.S. 1994. Ratites comparative medicine and management. In: B.W. Ritchie, G.J. Harrison, and , L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth. Florida, Wingers Publishing, pp. 1314–1316.
12. Stonebreaker, R. 1997. Ratites. In R.B. Altman, ed., Avian Medicine and Surgery, 1st Ed. Philadelphia, W.B. Saunders, pp. 929–932.
13. Wit, J.J. 1995. Mortality of rheas caused by a *Syngamus trachea* infection. The Veterinary Quarterly 17(1):39–40.
14. Yamaguti, S. 1961. Systema Helminthum: The nematodes of vertebrates, Vol. 3, Pt. 1. New York, Interscience Publishers, Inc.

8
Order Tinamiformes (Tinamous)

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BIOLOGY

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The order Tinamiformes is composed of only one family, Tinamidae (nine genera, 48 species), ranging from Mexico to southern South America.[18, 21] Tinamous vary little in shape, but size and weight vary among the genera, from the large grey tinamou (*Tinamus tao*) that weighs 2.3 kg (5 lbs) with a length of 500 mm (20 in.) to the tiny dwarf tinamou (*Taoniscus nanus*), that weighs only 40 g (1.4 oz) and measures only 150 mm (6 in.).[10] All species except those of the genus *Tinamus* pass their life cycle on the ground. *Tinamus* spp. use perches to roost. Tinamous occupy almost all the terrestrial environments of the neotropics. They are found from the Andean deserts, 5300 m (17,388 ft) above sea level [Puna tinamou (*Tinamotis pentlandii*)],[10] to the Atlantic forest at sea level [solitary tinamou (*Tinamus solitarius*)].

Tinamous are diversified in the Amazon region, where most of the species of the genera *Tinamus* and *Crypturellus*[18, 19] may be found. These two genera contain 26 of the 48 species currently accepted.[18, 21]

The body shape of tinamous resembles that of galliforms, and the first explorers that came to New World called these birds "quail" or "partridges" (both belonging to the family Phasianidae). However, the name "quail" or "partridge" is based on the superficial resemblance, and is unrelated to any phylogenetic relationship.

The bill of most species is weak, used only to search for food among the litter. However, the red-winged tinamou (*Rhynchotus rufescens*) and the spotted nothura (*Nothura maculosa*) have stronger bills, adapted to excavating the soil.[19] The bill is relatively short, slightly curved (more curved in *Nothoprocta* spp. and the red-winged tinamou), and the base is always covered by cere. The nares are located at variable points of the extension of the bill.[10] The head is small and some species have a small crest (crested tinamou, *Eudromia* spp.), the neck is slender, and the small size of the neck feathers emphasizes this characteristic.[10] The body is rounded and seems to be compact. The upper tail feathers are long and may pass over the rectrices. Tinamous have aftershaft, powder feathers, and a rudimentary uropygial gland.[16,18] The feathers have a kind of implant that resembles that of pigeons and doves (Columbiformes), because they loosen easily when tinamous are caught by a predator, which usually gets only the feathers while the bird escapes.[10]

The plumage of tinamous is cryptic, an efficient camouflage in the darkness of forests or open areas. The colors are shades of gray, brown, and rufous, with streaks that imitate the pattern of light and shadow of their habitats. The tarsi are particularly colorful: yellow in the white-bellied nothura (*Nothura boraquira*), green in the brown tinamou (*Crypturellus obsoletus*), red in the small-billed tinamou (*Crypturellus parvirostris*), and purplish in the tataupa tinamou (*Crypturellus tataupa*). The bill is also colored (e.g., red in *Crypturellus parvirostris*). The iris may be red (*e.g., Crypturellus obsoletus*), brown (*e.g., Tinamus solitarius*) or yellow (*e.g., Nothura maculosa*).

All species have discreet habits and are detected more easily by their calls than by sight. The birds are usually seen when crossing trails, roads, or when they fly, which occurs only when they cannot run, the preferential way to escape any threat.[3,10,14,19] Flight is a fast vertical take-off, noisy, and continuing horizontally to a protected place (which could be dense brush for species that live in grasslands). Flight is usually in a straight line, with few maneuvers because of the small size of the rectrices. Except for species of the genus *Tinamus,* all tinamous avoid flight, although the pectoral musculature is well developed.

When disturbed by a strange noise or a potential predator, the birds react with various behaviors. Before running or flying, they may try to detect the source of the threat by standing on the tips of the toes, stretching the neck, and freezing,[13] only moving again when they feel secure, or they may cower and freeze, seeking to remain unperceived by the predator (primarily species that inhabit open grasslands).[19] Some species (e.g., *Nothura boraquira*) hide in burrows dug by other animals (e.g., armadillos).[19] Running or flying is a last resort.

Tinamous bathe in small pools or take dust baths, and the plumage may take on the colors of the sediments. The pools in which tinamous bathe are frequently covered by a fine layer of powder from the feathers, a fact well-known by hunters.[3]

As a result of a modification of the scales on the plantar surface of the tarsus, species of the genus *Tinamus* are able to perch to rest or sleep.[3] The tarsus of these birds has rough scales that enable the bird to cling to its perch. The *Tinamus* do not use the toes to perch, rather they rest on the tarsi, balancing over them in a manner singular among birds.[3]

Most tinamous are solitary,[2,13] pairing only during the breeding season. However, some species that live in open habitats, such as the crested tinamous, may live in flocks of as many as 50 birds.[10]

The tinamous are mainly herbivorous, eating roots, leaves, flowers and fruits, and are considered to be good dispersers of seeds, which are found intact in the feces. The red-winged tinamou and the spotted nothura feed in pastures and plantations, eating maize and *Brachiaria* seeds or excavating the soil to get the roots of manioc. Tinamous also may feed on animals, more frequently in the breeding season. The diet is varied, including insects, worms, mollusks, and small vertebrates, such as amphibians and mice. They occasionally follow army ants.[1,3,9,10,14,15,17,19,20,23]

Calls are important in maintaining a territory and in obtaining a mate, becoming intense during the breeding season. The sounds are structurally simple, composed of whistles and trills. Species that inhabit forests tend to have deep-voice calls and those that inhabit open areas have more shrill calls.[19] Males and females have distinct calls, but specific studies of repertoires are rare; only one study dedicated to the vocal repertoire of one species (solitary tinamou)[3] has been published. Twelve distinct calls were identified, and more were suspected. Frequently, only brief descriptions or vocal samples are available to help in field identification.[7]

In places with little seasonality, such as tropical forests, breeding occurs throughout the year,[10,19] whereas in environments with more pronounced seasonality the breeding season is coordinated with food availability.[5,10] The mating system may be either polyandry or poligyny.[19] Courtship is simple, with calls assuming an important role in attracting mates and maintenance of the pair bonds during the breeding season.[19] The males incubate the eggs and rear the chicks.[3,10,14,16,18,19] Clutch size varies from one to eight eggs. More than eight eggs in a nest suggests that multiple females are laying eggs.[3,19] The nest consists of a small depression in the ground, excavated by the male, either at the base of a tree (forest species) or next to a bush (open area species).[10,19] The eggs are brilliantly colored: green (*Tinamus solitarius, Nothocercus bonapartei*), chocolate brown (*Crypturellus parvirostris, Nothoprocta cinerascens*), vinaceus (*Rhynchotus rufescens*), pinkish (*Crypturellus undulatus*), blackish (*Taoniscus nanus*), or yellowish (*Crypturellus erythropus*), and shiny like porcelain or polished metal. Eggs are conspicuous when exposed and easily discovered by predators.[3,10,14,19,20]

Offspring are nidifugous, hatched feathered with open eyes, and follow the father as soon the plumes dry. The plumage of chicks of forest species is in shades of rufous or chestnut, camouflaging the chicks in the darkness of the forest, while chicks of open area species are barred, in shades of brown, blackish and white, following the pattern of light and shadow of the grasslands. The chicks are similar to the offspring of ratites (rheas, ostriches, emus, and cassowaries).[3,10,11,19,23]

Because of the size of the breast musculature, tinamous are hunted for meat by indigenous people. Some species, such as the solitary tinamou, are now rare in most areas of its range. This sensitive species may disappear locally as a result of small alterations in its microhabitat, such as the fall of a great tree. Other species, for example, the lesser nothura (*Nothura minor*) and the dwarf tinamou (*Taoniscus nanus*), may disappear because of intensive modification and destruction of their habitats. Seven species of tinamous are considered to be threatened.[6,9,10,19,20]

Tinamous easily adapt to life in captivity and may be maintained in small enclosures. Because of their shy behavior, it is important that the enclosures offer some type of protection against excessive exposure of the birds. Perches are necessary only for tinamous of the genus *Tinamus*. The floors of the cages must be of soil, with bushes that offer refuge to the birds. Tinamous, when startled or frightened, may explode into rapid flight, and frequently strike hard against the walls of an enclosure, sustaining fractures of the skull and wings, often resulting in death. It is also important to offer protection to nests.

Tinamous may be maintained in pairs or in small groups, but cannibalism is known among the genera *Crypturellus, Rhynchotus, Nothoprocta,* and *Eudromia*. Eggs should be removed to an incubator as soon as they are laid. Newly hatched chicks accept food promptly. Chicks may be maintained in small groups, according to their ages.

Capture and restraint must be gentle to avoid fracturing the wings.

REFERENCES

1. Aguirre, A. 1959. Contribuição para o Estudo da Biologia do Macuco Tinamus solitarius (Vieillot)—Ensaios para a sua Criação Doméstica no Parque "Sooretama." Notas sobre as Outras Espécies do Gênero Tinamus. Rio de Janeiro, Ministério da Agricultura.
2. Azara, F. 1805. Apuntamientos para la Historia Natural de los Páxaros del Paraguay y Rio de la Plata, Vol. 4. Madrid, Imprenta de la Viuda de Ibarra, pp. 148–174.
3. Bokermann, W.C.A. 1991. Observações sobre a biologia do macuco, Tinamus solitarius (Aves—Tinamidae). Doctoral thesis, Instituto de Biociências, Universidade de São Paulo.
4. Brodkorb, P. 1961. Notes on fossil Tinamous. Auk 78:257.
5. Burger, M.I. 1985. Observações preliminares sobre a variação anual no desenvolvimento de testículos de *Nothura maculosa* (Temminck, 1815) (Aves, Tinamidae) no Rio Grande do Sul. Iheringia, ser. misc.1:71–78.
6. Collar, N.J.; Gonzaga, L.A. P.; Krabbe, N.; Madroño Nieto, A.; Naranjo, L.G.; Parker, T.A.; and Wege, D.C. 1992. Threatened Birds of the Americas: The ICBP/IUCN Red Data Book. Cambridge, International Council for Bird Preservation.
7. Hardy, J.W.; Vielliard, J.; and Straneck, R. 1993. Voices of the Tinamous: Order Tinamiformes, Family Tinamidae. Gainesville, Florida, Ara Records. [Cassette.]
8. Houde, P. 1988. Paleognathous birds from the early tertiary of the northern hemisphere. Publications of the Nutall Ornithology Club, Cambridge 22(1):1–148.
9. Höfling, E.; and Imperatriz-Fonseca, V.L. 1984. Aves em cartaz. Ciência Hoje 3(18):98.
10. del Hoyo, J.; Elliot, A.; and Sargatal, J. 1992. Handbook of the Birds of the World: Ostrich to Ducks, Vol. 1. Barcelona, Spain, Lynx Editions, pp. 111–138.
11. Jehl, J.R., Jr. 1971. The color patterns of downy young ratites and tinamous. San Diego Society of Natural History 16(13):291–302.
12. Kufner, M.B. 1997. Alimentacíon de *Eudromia elegans* (Aves, Tinamidae) en el desierto del Monte, Argentina. Iheringia sér. zool. 82:85–91.
13. Lancaster, D.A. 1964. Life history of the Boucard Tinamou in British Honduras, 1, Distribution and general behavior. Condor 66(3):165–181.
14. Maijer, S. 1996. Distinctive song of highland form *maculicollis* of the red-winged tinamou, *Rhynchotus rufescens:* Evidence for species rank. Auk 113:695–697.
15. Olalla, A.M; and Magalhães, A.C. 1956. Biblioteca Zoológica(Vida, Regime, Costumes, Caça, Utilidade e Preparação Taxidérmica (Embalsamação) das Aves e Mamíferos do Brasil, 3. Aves—Família Tinamidae: Inambu Açu, Inambu Azul ou Azulona e Macuco ou Macuca. São Paulo, Biblioteca Zoológica.
16. Penha, J.M.F. 1993. Alimentação de *Rhynchotus rufescens* na serra de São Vicente, município de Santo Antônio de Leverger, Mato Grosso (Tinamiformes-Tinamidae). Ararajuba 3:55–56.
17. Salvadori, T. 1895. Catalogue of the Chenomorphae (Palamedeae, Phoenicopteri, Anseres), Crypturi and Ratitae in the Collection of the British Museum, Vol. 27. London, Longmans and the British Museum Natural History, pp. 496–570.
18. Schubart, O.; Aguirre, A.; and Sick, H. 1965. Contribuição para o conhecimento da alimentação das aves brasileiras. Arquivos de Zoologia 12:95–249.
19. Sibley, C.G.; and Ahlquist, J.E. 1990. Phylogeny and Classification of Birds, Vol. 1. New Haven, Connecticut, Yale University Press, p. 111.
20. Sick, H. 1997. Ornitologia Brasileira. Rio de Janeiro, Nova Fronteira.
21. Silveira, L.F.; and da Silveira, V.J. 1998. The biology of dwarf tinamou *Taoniscus nanus,* with notes on its breeding in captivity. Cotinga 9:42–46.
22. Teixeira, D.M.; and Nacinovic, J.B. 1990. A plumagem natal de *Taoniscus nanus*. Ararajuba 1:113–114.
23. Willis, E.O. 1983. Tinamous, chickens, guans, rails and trumpeters as army ant followers. Revista Brasileira de Biologia 43(1):19–22.

RED-WINGED TINAMOU (*RHYNCHOTUS RUFESCENS*)—UTILIZATION FOR MEAT PRODUCTION

Maria Estela Gaglianone Moro

Among the family Tinamidae, the red-winged tinamou is a species of interest for commercial production because it adapts to captive conditions easily, produces high-quality meat, and has an optimum feed-to-meat conversion ratio.

BREEDING

Enclosures for breeding red-winged tinamou may be simple or deluxe. Chicks between 1-30 days old require special protection from cold winds. Chick brooders for domestic poultry are adequate. At 30 days of age, tinamou are adaptable to the facilities available.

FEEDING

Numerous studies of the nutrition and feeding of red-winged tinamous have been conducted.[5,10,14,15] It has been determined that protein levels should be varied during the growth period. Birds from 1-4 weeks of age require feed with a 24% protein level, birds 5-8 weeks, 20% protein, and birds 9-20 weeks, 16% protein. Adult birds should be fed a diet with 15% protein. The average age to sexual maturity is 10 months.

Energy consumption on a 15% crude protein diet should be between 2650 and 2800 Kcals/kg of body weight. Tinamous prefer large granules to small granules and yellow-colored granules rather than red or green granules.

FERTILITY

Infertility rates of eggs laid and incubated by free-ranging tinamous is 5.3%. If eggs are collected in the wild and incubated artificially, 12% are infertile.[4] Eggs laid by captive tinamous are 30-65% infertile. These figures indicate a need for investigation of factors impinging on breeding programs.

The average weight of hatched chicks is 38.6 g (±4.22 g).[3,8,9] Male juveniles weigh 389.16 g at 10 weeks of age, and females weigh 500.83 g.

CARCASS UTILIZATION

Reports in the literature have provided a large body of data concerning meat utilization from wild or semi-tamed animals. Researchers in Canada used a Chilean tinamou *Nothropocta perdicaria* to demonstrate its potential for meat production. Aggrey et al.[1] worked with 28 captive birds and concluded that tinamous are slow to reach marketable weight when compared with chickens or turkeys. At slaughter and without skin, the birds had an average weight of 455.8 g with 76.8% meat utilization. The leg and breast muscles are composed of white, translucent meat with carcass utilization similar to commercial broilers and turkeys. The breast meat was 138 g in weight, and 40.7% of the total eviscerated body was of superior-quality meat.[1] Another evaluation for commercial production of *N. perdicaria* meat demonstrated that a 450 g bird has a meat utilization of 77%, 40.7% of which is breast meat.[7] These studies show that this bird has either the same or better meat utilization than other wild or domestic birds that have already been raised commercially.

NUTRITIONAL CONTENT

The average nutritional values for red-winged tinamou meat compounds are 62.39% moisture of the thigh and leg and 55.96% for breast meat. Lipids are 5.56% and 1.60%, respectively—1.38% and 1.22% ash, 25.16% and 29.14% crude protein, and 234 mg and 70 mg/100g cholesterol—attesting to its excellent quality, which is demanded by merchants. Tinamou produces a high-quality meat with low cholesterol levels and high nutritive value.[12]

These data confirm that red-winged tinamou, even though it has not undergone selection for any of those features, is a species that should be genetically researched to maximize meat production.

REFERENCES

1. Aggrey, S.E.; Nichol, C.R.; and Cheng, K.M. 1992. The partridge tinamou for commercial meat production: Preliminary evaluation. In World's Poultry Congress 19, Amsterdam, Anais, p. 360.
2. Bokermann, W.C.A. 1991. Observações sobre a Biologia do Macuco *(Tinamus solitarius)* [Observation on the Biology of the Solitary Tinamou]. Doctoral thesis, Ciências, Instituto de Biociências, Universidade de São Paulo.
3. Carnio, A.; Moro, M.E.G.; and Giannoni, M.L. 1998. Estudos para criação e reprodução em cativeiro da ave

silvestre, *Rhynchotus rufescens* (Tinamiformes) [Study of reproduction and production of wild birds in captivity, red-winged tinamou]. In Ars Veterinária 48-A, pp. 45–48.
4. Cravino, J.L. Ed. La Martineta, *Rhynchotus rufescens,* cria y explotacion [The red-winged tinamou, *Rhynchotus rufescens,* breeding and exploitation]. Montevideo, Agropecuaria Hemisferio Sur.
5. Cromberg, V.U.; Moro, M.E.G.; and Toledo, L.M. 1996 Seletividade no forrageamento de perdizes (*Rhynchotus rufescens*), em três frações granulométricas de uma ração farelada [Food selectivity in red-winged tinamou, comparing three different granule sizes] (summary). In Congresso de Etologia. Uberlândia, Anais, p. 339.
6. Cromberg, V.U.; Moro, M.E.G.; and Toledo, L.M. 1997. Efeito da cor da ração na quantidade ingerida por perdizes (*Rhynchotus rufescens*) [The influence of color on the consumption of food by red-winged tinamou] (summary). In 6° Congresso Brasileiro de Ornitologia. Belo Horizonte, Anais, p. 123.
7. Kermode, D.; Blair, R.; Paulson, S.G.; and Cheng, K.M. 1995. Evaluation of two commercially available diets for partridge tinamou meat production (abstract 38). Poultry Science 7 (suppl. 1):13.
8. Menegheti, J.O.; Frozi, M.; and Burger, M.I. 1985. The growth curve of the red-winged tinamou *(Rhynchotus rufescens,* Temminck, 1815) (Aves, Tinamidae). Iheringia ser. misc., 1:47–54.
9. Moro, M.E.G. 1991 Análise citogenética e alguns aspectos produtivos da espécie *Rhynchotus rufescens*(Perdiz (Aves: Tinamidae*)* [Citogenetic analysis and some aspects on the productivity of red-winged tinamou]. Masters thesis, Zootecnia, Jaboticabal, Faculdade de Ciências Agrárias e Veterinárias, Universidade Estadual Paulista.
10. Moro, M.E.G. 1996. Desempenho e características de carcaças de perdizes *(Rhynchotus rufescens)* criadas com diferentes programas de alimentação [Growth, quality and characteristics of carcasses of red-winged tinamou raised with different food programs]. Doctoral thesis, Zootecnia, Jaboticabal, Faculdade de Ciências Agrárias e Veterinárias, Universidade Estadual Paulista.
11. Moro, M.E.G.; and Giannoni, M.L. 1994. Estudos da *Rhynchotus rufescens*(Perdiz (Aves: Tinamiformes) em cativeiro, 1. Sexagem [Study of the red winged tinamou, in captivity]. Ars Veterinária, 10(1):37–40.
12. Moro, M.E.G.; Ariki, J.; De Souza, P.A.; De Souza, H.B.A.; and Moraes, V.M.B. 1998. Rendimento de carcaça e composição nutritiva da carne da perdiz nativa (*Rhynchotus rufescens*) [Carcass quality and nutritional composition of the meat of red-winged tinamou] (summary). In 35° Reunião da SBZ, Botucatu/SP, pp. 513–515.
13. Moro, M.E.G.; Ariki, J.; Giannoni, M.L.; Malheiros, E.B.; and Junqueira, O.M. 1999. Níveis de proteína em rações para perdizes (*Rhynchotus rufescens*) na fase de crescimento [Protein levels in food of red-winged tinamou during growth]. Applied Poultry Science (in press).
14. Moro, M.E.G.; Tavares, F.A.; and Lima, C.G. 1999. Desempenho reprodutivo da perdiz (*Rhynchotus rufescens*) submetida a diferentes níveis energéticos na ração [Breeding performance of red-winged tinamou under different levels of energy in the food] (summary). In 36° Reunião da SBZ, Porto Alegre/RS, p. 328.
15. Moro, M.E.G.; Ariki, J.; Giannoni, M.L.; and Malheiros, E.B. 1999. Avaliação dos níveis de proteína da dieta sobre a idade à maturidade sexual e produção de ovos de perdiz (*Rhynchotus rufescens*) [Evaluation of protein levels in food in relation to sexual maturity and egg production of red-winged tinamou]. Brazilian Journal of Poultry Science (in press).

HELMINTHIASIS OF TINAMOUS

Adjair Antonio do Nascimento and Isaú Gouveia Arantes

The helminths of tinamiforms have not been well studied, contributing to a lack of information related to their pathogenicity, economic importance, and diagnosis, as well as helminthic control programs. Under natural conditions, the host-parasite relationship seems to be stable, with parasites well tolerated by their host.

Parasites that have been recovered from tinamous are listed in Table 8.1. The clinical signs, if any, and management are similar to protocols for other species of birds.

Treatment

For treatment of gastrointestinal nematodiasis of the tinamiforms, one of the following anthelmintics may be administered orally: fenbendazole at 15–25 mg/kg or at 30–100 mg/kg in the ration, administered for 4–5 days; flubendazole at 30–60 parts per million (ppm) in the ration for 7 days; and mebendazole at 15 mg/kg in the ration. Levamisole at 30 mg/kg and ivermectin at 0.2 mg/kg are administered subcutaneously.

Tetracheilonemiasis of the air sacs of *Rynchotus, Crypturellus,* and *Nothura* caused by *Tetracheilonema* spp. is frequently observed in birds captured from the wild. Macroscopically, lesions are not observed, except for tiny inflammatory nodules found in the air sacs. Diagnosis of the infection may be made by finding eggs on fecal flotation. The eggs of *Tetracheilonema* are elliptic (57–62 mm × 29–39 mm) and embryonated when eliminated in the feces. Because of the parasites' location in the air sacs, administration of anthelmintics is not recommended. Figures 8.1 through 8.11 illustrate some parasites of tinamous.

TABLE 8.1. Helminth parasites of tinamiforms

Species	Affected Organ	Host
Capillaria penidoi (N)	Esophagus and oral cavity (mucosa of inferior part) crop	*Nothura maculosa*
Acuaria posthelica (N)	Esophagus and oral cavity (mucosa of inferior part) gizzard	*Tinamus tao*
Hadjelia curvata (N)	Gizzard	*N. maculosa*
Procyrnea sp. (N)	Gizzard	*Crypturellus undulatus*
Capillaria crypturi (N)	Small intestine	*T. tao*
Capillaria rudolphi (N)	Small intestine	*T. solitarius*
Ascaridia brasiliana (N)	Small intestine	*N. maculosa, Rhynchotus rufescens*
Tinamustrongylus taotaoi (N)	Small intestine	*T. tao tao*
Ornithostrongylus almeidai (N)	Small intestine	*C. parvirostris, T. major*
Lutzema lutzi (N)	Small intestine	*T. solitarius*
Oswaldostrongylus cruzi (N)	Small intestine	*T. solitarius*
Mediorhynchus pintoi (A)	Small intestine	*N. maculosa*
Brachytaemus centrodes (T)	Small intestine	*C. variegatus, N. maculosa, T. solitarius*
Echinostoma siticulosum (T)	Small intestine	*C. undulatus, C. variegatus*
Stomylotrema sp. (T)	Small intestine	*C. undulatus*
Subulura olympioi (N)	Large intestine	*R. rufescens, C. undulatus, N. maculosa, C. parvirostrus*
Subulura strongylina (N)	Large intestine	*Tinamus* spp., *C. undulatus, C. parvirostris*
Heterakis gallinarum (N)	Large intestine	*N. maculosa, R. rufescens*
Heterakis nattereri (N)	Large intestine	*T. solitarius, T. tao tao*
Heterakis inglisi (N)	Large intestine	*C. variegatus, C. parvirostris*
Odontoterakis alata (N)	Large intestine	*C. undulatus, C. noctivagus*
Odontoterakis crypturi (N)	Large intestine	*C. variegatus*
Odontoterakis valvata (N)	Large intestine	*C. cupreus*
Haroldakis multidentata (N)	Large intestine	*C. variegatus*
Tetracheilonema quadrilabiatum (N)	Air sacs	*N. maculosa, R. rufescens, C. parvirostris*
Diplotriaena tinamicola (N)	Air sacs	*Tinamus* sp.
Paratanasia robusta (T)	Kidneys	*C. tataupa, R. rufescens*
Cesartrema pintoi (T)	Cloaca	*T. solitarius*
Hempelia hempeli (N)	Eyes	*N. maculosa, R. rufescens*
Thelazia campanulata (N)	Eyes	*T. tao tao, C. strigulosus*

N, nematoda; T, trematoda; A, acanthocephala

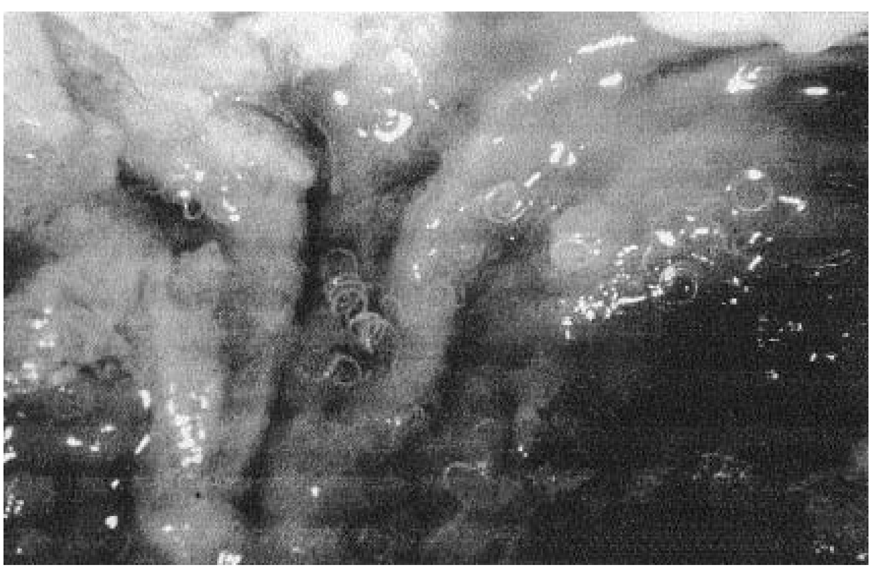

FIGURE 8.1. Thickened crop mucosa with *Capillaria penidoi*.

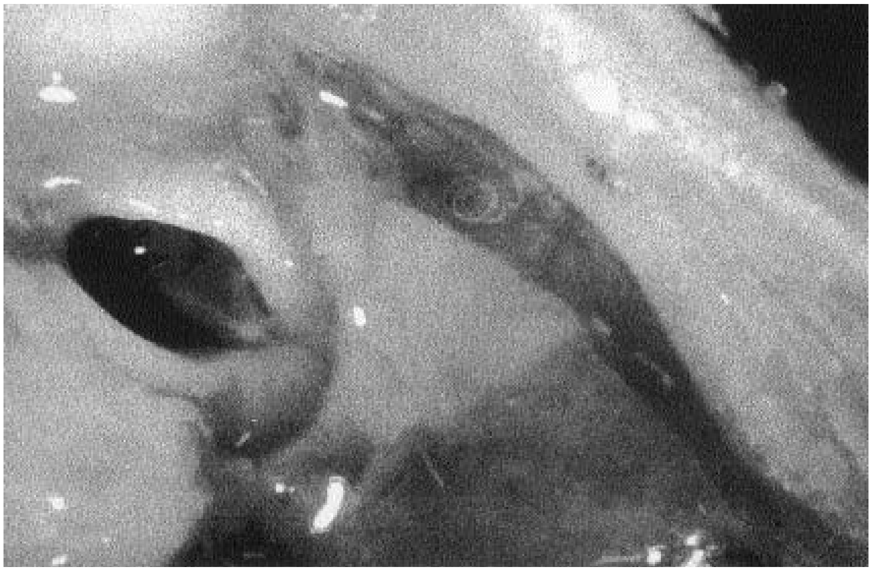

FIGURE 8.2. Oral cavity with *C. penidoi*.

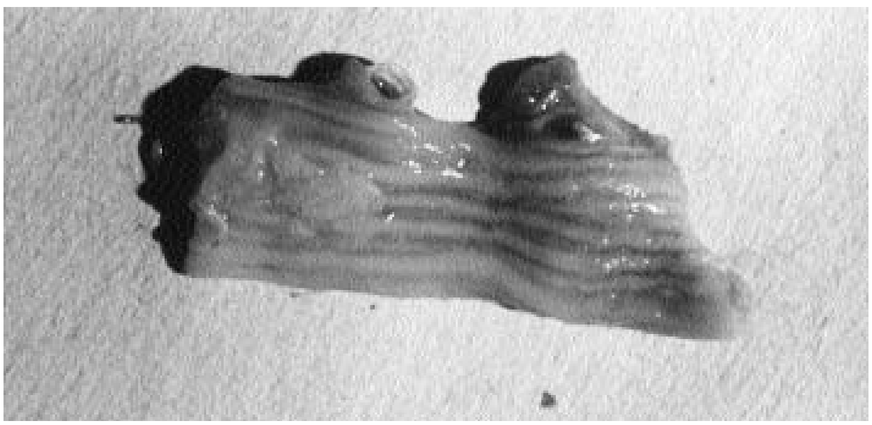

FIGURE 8.3. Thickened esophagus mucosa recovered with mucus and presence of *C. penidoi*.

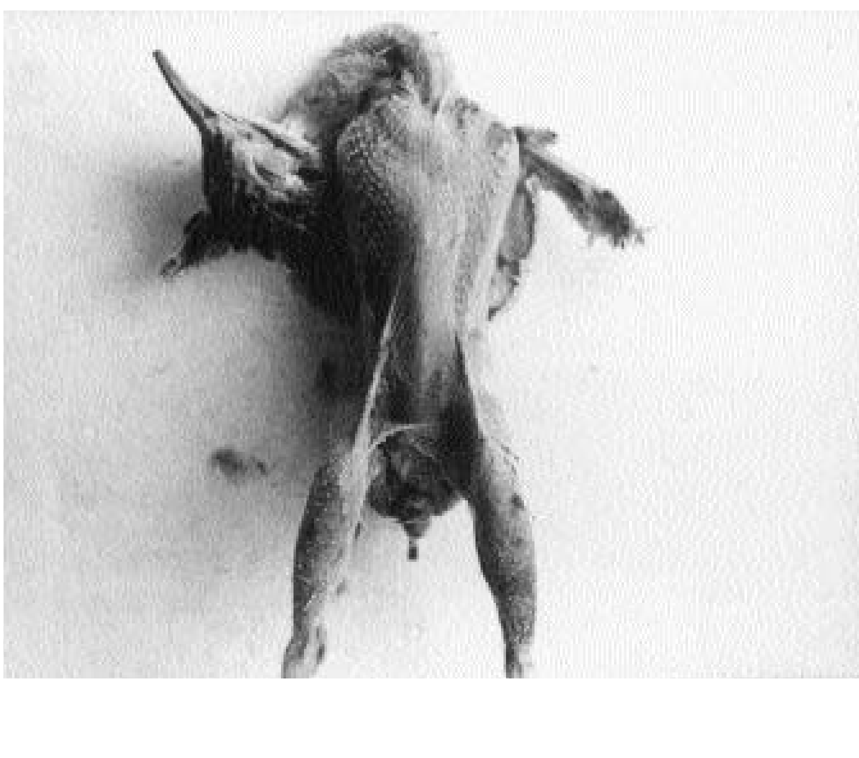

FIGURE 8.4. Cachectic partridge showing dehydration and muscle atrophy.

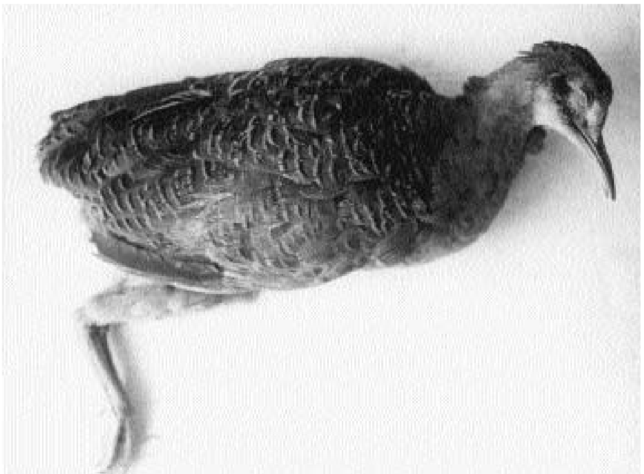

FIGURE 8.5. Partridge, died with 10,300 to 39,200 eggs per gram (epg).

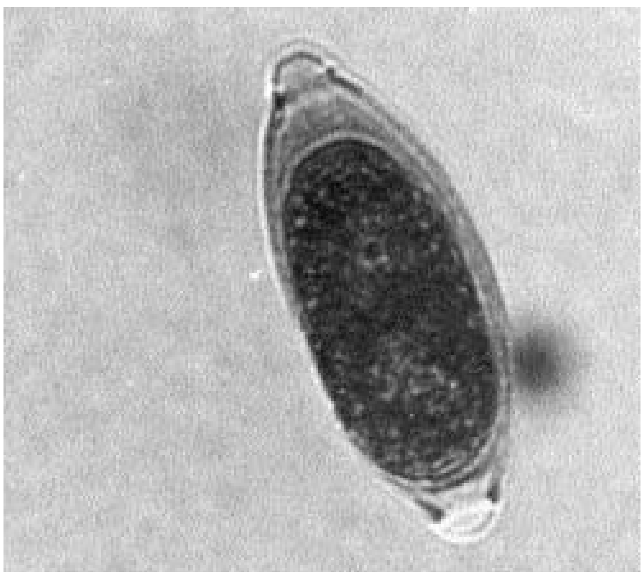

FIGURE 8.6. Egg of *C. penidoi*.

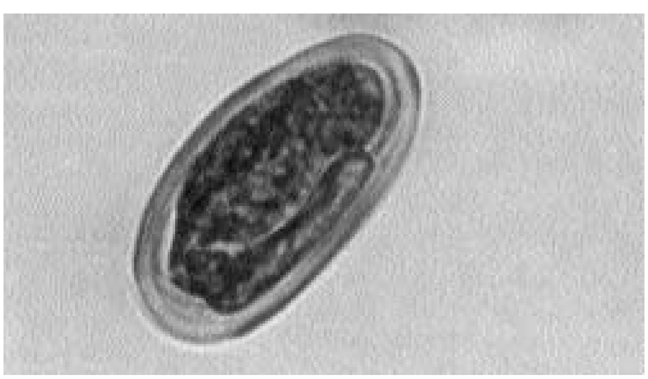

FIGURE 8.7. Egg of Habronematinae.

FIGURE 8.8. Egg of *Subulura olimpioi*.

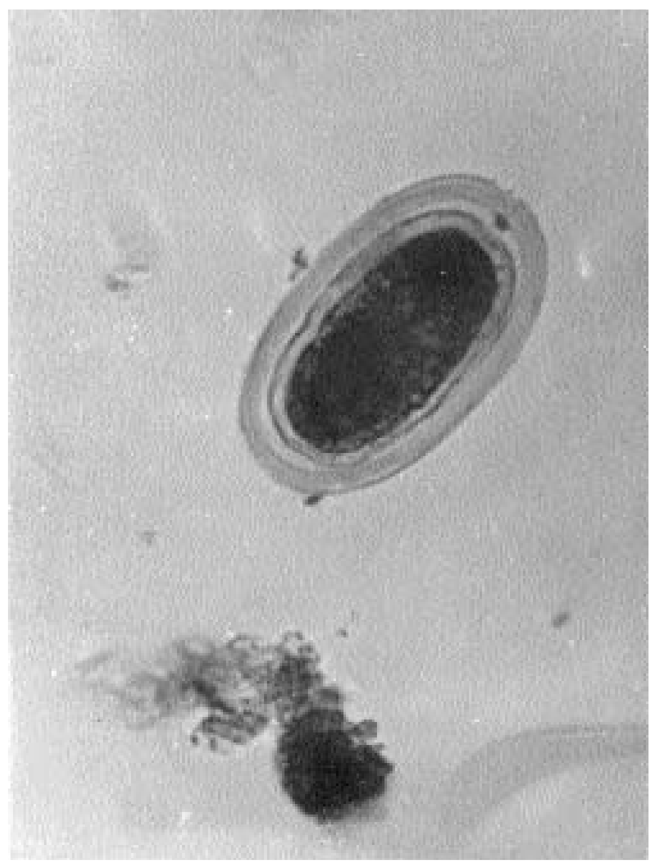

FIGURE 8.9. Egg of *Heterakis* sp.

FIGURE 8.10. Egg of *Tetracheilonema quadrilabiatum.*

FIGURE 8.11. Egg of *Paratanasia robusta.*

REFERENCES

1. Altman, R.B. 1997. Avian Medicine and Surgery. 1st Ed. Philadelphia, W.B. Saunders.
2. Arantes, I.G.; Nascimento, A.A.; Ascari, H.; Tebaldi, J.H.; and Antunes, R.C. 1992. *Tetracheilonema quadrilabiatum* (Molin, 1858) Diesing, 1861 (Nematoda: Filarioidea) Parasitos de Sacos Aéreos de *Rhynchotus rufescens, Nothura maculosa* e *Crypturellus parvirostris* no Estado de São Paulo, Brasil. Revista do Centro de Ciências Biomédicas da Universidade Federal de Uberlândia 8(1):37–43.
3. Bruning, D.F.; and Dolensek, E.P. 1986. Ratites (Struthioniformes, Casuariiformes, Rheiformes, Tinamiformes and Apterygiformes). In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 1st Ed. Philadelphia, W.B. Saunders.
4. Chabaud, A.G. 1976. Keys to genera of the order Spirurida. In R.C. Anderson, A.G. Chabaud, and S. Willmott, eds., CIH Keys to the Nematode Parasites of Vertebrates. Wallingford, U.K., CAB International, pp. 29–58.
5. Euzeby, J. 1961. Les Maladies Vermineuses des Animaux Domestiques et Leurs Incidences sur la Pathologie Humaine, Vol. 1, 1st Ed. Paris, Vigot Frères.
6. Euzeby, J. 1961. El Parasitismo Aviar, 1st Ed. Zaragoza, Spain, Acribia.
7. Freitas, J.F.T.; and Almeida, J.L. 1935. Sobre os nematoda Capillariinae parasitas de esôfago e papo de aves. Memórias do Instituto Oswaldo Cruz 30(2):123–156.
8. Greiner, E.C. 1997. Parasitology. In R.B. Altman, ed., Avian Medicine and Surgery, 1st ed. Philadelphia, W.B. Saunders, pp. 332–349.
9. Greiner, E.C.; and Ritchie, B.W. 1994. Parasites. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing.
10. Nascimento, A.A.; Tebaldi, J.H.; Antunes, R.C.; and Arantes, I.G. 1992. Espécies de *Subulura* Molin, 1860 (Nematoda: Subuluroidea) parasitos de Tinamidae nos Estados de São Paulo e Mato Grosso do Sul. Revista Brasileira de Parasitologia Veterinária 1(2):93–95.
11. Ritchie, B.W.; Harrison, G.I.; and Harrison, L.R. Eds. 1994. Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing.
12. Travassos, L.; Freitas, J.F.T.; and Kohn, A. 1969. Trematódeos do Brasil. Rio de Janeiro, Memórias do Instituto Oswaldo Cruz 67(1):1–885.
13. Vicente, J.J.; Pinto, R.M.; and Noronha, D. 1993. Remarks on six species of heterakid nematode parasites of Brazilian tinamid birds with a description of a new species. Memórias do Instituto Oswaldo Cruz 88(2):271–278.
14. Yamaguti, S. 1961. Systema Helminthum: The Nematodes of Vertebrates, Vol. 3, Pts. 1–2. New York, Interscience Publishers.

9
Order Ciconiiformes (Herons, Storks, Ibises)

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BIOLOGY

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TAXONOMIA

The order Ciconiiformes is represented in South America by three families, 23 genera, and 37 species (Table 9.1).

DISTRIBUTION AND HABITAT

The representatives of the family Ardeidae are broadly distributed, but with the greatest concentration and diversity of species in the tropics. Some genera, such as *Nycticorax,* are cosmopolitan. They occupy aquatic environment most of the time, such as bays, tide pools, groves of mangroves, swamps, rivers, and lakes, from which they obtain food. The birds wade in the water and wait for prey to appear. They feed mainly on fish and crustaceans, but small birds may also be consumed. Some species are not aquatic feeders, such as *S. sibilatrix,* and the cattle egret, *Bubulcus ibis,* which forage in pastures, cultivated fields and suburban zones.

Herons are often harassed because they are thought to compete with fishermen, which is not true, because they consume only small fish at the water surface. Besides, the presence of these birds is essential for the maintenance of balance in the environment. They have also been heavily hunted for feathers for decoration.

Some populations may be decimated by hurricanes and earthquakes. Human activities also cause environmental disturbances in declining populations of Ardeidae, such as modifications in the aquatic habitat, destruction of forests, water pollution and indiscriminate use of pesticides.

The family Ciconiidae has worldwide distribution, except in Antarctica and the Nearctic region, but the largest number of species is found in the tropics. They occupy aquatic habitats. Some species are totally dependent on water (*Mycteria*), but most species may also live and feed in areas where water is scarce.

Threskiornithidae is a family of vast distribution throughout the world, except in Antarctica. The only cosmopolitan member of the family is *P. falcinellus.* Most of these birds live in an aquatic environment, although some species are not necessarily linked to water. They are found in estuaries, coastal ponds, groves of mangroves, swamps, rice fields, river sandbanks, shallow margins and bays of ponds, lakes and

TABLE 9.1. Ciconiiformes of South America

Family	Genus	Species	Distribution
Ardeidae family	*Syrigma*	*S. sibilatrix*	East of Colombia and Venezuela, Bolivia to southeast Brazil, and northwest Argentina.
	Pilherodius	*P. pileatus*	East Panama to South Bolivia and Paraguay.
	Ardea	*A. herodias*	Southeast Alaska to Central America, Venezuela's island and Galapagos.
		A. cocoi	The major part of South America, excluding the Andes.
	Egretta	*E. alba*	Some regions of Europe, Asia and Africa, north USA to south Argentina.
		E. rufescens	South USA to north Colombia and Venezuela.
	Egretta	*E. caerulea*	South USA to south Peru, Bolivia and, south Brazil.
		E. thula	West USA to south Chile.
		E. tricolor	South coast of USA to north Peru. East coast of USA to northeast Brazil.
	Bubulcus	*B. ibis*	Some regions of Europe, Africa, Asia, and Australia. South Canada to northeast Argentina.
	Butorides	*B. striatus*	Some regions of Africa, Ásia, and Australia. West USA to north Argentina.
	Agamia	*A. agami*	South Mexico to east Bolivia, north and central Brazil.
	Nycticorax	*N. violaceus*	East Coast of USA to East coast of Brazil. West Coast of USA to Coast of Peru. Galapagos.
		N. nycticorax	Regions of Africa, Asia, and Europe. North Canada to south South America.
	Cochlearius	*C. cochlearius*	Center and west of Mexico to northeast of Argentina.
	Tigrisoma	*T. mexicanum*	West and east coasts of Mexico to northwest Colombia.
		T. fasciatum	Costa Rica to north Bolivia. Southeast Brazil to northeast Argentina.
		T. lineatum	Southeast Mexico to west Equador and northeast of Argentina.
	Zebrilus	*Z. undulatus*	East Colombia to central north Brazil. Southwest and east Peru to northeast Bolivia.
	Ixobrychus	*I. involucris*	North Colombia and Venezuela, regions of Guyana. South Bolivia and Brazil to central Argentina. Central Chile.
		I. exilis	South Canada to southeast Brazil and Paraguay and Central west Peru.
	Botaurus	*B. pinnatus*	East Mexico to northeast Argentina.
Ciconiidae family	*Mycteria*	*M. americana*	Southeast USA to north Argentina.
	Ciconia	*C. maguari*	East Andes, Venezuela to Argentina.
	Jabiru	*J. mycteria*	Mexico to north Argentina and Uruguay.
	Theristicus	*T. caerulescens*	Bolivia and central Brazil to Paraguay, north Argentina, and Uruguay.
Threskiornithidae family		*T. caudatus*	Colombia to north Argentina and Uruguay.
		T. melanopis	Ecuador, Peru, northwest Bolivia and north Chile. South Chile and Argentina.
	Cercibis	*C. oxycerca*	East Colombia, Venezuela, and Guyana. Regions of Brazilian Amazonia.
	Mesembrinibis	*M. cayennensis*	East Costa Rica to northeast Argentina.
	Phimosus	*P. infuscatus*	Northeast Colombia to south Amazonia. Central east of Brazil to northeast Argentina and Uruguay.
	Eudocimus	*E. albus*	East and west coasts of south USA to Colombia. Northwest Venezuela, west Equador and northwest Peru.
	Eudocimus	*E. ruber*	Northeast Colombia and east Ecuador and north Venezuela to delta of Amazon River. Southeast Brazil.
	Plegadis	*P. falcinelus*	Some regions of Africa, Europe Ásia, and Australia. Atlantic Coast of North America and Venezuela.
		P. chihi	Central USA to south Central America, Southeast Bolivia, Paraguay, and south Brazil to central Chile, Argentina, and Uruguay.
		P. ridgwayi	Central Peru to south Bolivia, north Chile, and northwest Argentina.
	Platalea	*P. ajaja*	Southeast coast of USA to north Argentina (to east Andes). West Ecuador and northwest Peru.

reservoirs. At times they forage in savannas and pastures (*Theristicus*). The majority of these birds live in open areas, but some species live exclusively in dense forests, such as *M. cayennensis*. Some species occur in high altitudes (*T. melanopis* at more than 5000 m and *P. ridgwayi* at 3500–4800 m).

These birds are susceptible to environmental changes, tourism, excessive fishing, hunting, collection of eggs, and pollution. Studies of a population of *E. ruber* in Venezuela demonstrated that in 1983 there were 22 colonies composed of about 65,439 pairs. The following year, a repeat census found only seven colonies with 42,236 pairs.

REFERENCES

1. Antas, P.T.Z. 1979. Breeding the scarlet ibis *Eudocimus ruber* at the Rio de Janeiro Zoo. International Zoo Yearbook 19:135–139.
2. Bates, J.M.; Garvin, M.C.; Schmitt, D.C.; and Schimtt, C.G. 1989. Notes on the bird distribution in northeastern department Santa Cruz, Bolívia, with 15 species new to Bolívia. Bulletin of the British Ornithology Club 109(4):236–244.
3. Cracraft, J. 1967. On the systematic position of the boat-billed heron. Auk 84:529–533.
4. Cracraft, J. 1981. Towards a phylogenetic classification on the recent birds of the world (Class Aves). Auk 98:681–714.
5. Custer, T.W.; Osborn, R.G.; and Stout, W.F. 1980. Distribution species abundance and nesting-site use of Atlantic Coast colonies of herons and their allies. Auk 97:591–600.
6. Elbin, S.B.; and Lyles, A.M. 1994. Managing colonial waterbirds: the scarlet ibis *Eudocimus ruber* as a model species. International Zoo Yearbook 33:85–94.
7. Evans, K. 1983. Hand-rearing a scarlet ibis, *Eudocimus ruber,* at Padstow Bird Gardens, Cornwall. Avicultural Magazine 84(4):215–217.
8. Frederick, P.C.; and Bildstein, K.L. 1992. Foraging ecology of seven species of neotropical ibises (Threskiornithidae) during the dry season in the Llanos of Venezuela. Wilson Bulletin 104(1):1–21.
9. Hancock, J.; and Kushlan, J. 1984. The Herons Handbook. London, Croam Helm.
10. del Hoyo, J.; Elliott, A.; and Sargatal, J. 1992. Handbook of the Birds of the World, Vol. 1. Barcelona, Spain, Lynx Editions.
11. King, C.E.; and Coulter, M.C. 1989. Status of storks in zoos: 1987 survey. International Zoo Yearbook 28:225–229.
12. Lever, K.K. 1980. Habitat utilization in a tropical heronry. Brenesia 17:97–136.
13. Marcondes-Machado, L.O.; and Monteiro-Filho, E.L.A. 1990. The scarlet ibis *Eudocimus ruber* in southeastern Brazil. Bulletin of the British Ornithologists' Club 110(3):123–126.
14. Powell, G.V.N. 1987. Habitat use by wading birds in a sub-tropical estuary: implications of hydrography. Auk 104:740–749.
15. Sick, H. 1997. Ornitologia Brasileira. Rio de Janeiro, Nova Fronteira.
16. Sprunt, A.; Ogden, J.C.; and Winckler, S. 1978. Wading Birds. Research Report 7. New York, National Audubon Society.
17. Thomas, B.T. 1986. The behavior and breeding of adult Maguari Storks. Condor 88:26–34.

MANAGEMENT IN CAPTIVITY

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Birds in this order are often maintained in captivity because they are popular with the public and are also subjects for captive propagation for reintroduction to the wild.[3] These birds are of interest to world conservation organizations such as the ICBP/IWRB Specialist Group on Storks, Ibises and Spoonbills, and the American Association of Zoological Parks and Aquariums (AAZPA) Stork Taxon Advisory Group.[2]

South American Ciconiidae are rarely exhibited outside of South America, and there has been little breeding success. However, these birds have a long life span, more than 25 years.[2]

In South America there are only a few reports of captive hatching. *Mycteria americana* was hatched in the Zoo of Sorocaba, Brazil, in 1987 (Nunes, personal com.).[5] Chicks of *Ciconia maguari* hatched in the Buenos Aires Zoo between 1946 and 1950, and in the Discovery Island Zoo in Florida there was successful hatching in 1991.[2] There has been more success in hatching species of Threskiornithidae. They, too, live long lives; certain South American species have lived more than 30 years in zoos.[1]

Some species reproduce easily in captivity, such as *Eudocimus albus* and *E. ruber,* as well as *Ajaia ajaja,* with hatching occurring in many institutions. For other species there are isolated reports, such as *Theristicus caudatus,* in which a hatching was recorded at the Brasília Zoo in 1992.[4]

REFERENCES

1. Browner, K.; Chifter, H.; and Jones, M.L. 1994. Longevity and breeding records of ibises and spoonbills (Threskiornithidae) in captivity. International Zoo Yearbook 33:94–102.
2. Browner, K.; Jones, M.L.; King, C.E.; and Chifter, H. 1992. Longevity and breeding records of storks (Ciconiidae) in captivity. International Zoo Yearbook 31:131–139.

3. Kawata, K. 1996. Large wading birds in Japanese collections, 1994. International Zoo News 43(2):100–106.
4. Sociedade de Zoológicos do Brasil. 1992. Censo de Animais. São Paulo, Sociedade de Zoológicos do Brasil.
5. Olney, P.J.S. 1989. Birds bred in captivity and multiple generation births. International Zoo Yearbook 29: 257–306.

CICONIIFORMES MEDICINE

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RESTRAINT

Storks and herons have long, sharp bills that are hazardous to the eyes, face, and body of the handler. The bird's head should always be controlled. A small, clear plastic shield should be used to protect the handler's face and eyes when approaching aggressive individuals. If a bird is to be held for a few minutes or is to be transported in cage, the bill may be taped shut. A blunt object, such as a cork or rubber tubing, may be placed over bills that have a sharp tip. Smaller species of this group are easily captured with a small net and can then be held in the hand.[5] Long-legged birds may be carried; the right and left humerus should be held together.[26]

Several methods of deflighting are acceptable for these birds in captivity (see the section on flamingos). Even though properly pinioned, storks may jump over fences and shields if sufficiently excited.[4,5]

Anesthesia and chemical immobilization are similar to procedures used for other avian species. Ketamine hydrochloride at 20 mg/kg[5] is an effective immobilizing agent. Recovery from anesthesia is a dangerous time for long-legged birds because they attempt to stand before being capable of doing so. Placing the bird in a burlap sack so that its head extends through the opening minimizes the risk. The bird's legs must not be cramped, because this may cause a necrotizing myopathy.[5]

Blood may be collected by venipuncture from the radial, saphenous, and jugular veins. Normal values have been reported for some species of Ciconiiformes (Tables 9.2 and 9.3).

TABLE 9.2. Hematologic values for Ciconiiformes

	Night Heron	Little Egret	Cattle Egret	Maguari Stork	Scarlet Ibis
PCV	44.8 ± 3.0	47.7 ± 3.9	45.6 ± 2.7	0.46	0.49 ± 0.05
(L/L)	(38.0 – 51.0)	(38.5 – 55.0)	(41.0 – 50.0)	(0.42 – 0.50)	(0.41 – 0.53)
Hb	13.6 ± 1.1	11.3 ± 1.5	12.6 ± 1.8	16.1	15.3 ± 1.0
(g/100 mL)	(10.7 – 15.4)	(9.1 – 14.9)	(9.1 – 15.5)	(13.8 – 17.8)	(13.3 – 17.1)
RCB	2.861 ± 0.237	2.800 ± 0.221	2.748 ± 0.293	2.3	3.2 ± 0.3
(10^{12} /L)	(2.325 – 3.328)	(2.464 – 3.201)	(2.387 – 3.468)	(2.2 – 2.7)	(2.6 – 3.8)
MCV	156 ± 9	171 ± 15	168 ± 18	195	153 ± 7
(fL)	(141 – 189)	(139 – 194)	(126 – 202)	(186 – 210)	(142 – 164)
MCH	47.5 ± 3.9	40.7 ± 5.7	45.9 ± 6.1	69.0	48.8 ± 2.9
(pg)	(39.7 – 61.8)	(30.4 – 50.6)	(34.5 – 54.3)	(61.3 – 75.7)	(45.2 – 53.4)
MCHC	30.2 ± 1.5	24.0 ± 4.0	27.7 ± 4.5	35.3	31.5 ± 1.3
(g/100 mL)	(27.4 – 32.7)	(18.5 – 33.8)	(19.9 – 33.3)	(32.9 – 36.2)	(29.2 – 33.7)
WBC	8.31± 3.64	6.11 ± 3.19	6.43 ± 4.15	9.8	7.1 ± 3.2
(10^9/L)	(2.13 – 19.0)	(1.83 – 12.37)	(2.00 – 16.75)	(7.2 – 15.5)	(2.6 – 13.6)
Heterophils (%)	68.9 ± 10.7	65.1 ± 11.9	56.2 ± 15.3	5.9[a]	4.5 ± 2.5[a]
	(36.0 – 84.0)	(14.3 – 58.0)	(23.8 – 78.2)	(2.0 – 11.5)[a]	(1.6 – 8.5)[a]
Lymphocytes	29.4 ± 10.5	31.8 ± 11.9	41.3 ± 15.3	2.6[a]	2.0 ± 0.7[a]
(%)	(15.0 – 64.0)	(14.3 – 58.0)	(18.8 – 73.3)	(1.4 – 3.3)[a]	(0.8 – 3.0)[a]
Monocytes (%)	0.8 ± 1.3	1.8 ± 2.2	1.0 ± 0.8		
	(0.0 – 8.0)	(0.0 – 4.1)	(0.0 – 2.4)	(0.0 – 0.7)[a]	0
Eosinophils	0.8 ± 1.3	0.6 ± 1.0	0.9 ± 1.0		
(%)	(0.0 –5.0)	(0.0 – 4.1)	(0.0 – 2.7)	(0.7 – 2.2)[a]	(0.0 – 0.8)[a]
Basophils[a]	0.0 ± 0.1	0.5 ± 0.7	0.6 ± 0.7		
(%)	(0.0 – 1.0)	(0.0 – 2.2)	(0.0 – 1.6)	(0.0 – 0.8)[a]	(0.0 – 0.7)[a]
Thrombos	_	_	_	10	22 ± 8
(10^9/L)				(8 – 11)	(11 – 35)
Fibrinogen (g/L)	_	_	_	1.7	2.6 ± 0.6
				(1.3 – 2.1)	(1.9 – 3.7)
Number	48	28	16	5	15

Source: See references 3 and 9.
[a]10^9/L.

INFECTIOUS DISEASES

Storks and herons have no unique infectious diseases; however, viral, bacterial, and fungus infections occur. Reported diseases include aspergillosis,[4,26] salmonellosis,[14,20] aeromonia,[6] streptococcosis,[8] chlamydiosis,[15] herpesvirus,[7,13] and eastern equine encephalitis.[17,24,26]

Viral hemorrhagic enteritis in storks, caused by a herpesvirus,[7] may occur in endemic to subclinical form. The presence of circulating antibodies does not impede the viremia.[13] Deleterious effects in the natural environment, such as from the use of insecticides, herbicides, and changes of the natural systems, promote a continuous stress situation that may activate latent infections. The virus is mainly shed in the feces. In an outbreak, mortality may be as high as 90%, with sudden death or death after 3 to 4 days. Signs include depression, anorexia, drooping wings, hypothermia, vomiting, and hemorrhagic diarrhea.

Infected storks present moderate leucocytosis with monocytosis, mild lymphocytosis, and neutropenia. Gross lesions include severe hemorrhagic enteritis with occasional necrosis, mainly in the ileum and first portions of the colon. Small necrotic foci may be observed in the liver. Histologically there is intestinal necrosis and mononuclear infiltration of mucous membranes, dilation of the crypts, multifocal necrosis in the spleen, and areas of coagulative necrosis in the liver without an inflammatory reaction. Intranuclear inclusion bodies may be observed in several tissues.

Snowy egret, great egret, glossy ibis, roseate spoonbill, and cattle egret are species susceptible to eastern equine encephalitis (EEE), an alphavirus. Viremia with moderate titers and developed neutralizing antibodies in 2% of the population should cause a species to be considered as a potentially important host in epizootics.[24] Clinical signs of EEE infections vary from clinically inapparent illness to fatal disease.[26] Sick birds may present with anorexia, lethargy, drooping wings, ataxia, and bloody discharge from the mouth. Death may occur 1 to 3 days after the onset of behavioral changes.[17]

Various fish pathogens, such as *Edwardsiella ictaluri* and the infectious pancreatic necrosis virus, have been isolated from the intestinal tract of fish-eating birds, causing enteric septicemia.[16,25]

NUTRITIONAL DISEASES

Metabolic bone disease as a result of calcium deficiency or calcium-phosphorus imbalance has been seen in young birds fed unsupplemented meat or fish diets. The

TABLE 9.3. Blood chemistry values for Ciconiiformes

	Night Heron	Little Egret	Cattle Egret
Urea	1.8 ± 1.3	1.6 ± 0.8	1.6 ± 0.7
(mM/L)	(0.6 – 4.9) *n* = 45	(0.9 – 4.3) *n* = 22	(0.6 – 4.4) *n* = 16
Uric acid	382 ± 129	475 ± 267	226 ± 101
(µM/L)	(167 – 734) *n* = 45	(177 – 1159) *n* = 22	(67 – 403) *n* = 16
Cholesterol	6.2 ± 1.4	9.7 ± 2.4	8.2 ± 2.6
(mM/ L)	(2.4 – 8.5) *n* = 45	(5.6 – 18) *n* = 22	(5.1 – 16) *n* = 16
Triglycerides	0.94 ± 0.2	1.7 ± 0.7	1.4 ± 0.3
(mM/L)	(0.4 – 1.8) *n* = 45	(0.7 – 2.9) *n* = 22	(0.9 – 2.1) *n* = 16
Creatinine	138 ± 182	120 ± 143	90 ± 12
(µM/L)	(58 – 831) *n* = 45	(50 – 708) *n* = 22	(66 – 114) *n* = 16
Glucose	13.3 ± 2.1	16.8 ± 3.3	14.7 ± 4.4
(mM/L)	(8.2 – 18.1) *n* = 45	(11.8 – 23.9) *n* = 22	(6.6 – 19.4) *n* = 16
LDH	534 ± 270	647 ± 358	510 ± 385
(IU/L)	(195 – 1183) *n* = 45	(177 – 1435) *n* = 23	(176 – 1020) *n* = 13
ASAT	78.6 ± 44.4	147.3 ± 79.3	43.2 ± 21.9
(IU/L)	(6.9 – 217) *n* = 45	(32.9 – 350) *n* = 23	(22.8 – 92.8) *n* = 13
ALAT	13.2 ± 6.4	13.9 ± 5.4	19.2 ± 7.9
(IU/L)	(1.6 – 34.6) *n* = 45	(5.3 – 27.3) *n* = 23	(8.3 – 39.6) *n* = 13
CPK	407 ± 296	698 ± 396	528 ± 218
(IU/L)	(112 – 1456) *n* = 45	(229 – 2068) *n* = 23	(293 – 902) *n* = 13
AP	147 ± 76	536 ± 311	54 ± 38
(IU/L)	(37 – 437) *n* = 45	(75 – 1205) *n* = 23	(12.6 – 142) *n* = 13
Albumin	43 ± 7	50 ± 11	48 ± 10
(%)	(30 – 55) *n* = 39	(29 – 67) *n* = 18	(36 – 66) *n* = 13
Proteins	34 ± 5	33 ± 5	34 ± 6
(g/L)	(21 – 43) *n* = 39	(26 – 39) *n* = 18	(25 – 40) *n* = 13

Source: See reverence 17[a].

feeding of red meat or boned fish alone is the most common cause of this nutritional disease[4].

Thiamin deficiencies occur in birds fed fish diets high in thiaminase (e.g., smelt). Frozen fish should be thawed under refrigeration and supplemented with thiamin at a rate of 25–30 mg/kg of fish fed to adults or parent-reared chicks.[26]

Steatitis results from hypovitaminosis E in Ardeidae fed diets composed primarily of species of fish containing high levels of polyunsaturated fats, as well as in those fed with dead rancid fish that may have been polluted with heavy metals.[2,18] Clinical signs include large amounts of firm, lobulated, subcutaneous fat over most of the body, causing the overlying skin to have a nodular appearance, weakness from skeletal muscle atrophy, lethargy, and dehydration. Firm masses may be palpable in the abdominal cavity. The diagnosis is confirmed by biopsy of the fatty tissue and determination of a low level of vitamin E in the serum.

Lesions seen at necropsy are firm and waxy adipose tissues with a mottled yellow-to-brown coloration throughout. The adipose tissue is often lobulated and has a prominent fishy odor. Microscopically, there is widespread necrosis of adipose tissues with the presence of ceroid pigment and multifocal myodegeneration of cardiac and skeletal muscles.[18]

Diets requiring additional vitamin E should be supplemented at a rate of 100 IU/kg of fish.[26] Daily treatment with 400 IU of vitamin E orally for a long period may prevent development of the disease.

POISONINGS

Sodium chloride (NaCl) toxicity occurs in nestlings that still possess an immature renal system and are fed frozen herring in brine.[1] Clinical signs include lethargy, regurgitated food, anorexia, dyspnea, spastic paralysis of the legs, and death. Necropsy findings are marked by bilateral kidney enlargement with pale coloration. Microscopic lesions include mild to severe nephrosis and necrosis of the proximal convoluted tubular epithelium and dilation of the distal convoluted tubules and collecting ducts.

Mercury is used extensively to mine gold in South America. Rivers may be contaminated, and mercury becomes incorporated into the food chain. The Amazon basin receives 90–120 tons of mercury per year.[19] In birds, the mercury concentration may vary seasonally, correlated with the contamination of the species of fish consumed, and distributed irregularly in the brain, muscles, liver, and feathers.[11] Elevated concentrations in the liver (>6 parts per million [ppm]) predispose the bird to chronic diseases. At concentrations larger than 25 ppm, renal degeneration and articular gout were observed.[23]

Organochloride pesticides are still used in South America and may cause a decline in fish-eating bird populations from death, as well as poor hatching success because of eggshell thinning.[10]

TRAUMA

Some species of Ciconiiformes, particularly birds in or near urban areas, are frequent victims of traumatic accidents or aggression. Fractures may occur from vehicle collisions or by flying into electrical transmission lines. Nerves may be traumatized, causing paralysis that may persist after resolution of the fracture. Verification of fibrillation potential may be determined by electromyography 2–5 weeks after the injury to determine whether or not the nerve is likely to become functional.[12] Standard orthopedic techniques of wiring, plating, or intramedullary pinning may be employed.[22] Bumblefoot lesions are common in storks kept continuously on hard surfaces such as concrete cage floors.[4]

REFERENCES

1. Bennett, D.C.; Bowes, V.A.; Hughers, M.R.; and Hart, L.E. 1992. Suspected sodium toxicity in hand-reared great blue heron (*Ardea herodias*) chicks. Avian Diseases 36:743–748.
2. Carpenter, J.W.; Spann, J.W.; and Novilla, M.N. 1979. Diet-related die-off of captive black-crowned night herons. Proceedings American Association Zoological Veterinarians pp. 51–55.
3. Celdrán, J; Polo, F.J.; Peinado, V.I.; Viscor, G.; and Palomeque, J. 1994. Haematology of captive herons, egrets, spoonbills, ibis and gallinules. Comparative Biochemistry and Physiology A 107(2):337–341.
4. Coulter, M.C.; Balzano, S.; Johnson, R.E.; King, C.E.; and Shannon, P.W. Eds. 1989. Conservation and Captive Management of Storks. Proceedings International Workshop at the New York's Zoological Society's Wildlife Survival Center, and Saint Catherine's Island, Georgia.
5. Fowler, M.E. 1986. Storks and flamingos (Ciconiiformes and Phoenicopteriformes). In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 327–331.
6. Glünder, G.; and Siegmann, O. 1989. Occurrence of *Aeromonas hydrophyla* in wild birds. Avian Pathology 18:685–695.
7. Gómesx-Villamandos, J.C.; Hervás, J.; Salgueiro, M.; Quevedo, M.A.; Aguilar, J.M.; and Mozos, E. 1998. Haemorrhagic enteritis associated with herpesvirus in storks. Avian Pathology 27:229–236.

8. Greenwood, A.G.; Marshall, J.; and Tinsley, E.G.F. 1996. Vegetative endocarditis in a Waldrapp ibis. Avian Pathology 25:387–391.
9. Hawkey, C.M.; and Samour, H.J. 1988. The value of clinical hematology in exotic birds. In E.R. Jacobson and G.V. Kollias, Jr. eds., Contemporary Issues in Small Animal Practice, Vol. 9. Exotic Animals. New York, Churchill Livingstone, pp. 109–141.
10. Henny, C.J.; Blus, L.J.; Krynitsky, A.J.; and Bunck, C.M. 1984. Current impact of DDE on black-crowned night herons in the intermountain west. Journal Wildlife Management 48(1):1–13.
11. Hoffman, R.D.; and Curnow, R.D. 1979. Mercury in herons, egrets and their foods. Journal of Wildlife Management 43(1):85–93.
12. Holland, M.; and Jennings, D. 1997. Use of electromyography in seven injured wild birds. Journal of the American Veterinary Medical Association 211(5):607–609.
13. Kaleta, E.F.; and Kummerfeld, N. 1986. Persistent viremia of a cell-associated herpesvirus in white storks (*Ciconia ciconia*). Avian Pathology 15:447–453.
14. Kirkpatrick, C.E. 1996. Isolation of *Salmonella spp.* from a colony of wading birds. Journal of Wildlife Diseases 22(2):262–264.
15. Locke, L.N. 1987. Chlamydiosis. Field guide to wildlife diseases. In M. Friend, ed., General Field Procedures and Diseases of Migratory Birds, Vol. 1. National Wildlife Health Center, Madison, Wisconsin, pp. 107–113,
16. McAllister, P.E.; and Owens, W.J. 1992. Recovery of infectious pancreatic necrosis virus from the feces of wild piscivorous birds. Aquaculture 106:227–232.
17. McLean, R.G.; Crans, W.J.; Caccamise, D.F.; McNelly, J.; Kirk, L.J.; Michell, C.J.; and Calisher, C.H. 1995. Experimental infection of wading birds with eastern equine encephalitis virus. Journal of Wildlife Diseases 31(4):502–508.
18. Nichols, D.K.; Campbell, V.L.; and Montali, R.J. 1986. Pansteatitis in great blue herons. Journal of the American Veterinary Medical Association 189(9):1110–1112.
19. Nriagu, J.O.; Souza, C.M.M.; and Mierle, G., 1992. Mercury pollution in Brazil. Nature 356:389–390.
20. Nunes, N.A.L.V. 1996. Levantamento da flora bacteriana em aves da família Psitacidae e Ardeidae no Zoológico Sorocaba, SP no período de 1988 a 1995. In Anais do 20° Congresso Brasileiro de Zoológicos do Brasil. SZB, Cuiabá, Brazil.
21. Polo, F.J.; Celdrán, J.; Viscor, G.; and Palomeque, J. 1994. Blood chemistry of captive herons, egrets, spoonbills, ibis and gallinules. Comparative Biochemistry and Physiology A 107(2):343–347.
22. Redig, P.T. 1986. A clinical review of orthopedic techniques used in the rehabilitation of raptors. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 388–401.
23. Spalding, M.G.; Bjork, R.D.; Powell, G.V.N.; and Sundlof, S. 1994. Mercury and cause of death in great white herons. Journal of Wildlife Management 58(4):735–739.
24. Spalding, M.G.; McLean, R.G.; Burgess, J. H.; and Kirk, L.J. 1994. Arboviruses in water birds (Ciconiiformes, Pelecaniformes) from Florida. Journal of Wildlife Diseases 30(2):216–221.
25. Taylor, P.W. 1992. Fish-eating birds as potential vectors of *Edwardsiella ictaluri*. Journal of Aquatic Animal Health 4:240–243.
26. Wallach, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals. Philadelphia, W.B. Saunders.

PARASITES AND PARASITIC DISEASES OF SOUTH AMERICAN CICONIIFORMES

Luciano Antunes Barros

INTRODUCTION

The Ciconiiformes are generally associated with an aquatic environment and consume primarily fish and crustaceans. Ciconiiformes are a good indicator of environmental conditions related to pollution. Little is known about the role Ciconiiformes may play as reservoirs for pathogenic microorganisms.

The parasitic fauna affecting these birds attracts attention because of the wide variety of species of internal parasites (trematodes, cestodes, nematodes, acanthocephalans, and protozoa) and external parasites (lice, louse fly, and mites). However, little is known about the pathological importance of these parasites. This may be explained by the fact that probably most of the parasites have little pathogenic effect, due to long-time parasite–host relationships, in which these parasites became adapted to their hosts and the signs of organic problems in the birds are no longer easily noticed. Another probable explanation is the lack of studies on the pathological effects of these parasites on birds. The difficulties faced in handling the birds and performing serologic tests also contribute negatively to the gathering of information.

PARASITIC FAUNA

External and internal parasites found in Ciconiiformes and their parasitic sites are listed in Table 9.4. Only an appropriate analysis done by a morphologist can definitely identify a species of parasite. It is recommended that all parasites or lesions that may be of parasitic origin be preserved appropriately and sent to specialists at reference institutions.

TABLE 9.4. Parasites found in South American Ciconiiformes

Category	Scientific Name	Location of Adult
External—Louse flies	*Columbicola* spp.	Skin and feathers
	Lynchia albipennis	Same
	Menopon spp.	Same
	Olfersia spp.	Same
	Ornithoctona erythrocephala	Same
	Ornithoica confluenta	Same
External—Mites	*Ardeaarus ardeae*	Skin
	Neardeacarus sp.	Skin
Trematodes	*Ascocotyle* spp.	Intestine
	Cathaemasioides callis	Intestine
	Clinostomum spp.	Ventriculus
	Clinostomatopsis sorbens	Same
	Cloacitrema oswaldoi	Cloaca
	Echinochasmus dietzevi	Same
	Echinostoma spp.	Same
	Episthmium spp.	Bursa fabricii
	Ignavia venusta	Kidney
	Ithyoclinostomum dimorphum	Esophagus
	Lyperosomum sinuosum	Bile duct and bladder
	Mesaulus grandis	Intestine
	Nephrostomum limai	Same
	Opithorchis sp.	Bile duct
	Parorchis proctobium	Bursa fabricii and cloaca
	Parastrigea brasiliana	Intestine
	Patagifer consimilis	Same
	Phagicola longus	Same
	Philoophtalmus lachrymosus	Conjunctival sac
	Prohemistomum odhneri	Intestine
	Prosthogonimus ovatus	Oviduct, cloaca
	Posthodiplostomum spp.	Intestine
	Pygidiopsis pindoramensis	Same
	Renicoloa ralli	Kidney
	Ribeiroia insignis	Esophagus
	Sphincterodiplostomum musculosum	Intestine
	Stepanosporora singularis	Same
	Stomylotrema vicarium	Intestine, cloaca
	Strigea bulbosa	Intestine
Cestodes	*Dendroutenia* spp.	Intestines
	Dilepis spp.	Same
	Cyclustera capito	Same
	Paradilepis transfuga	Same
	Prvitaenia spp.	Same
	Proorchida lobata	Same
	Tetracisdicotyla macroscolecina	Same
	Valiporus spinosus	Same
Acathocephalids	*Arhythmorhynchus* sp	Same
	Hystrichis acanthocephalicus	Ventriculus
	Leptorhynchoides sp.	Intestine
	Microsomacanthus sp.	Same
	Polymorphus sp.	Same
	Southwellina hispida	Same
Nematodes	*Capillaria* spp.	Esophagus
	Contracaecum spp.	Ventriculus
	Desmidocercella ardeae	Oral
	Despostesisus invaginatus	Ventriculus
	Echinuria longenavinata	Same
	Eustrongylides ignotus	Oral cavity
	Excisa excisa	Esophagus, ventriculus
	Heterakis valdemucronata	Intestine
	Porrocaecum spp.	Ventriculus
	Tetrameres micropenis	Same
	Thelazia aquilina	Conjunctival sac
	Viktorocara sp.	Ventriculus

CLINICAL AND PATHOLOGICAL ASPECTS

The effects of parasitism that result in pathologies that can be clinically detected in Ciconiiformes deserve larger attention on the part of veterinarians and avian pathologists. There are rare reports of signs, and generally the diagnosis is restricted to necropsy findings. However, for birds maintained in captivity, it is possible to establish diagnostic methods, control protocols, and treatment regimens. The main parasites that affect these birds are listed below including prevalence and description of the pathologies found.

INTERNAL PARASITES

Helminths

The helminthic fauna of Ciconiiformes is composed of parasites that are mainly transmitted by the ingestion of fish that are intermediary hosts, bearing larvae that mature into the adult form of the parasite in the bird.

Interest in the parasites that affect Ciconiiformes has recently increased significantly, not only because of their importance in public health, but also for the need to understand these parasites and their biological and pathological peculiarities. Among countless helminthoses, the following are those of most clinical importance.

EUSTROGILOIDOSIS: INFECTIONS CAUSED BY *EUSTRONGYLIDES IGNOTUS* This disease is caused by a nematode of the family Dioctophymatidae of the genus *Eustrongylides,* generally of the species *E. ignotus,* found in the celomic cavity of Ciconiiformes. It causes erosions in the celomic cavity and usually leads to death from peritonitis.

The larvae of this parasite are found in various species of fish, and they are common in piranhas (*Serrasalmus nattereri*) in Brazil. The dark red larvae may be found in the muscles and/or intestines of these hosts. The ingestion of fish that contain fourth stage larvae is the method of transmission to birds. Some hours after ingestion, the bird's gastric mucosa will be perforated. These larvae cross the wall of the stomach and embed beneath the serosa in other sites in the celomic cavity, where they mature. The parasite is always associated with the production of tubular formations that communicate with the gastric lumen. Through these tubular channels the eggs pass to the intestinal lumen and the feces.

Clinical examinations of carcasses of birds received for necropsies in Florida, USA, discovered lesions that may be detected by touch.[13] The diagnosis may be made by touching the ventral area of the bird, feeling for fibrous lesions between the liver and the surface of the stomach or on the connective tissue between the stomach and the cloaca. This diagnostic method was accurate in 95% of the positive cases.

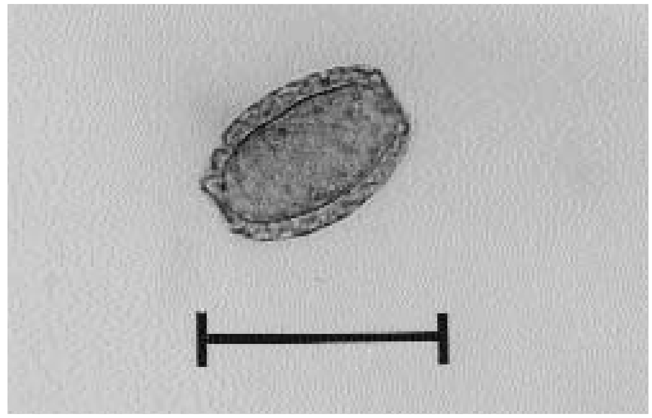

FIGURE 9.1. Egg of *Eustrongylides* in feces of white-necked heron (bar = 0.4 mm).

There is no research into the sensitivity of this parasite to the therapeutic drugs available on the market. The diagnosis should be based on the finding of fibrous lesions and parasite eggs in the feces. The egg of *Eustrongylides* is large, elliptic, and has a thick and irregular shell (0.3×0.18 mm) (Figure 9.1).

POLYMORPHOSIS: INFECTIONS CAUSED BY *POLIMORPHUS SPINDLATUS* *P. spindlatus* is a parasite that attaches to the intestinal mucosa with a set of hooks on its head. Histological studies of affected intestines revealed hemorrhagic processes and intense leukocyte infiltration, causing obstruction of the intestinal lumen and compressing villi in "socós" (*Nycticorax nycticorax*). The head of the parasite penetrates the mucosa and submucosa, stimulating a fibrous reaction.

Generally, there are no signs of diarrhea or prostration. The bird dies suddenly as a result of peritonitis. The diagnosis should be based on the presence in the feces of long, elliptic eggs (0.1×0.02 mm) with a thick shell, containing an embryo (Figure 9.2).

MESAULOSIS: INFECTIONS CAUSED BY *MESAULUS GRANDIS* *Mesaulus* is a large trematode (3–5 cm) found in the intestine of the spoonbill (*Ajaia ajaja*). This parasite has been found in birds from the swampland of Mato Grosso. Its presence in the intestines causes little serious disease; however, when found in the celomic cavity, peritonitis and sudden death results (Figure 9.3). This is an aberrant site for the adult parasite, but it occurs frequently, always causing high mortality rates. Radio-opaque fibrous

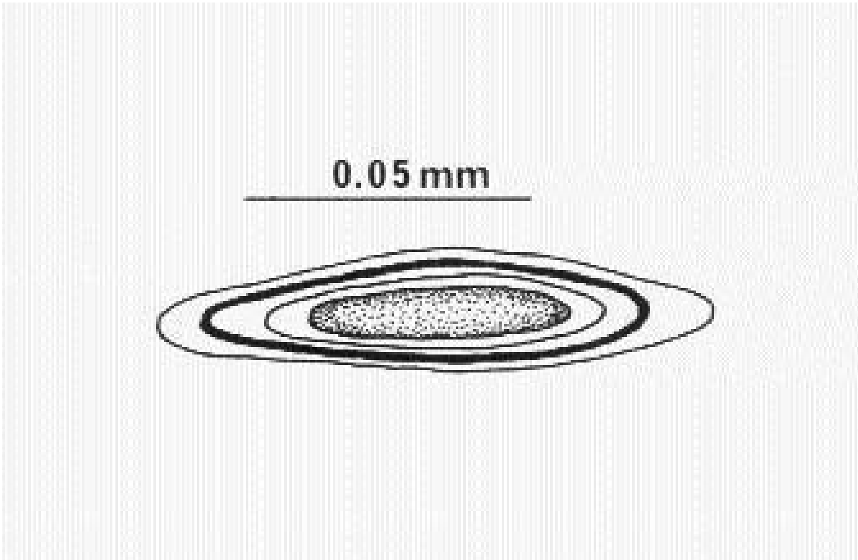

FIGURE 9.2. Drawing of *Polymorphus* egg.

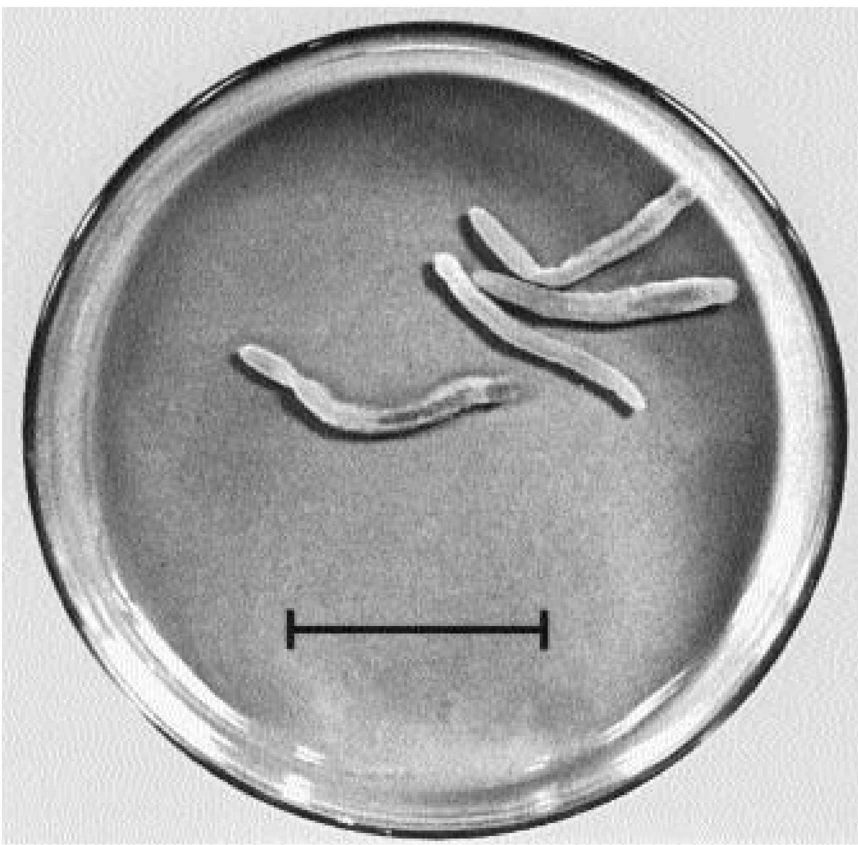

FIGURE 9.3. Specimens of *Mesaulus* in a Petri dish (bar = 2.5 cm).

lesions in the celomic cavity are diagnostic, along with eggs laid by adult forms resident in the intestine. The eggs are elliptic, large (0.1×0.07 mm), and light brown (Figure 9.4).

Once this parasite is identified, all the birds should be treated. An oral dose of 10 mg/kg and 22 mg/kg of albendazole and fenbendazole, respectively, for three consecutive days, is effective for control of the intestinal form. Although Tryclabendazole is probably the most effective drug against extraintestinal forms of trematodes, there are no suggestions of therapeutic doses for Ciconiiformes.

CONTRACECOSIS: INFECTION CAUSED BY *CONTRACAECUM* SPP. *Contracaecum* is a nematode found in the intestinal mucosa of various species

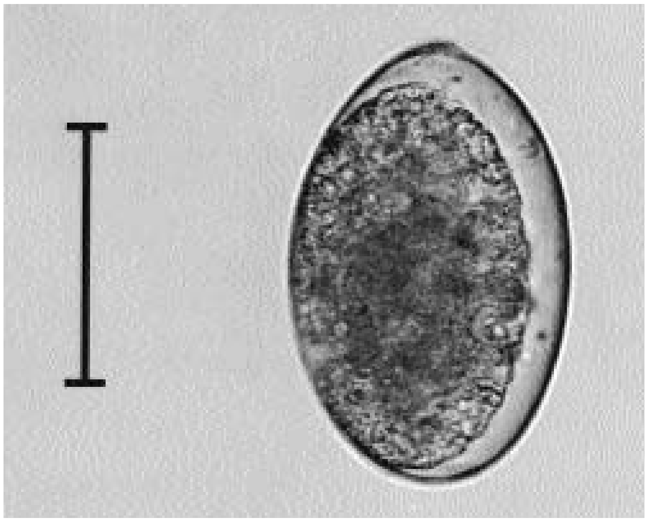

FIGURE 9.4. Egg of *Mesaulus* in feces of spoonbill (bar = 0.05 mm).

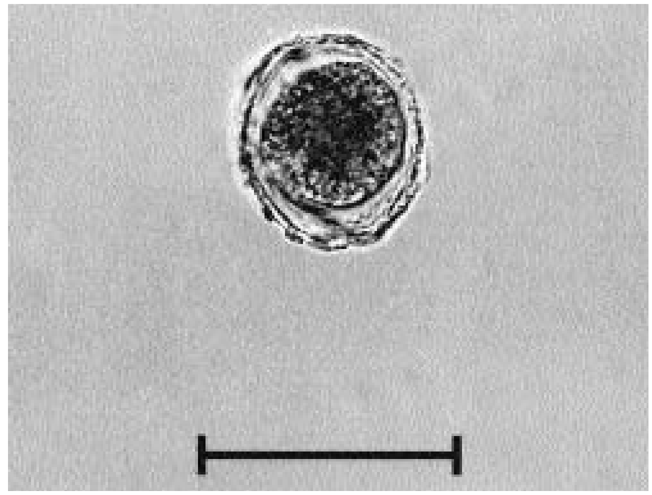

FIGURE 9.5. Egg of *Contracaecum* in feces of white heron (bar = 0.3 mm).

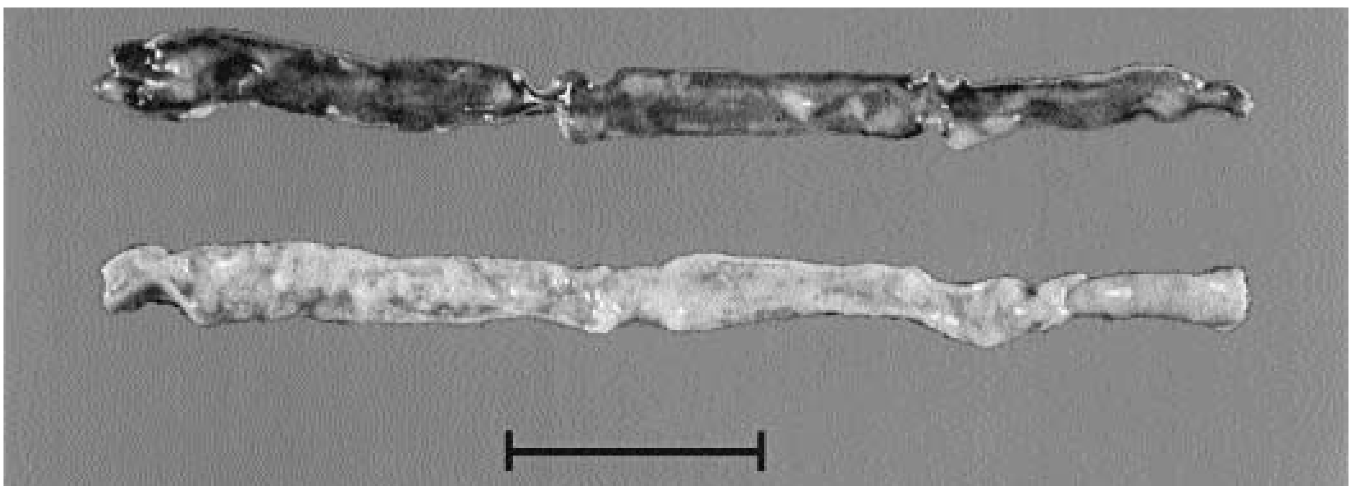

FIGURE 9.6. Macroscopic lesions in the intestinal mucosa of a night heron caused by *Phagicola longus*. The light segment is from an uninfected night heron(bar = 4 cm).

of Ciconiiformes. The adult forms are found attached to the mucosa, causing ulcerative lesions, gastritis and edema. Fibrous lesions similar to those observed in *Eustrongylides* infections may be observed, but this parasite doesn't migrate to the serosa of the stomach or to the celomic cavity. It is believed that this nematode may infect people who ingest raw fish, causing digestive disturbances.

Diagnosis is made by fecal flotation. The eggs are elliptic (0.2 × 0.18 mm) and have a thin shell (Figure 9.5).

Treatment involves administration of albendazole (10 mg/kg), fenbendazole (22 mg/kg), or mebendazole (30 mg/kg), once a day, for 3 days.

PHAGICOLOSIS: INFECTION CAUSED BY *PHAGICOLA LONGUS* This is a small trematode (1–2 mm), found in the intestinal mucous layer among the intestinal villi, and it may or may not cause serious erosion, depending on the host species and on the number of parasites. This erosive process is always accompanied by bacterial and virus infections, resulting in enteritis.

Phagicola longus is a parasite transmitted by the ingestion of fish of the family Mugilidae, known as "tainhas." It affects birds and mammals of different species, including humans. Signs are not specific, but diarrhea, dehydration, and prostration may occur. Careful search for eggs in the feces is necessary, because they are small, and the parasites do not produce large numbers of ova. The ova are light brown, elliptic (0.07 × 0.04 mm) and have a small operculum in one of the apexes (Figures 9.6 and 9.7).

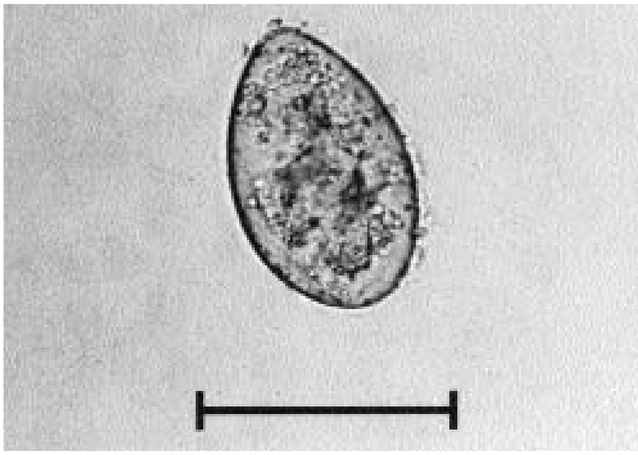

FIGURE 9.7. Egg of *P. longus* in feces of a night heron (bar = 0.42 mm).

At necropsy affected birds generally have a hemorrhagic enteritis with a severe infestation by this trematode. However, due to the size of this parasite, it is only visualized when the intestinal mucous layer is scraped, diluted in physiologic solution, and kept in Petri dish to be examined through a stereoscopic microscope.

Pelicans affected by *Phagicola longus* have been treated with success using 10 mg/kg and 22 mg/kg of albendazole and fenbendazole, respectively, when used orally, once a day, for 3 consecutive days.

Protozoa

Protozoa are frequently found in Ciconiiformes and epidemiological studies should be conducted because they may be transmitted from the birds to other species of animals. *Sarcocystis* is one of the genera that affect Ciconiiformes. It is a protozoa that causes cystic formations in the musculature of herbivore mammals, but it has also been described in other species of vertebrates. A study in Ciconiiformes in Florida, USA, revealed that 24% of the birds were affected by parasites of these genera. Diagnosis was based on necropsies of young birds found dead during the mating period.[14] Generally, the clinical manifestations for *Sarcocystis* are seen in carnivorous animals that feed on birds infected with *Sarcocystis*. These animals present with intestinal problems.

Blood parasites are of little importance as pathological agents in these birds, however, infections caused by *Leucocytozoon* and *Haemoproteus* are common in birds and reptiles. Studies of Ciconiiformes in Europe revealed that 23% of free-ranging birds were positive for parasites, whereas captive birds were free of parasitism.

No investigation of protozoan parasites of Ciconiiformes has been carried out in South America.

Coproparasitological Exam

The examination of feces for parasitic ova and larvae is the same as for other birds.

External Parasites

Because they live in aquatic sites, in areas that are not crowded, the transmission of external parasites is not common in these hosts. However, some species of lice and louse flies can be found in these birds during the reproductive period, especially in nests, where young birds are more susceptible to this kind of parasitism. On other continents, we can note the occurrence of acarids like *Argas robertsi* in nests of birds of this order, causing a decrease in the level of hematocrit, policromasy, and even death of young birds. Up to now, this kind of parasitism has not been observed on the American continent.

LICE (MALLOPHAGA) In the swamp land area of Mato Grosso, Brazil, the occurrence of lice (Mallophaga) in tuiuius, cabeças-secas, and maguaris is common. In young animals these parasites are observed in the ventral area and under the wings where they cause irritation. In adult animals, no evident clinical signs have been observed. The parasites do not interfere in the alimentary conversion. These external parasites are members of the families Menoponidae and Philopteridae.

Affected birds have been treated with talcum powder containing parasiticide, which is sprinkled onto nests and onto the birds. Just one application was enough to treat birds living in captivity. This treatment should not be carried out on rainy days.

LOUSE FLIES The occurrence of louse flies is also related to the reproductive period. These insects can be winged or apterous, don't generally fly, and stay on the host's skin for a long time. Transmission generally happens through direct contact. They feed on blood, but the negative consequences these parasites might cause to birds are not known. The transmission of blood parasites, like those belonging to the genus *Haemoproteus,* is what makes this insect important on other continents.

The most important species that affects the Ciconiiformes is *Lynchia albipennis*.

Affected birds are also treated with talcum powder containing parasiticide, which is sprinkled onto nests and onto the birds. See Figures 9.8 through 9.10.

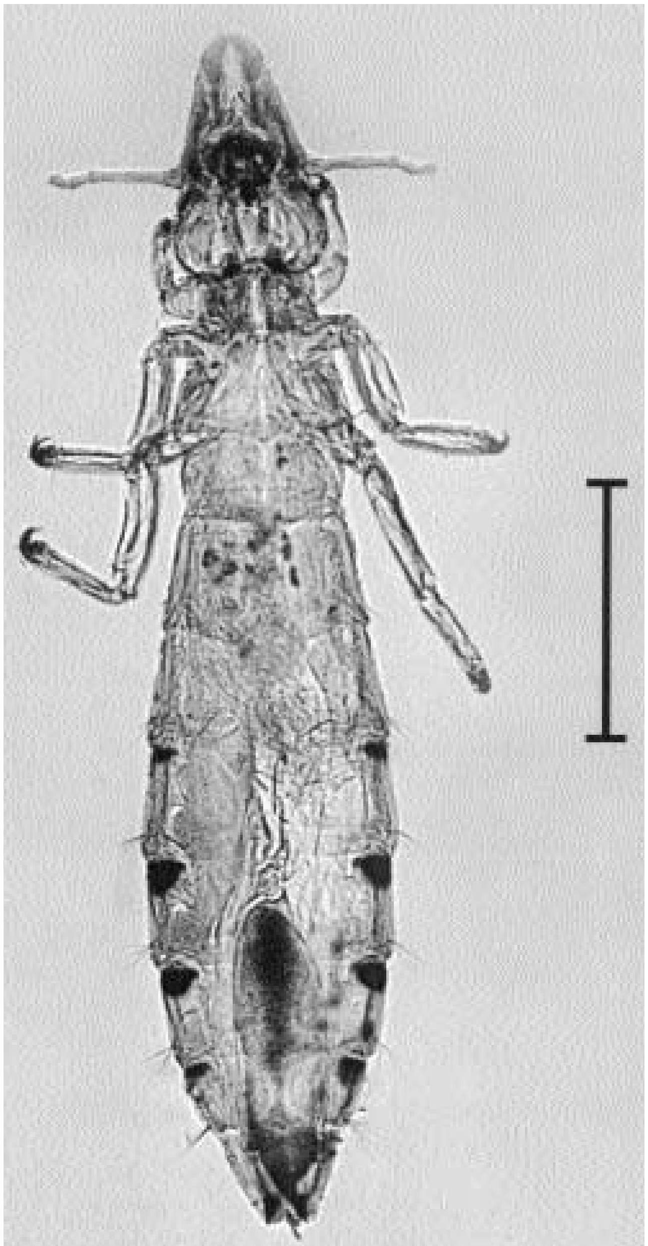

FIGURE 9.8. Phylopteridae from Tuiuiu (bar = 1.47 mm).

FIGURE 9.9. Menoponidae from white-necked heron (bar = 1.47 mm).

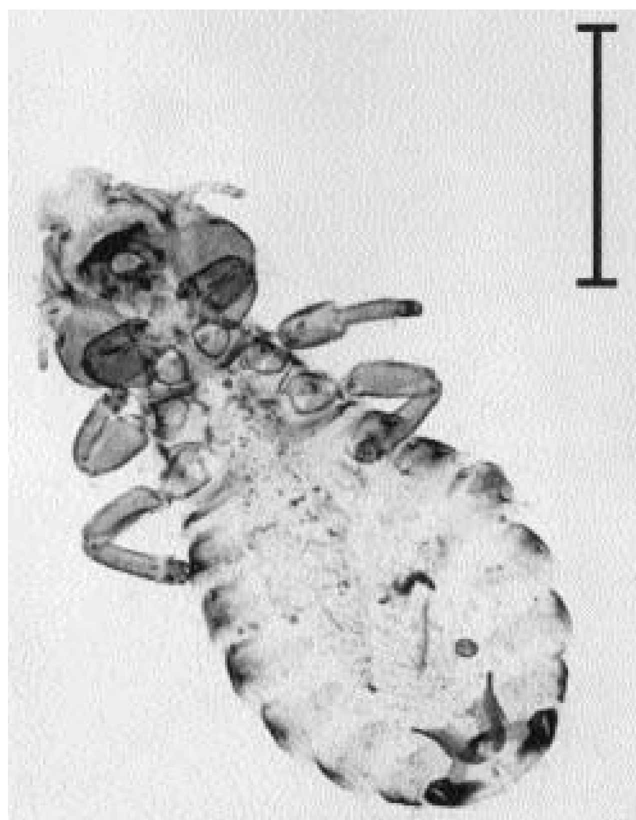

FIGURE 9.10. Menoponidae from wood stork (bar = 1.47 mm).

REFERENCES

1. Amin, O.M.; and Heckmann, R.A. 1991. Description and Host Relationships of *Polymorphus spindlatus* n. sp. (Acanthocephala, Polymorphidae) from the heron *Nycticorax nycticorax* in Peru. Journal of Parasitology 77(2):201–205.
2. Arandas Rego, A.; and Eiras, J.C. 1988. Ecologia da Parasitose de Aves e Peixes do Rio Cuiabá (Mato Grosso, Brasil) por *Eustrongylides ignotus* (Nematoda, Dioctophymatoidea) in piscivorous birds. Canadian Journal Zoology 66:2212–2222.
3. Khail, L.F.; Jones, A.; and Bray, R.A. Eds. 1994. CID Key to Cestode Parasites of Vertebrates. St. Albans, U.K., International Institute of Parasitology, Commonwealth Agricultural Bureaux.
4. Measures, L.N. 1987. The development and pathogenesis of *Eustrongylides tubifex* (Nematoda, Dioctophymatoidea) in piscivorous birds. Canadian Journal Zoology 66:2223–2232.
5. Di Modugno, G.; Sacchi, L.; Depalma, P.S.C.; Camarda, A.; Biscaldi, G.; Di Strosselli, S.; and Modugno, G. 1992. Blood parasites in wild waterfowl living free or in captivity. In Proceedings 9th International Symposium on Waterfowl. pp. 182–184.
6. Price, A.; and Graham, O.H. 1997. Chewing and Sucking Lice as Parasites of Mammals and Birds.
7. Prigioni, C.; and Sacchi, L. 1984. Blood parasites recorded in Italian birds. Avocetta 8:11–17.
8. Schaffer, G.V.; Armas Rego, A.; and Pavanelli, G.C. 1990. Estudo das lesões causadas por *Eustrongylides ignotus* (Jagerskiold, 1909) (Nematoda, Dioctophymatoidea) em algumas aves piscivoras do Brasil. Revista Unimar 12(2):201–208.
9. Sepulveda, M.S.; Spalding, M.G.; Kinsella, J.M.; Bjork, R.D.; and McLaughlin, G.S. 1994. Helminths of the roseate spoonbill, *Ajaia ajaja,* in southern Florida. Journal of the Helminthological Society of Washington 61(2):179–189.
10. Spalding, M.G. 1990. Antemortem Diagnosis of Eustrongylidosis in wading birds (Ciconiiformes). University of Florida, Gainesville, Florida. [Reprinted from Colonial Waterbirds 13 (1):75–77.]
11. Spalding, M.G.; Atkinson, C.T.; and Carleton, R.E. 1994. *Sarcocysti* sp. in wading birds (Ciconiiformes) from Florida. Journal of Wildlife Diseases 30(1):29–35.
12. Travassos, L. 1965. Contribuição para o inventário crítico da zoológia no Brasil. Fauna Helmintológica: Considerações Preliminares. Cestodeos. Rio de Janeiro, Brazil, Publicações avulsas do Museu nacional.
13. Travassos, L.; Teixeira de Freitas, J.F.; and Kohn, A. 1969. Trematodeos do Brasil. Vol. 1, Memórias do Instituto Oswaldo Cruz.
14. Vicente, J.J.; Rodrigues, H. de O.; Gomes, D.C.; and Pinto, R.M. 1996. Nematodes do Brasil, 4. Nematoides de aves. Revista Brasileira de Zoologia 12:1–273.
15. Yamagutti, S. 1971. Synopsis of Digenetic Trematodes of Vertebrates, Vols. 1–2. Tokyo, Keigaku Publishing.

10
Order Phoenicopteriformes (Flamingos)

Murray E. Fowler

BIOLOGY

Flamingos are beautiful, popular birds that are frequently exhibited in a prominent enclosure near the entrance of zoos throughout the world. Historically, flamingos were harvested for food by native peoples, and flamingo tongue was considered a delicacy by Roman royalty. Flamingos are long-lived birds, some reaching 50 years of age.

Taxonomy

Flamingos have been classified historically in various orders (Ciconiiformes, Anseriformes), but are currently classified in their own order, Phoenicopteriformes. There are six species worldwide, four of which are found in South America, Table 10.1.[6,8]

Distribution

See Table 10.1.

Habitat

Flamingos occupy remote, shallow saline and soda lakes and lagoons.

Anatomy and Physiology

Long, thin legs and the capacity to stand on one leg with the head tucked beneath a wing for a considerable time characterize flamingos. The neck is proportionately the longest of any bird, containing 17 cervical vertebrae. The feet are fully webbed with four digits in American and Chilean flamingos. The hallux is absent in James' and Andean flamingos. The bill is uniquely adapted for filter feeding. See feeding behavior under field data.

Gender Determination

Males and females are alike in color. The male is usually larger than the female, but experience is required to discern the difference. The most popular method of gender determination is by DNA testing (polymerase chain reaction, or PCR, technology), which requires only a drop of whole blood, submitted in a kit provided by the laboratory conducting the test (PE AgGen, 1756 Picasso Ave, Davis, California, USA). Fecal steroid hormone analysis compares the balance of estrogen and testosterone using radioimmunoassay (AUD laboratory, Aztec, New Mexico, USA). The bird must be sexually mature.

Another method of sexing is to karyotype the bird using cells from feather pulp. Bird karyotypes are different than those of mammals. The female is ZW heterogametic, and the male is ZZ homogametic. To

TABLE 10.1. South American flamingos—biological data (*Class: Aves; order: Phoenicopteriformes; family: Phoenicopteridae*)

Scientific Name	Common Name (English)	Common Name (Español)	Common Name (Portuguese)	Distribution	Coloration	Diet	Weight (kg)
Phoenicopterus ruber ruber	American flamingo (Caribbean, rosy or Cuban flamingo)	Flamenco Americano	Flamengo Americano	Atlantic coast of tropical and subtropical America, Bahamas, Cuba, Hispaniola, Yucatan, Guiana, Galapagos Islands	Generally pinkish to crimson with most intense color in wings. Legs light pink with red joints. Bill white, pinkish base, black tip	Small mollusks, crustaceans. Secondarily, seeds and blue-green algae and diatoms	M: 2.8 F: 2.2
Phoenicopterus chilensis	Chilean flamingo	Flamenco austral	Flamengo Chileano	Temperate South America, Peru, Bolivia, Paraguay, Uruguay, and south to Tierra del Fuego	General body light pinkish. Vermillion wings. Bill black tip and reddish base. Legs greenish grey with reddish joints and toes	Annelid worms, small mollusks, crustacea, protozoa	M: 3.9 F: 1.9
Phoenicoparrus jamesi	James' flamingo or Puno flamingo	Parina chica	James' Flamengo	High Altiplano salt-lakes of the Andes in the extreme south of Peru, western Bolivia and northern Chile and Argentina.	Generally white. Bill tip black, base yellow. Feet and legs red	Diatoms—Unicellular aquatic algae with hard shells of silica	
Phoenicoparrus andinus	Andean flamingo	Parina grande	Andean Flamengo	Andes of Southern Peru, Bolivia, Northern Chile and northwestern Argentina	Generally white with wing coverts, neck and breast a brilliant magenta. Bill tip black (more than half), base whitish yellow. Legs and feet whitish yellow	Diatoms	M: 2.3–2.4 F: 2.0–2.1

obtain feather pulp cells, the flamingo is physically restrained, and a mature tail feather is pulled from its follicle. If a pin feather is present, this step may be redundant. In 2-3 weeks the bird is again restrained, and the newly emerging feather (pin feather) is also extracted. Three centimeters of the feather shaft is cut and placed in a culture medium supplied by the laboratory (Avian Genetic Sexing Laboratory, Barlette, Tennessee, USA).

Surgical sexing through laparoscopy is also performed, but is more invasive than the previous methods.

Field Data

Social Structure

Flamingos are highly social and congregate in huge flocks. The author witnessed an assemblage of over a million lesser flamingos on Lake Nakuru in Kenya. The productivity of food for these flamingos approached that of an intensively managed, irrigated pasture in California. The lake appeared to be a pink field.

Diets and Feeding Behavior

Flamingos are separated by bill shapes; shallow-keeled bills (American and Chilean), and deep-keeled bills (Andean and James'). Shallow-keeled species are able to use seeds and invertebrates (brine flies, *Ephydra* spp.; brine shrimp, *Artemia* spp.; and small mollusks, *Corithium* spp.), which are found in the mud of shallow alkaline/saline lakes. Deep-keeled species filter water near the surface, ingesting blue-green algae and diatoms.[1,3,6,8,9]

The bill of a flamingo is uniquely adapted into a specialized food-gathering mechanism, which involves filtration of organisms from mud and water. A flamingo chick is hatched with a straight bill. As the bird matures, a bend develops that allows the birds to feed with the head inverted so that the mandible is uppermost.[3]

During feeding, the beak is slightly open. The thick, fleshy tongue is withdrawn into the pharynx, producing a negative pressure in the mouth and allowing mud or water to enter. The beak is then closed and the tongue brought forward to express water and mud through the lamellae of the upper and lower beak. Particulate matter (algae, diatoms, crustacea, depending on the species of flamingo) is retained in the mouth. As the tongue is withdrawn to begin the next cycle, the food particles are picked up by tiny projections on the tongue and pushed backward into the oral cavity and swallowed. The feeding cycle is rapid (three to four per second).[3]

Water is expressed as the tongue presses against the lamellae of the beak. Thus, the bird does not consume the saline or alkaline waters characteristic of the habitat of flamingos. When a flamingo drinks, it goes to a freshwater source; however, flamingos have a well-developed salt gland that enables them to drink brackish water.

Predators

In South America, foxes may prey upon nesting flamingo colonies, as do eagles, vultures, and gulls. Human egg collectors may impact a nesting colony. In Africa, spotted hyenas and marabou storks prey on flamingos.

Conservation

The world population estimate for South American flamingos is as follows: Caribbean, 80,000–90,000; Chilean, 200,000; James', 30,000–50,000; and Andean, 50,000.[8] All the South American flamingos are listed in Convention on International Trade in Endangered Species (CITES), Appendix II. There is no Species Survival Plan operating for flamingos, but there is a Stud Book, that has been maintained by Peter Shannon, St. Catherine's Wildlife Conservation Center, Rt. 1, Box 207-Z, Midway, Georgia, 31320-9801, USA. Flamingos are included in the Ciconiiformes Taxon Advisory Group (TAG) of the American Zoo and Aquarium Association.(The group leader is Anna Marie Lyles, Central Park Wildlife Center. Phone: 1 9 212 439 6503, E-mail: Alyles.WCS@mcimail.com.)

Reproduction in the Free-Ranging State

Flamingos are basically monogamous, with pairings lasting for years. Courtship is initiated by complex flock displays involving birds rushing back and forth, heads swaying and bobbing. During the flock social display, pairing takes place, and courtship between the pairs leads to copulation and egg laying (Table 10.2).

Nests are usually constructed at a site separate from feeding areas and frequently in remote places with limited access by land predators. The nests are inverted, truncated mud cones 18-36 cm (6-14 in.) tall, with a shallow depression on the top. Both sexes share incubation duties. The elevated nest is a protection against flooding and high soil temperatures in some nesting colonies.

A single egg is incubated for 30-32 days. The chick is dull gray at hatching, and the beak is straight. The chick is fed by "crop milk" regurgitated from both parents, which may have a reddish color and be confused with hemorrhage. Crop-milk is produced after food stored in the crop has moved on to the proventriculus and ventriculus. Table 10.3. compares flamingo and pigeon crop milk.[8]

TABLE 10.2. Reproductive data of South American flamingos

Scientific Name	Breeding Season	Breeding Location	Egg Size
Phoenicopterus ruber ruber	No fixed breeding season, mostly depends on available shallow water and food. May not breed every year	Saline lagoons and lakes in the Caribbean Islands south to Venezuela and Brazil.	(82.6–99.6) by (50.8–59.1)
Phoenicopterus chilensis	No fixed breeding season, mostly depends on available shallow water and food. May not breed every year	Saline and soda lakes associated with deserts from Peru south to Tierra del Fuego	(82.7–94.8) by (49.8–59.7)
Phoenicoparrus jamesi	No fixed breeding season, mostly depends on available shallow water and food. May not breed every year	Widely scattered saline and soda lakes in Andes of Bolivia, southern Peru and northern Chile	(78.1–87.8) by (48.4–50.3)
Phoenicoparrus andinus	No fixed breeding season, mostly depends on available shallow water and food. May not breed every year	Widely scattered saline and soda lakes in Andes of Bolivia, southern Peru and northern Chile	(80.9–90.9) by (52.8–57.2)

TABLE 10.3. Composition of crop milk

Nutrient	Flamingo Crop Milk	Pigeon Crop Milk
Protein (%)	8–9	13.3–18.6
Fat (%)	15	6.9–12.7
Carbohydrate (%)	0	0
Erythrocytes (%)	1	1

Crop milk secretion is controlled by prolactin in both the male and female. Initially, the crop milk contains large amounts of canthaxanthin (giving a reddish color to the secretion), which is stored in the liver until both the down and juvenile moltings have taken place. Then the feathers take on adult coloration.

Whereas adults rest by standing on one leg, the chicks squat on the tarsometatarsus. Once a chick leaves the nest it enters a creche of dozens, hundreds, and even thousands of juveniles. Parents are able to identify their own offspring by vocalization.

MANAGEMENT IN CAPTIVITY

Housing

The most satisfactory enclosure has a substrate of natural dirt or grass and a shallow pool. It is desirable for the pool to have an earthen surface, but this is usually not possible. Concrete surfaces should be relatively smooth to avoid foot abrasions and subsequent bumblefoot. Indoor housing must be provided in cold-winter climates.

Space Requirements

Flamingos are highly social flock birds. If breeding is to take place, 15 or more birds must be kept together. It is most desirable to have sufficient space to allow the birds to rush back and forth as part of their courtship behavior.

Water Quality

In the free-ranging state, flamingos inhabit some of the most odoriferous vile waters of any animal. Because captive flamingos are fed separately from the wading pool, managers must be able to maintain a clean pool, which necessitates periodic cleaning.

Environment

Flamingos are able to tolerate cool to hot environments, but must be protected from freezing temperatures.

Feeding

Many flamingo managers compound their own flamingo diets, which may contain one or more of the following food items: bread soaked in water, poultry pellets, alfalfa meal, shrimp meal, ground carrots (*Dacus carrota*), red beets (*Beta vulgaris*), brewer's yeast, soybean meal, or fish meal. It is essential that pigment be included in the diet as carotinoids or canthaxanthin to maintain feather color.

Several feed companies in North America market flamingo diets.

- Formula 1 (Flamingo-Fare, Reliable Protein Products, Palm Desert, California, USA)—The guaranteed analysis of this feed is crude protein (>25%), crude fat (>10%), crude fiber (<5%), and moisture (<45%). The ingredients (in descending order, according to the amount in the diet), include meat by-products, poultry by-product meal, fish meal, shrimp meal, ground oats, ground wheat, wheat germ, wheat bran, dried kelp, ground barley, wheat shorts, brewer's dried yeast, dried whey, bakery by-products, crushed roasted peanuts, alfalfa meal, ground oyster shell, water, dehydrated bluegrass, salt, spirulena, concentrated carotene, canthaxanthin beadlets, and other mineral and vitamin supplements to balance the diet. Feeding directions are: May be fed dry or mixed with warm water (0.5 kg–2.5 L of water), placed in a shallow feeding pan.

- Formula 2 (Mazuri Flamingo Complete, Purina Mills, Inc., P.O. Box 66812, St. Louis, Missouri, USA)—The guaranteed analysis is crude protein (>19%), crude fat (>5%), crude fiber (<4%), calcium (1.55%), phosphorus (0.93%), canthaxanthin (40 ug/kg of feed), metabolizable energy (3.07 Kcals/g). The ingredients include ground yellow corn, ground wheat, wheat middlings, fish meal, soybean meal, meat and bone meal, brewer's dried yeast, soybean oil, blood meal, cyanocobalmin, dicalcium phosphate, canthaxanthin, and other minerals and vitamins to balance the diet. Feeding directions are: The product is an extruded pellet, which floats and may be scattered directly on water in a long shallow pan. Consumption is 150–200 g per day.
- Formula 3 (Mazuri Flamingo Breeder, Purina Mills, Inc., P.O. Box 66812, St. Louis, Missouri, USA)—The guaranteed analysis is crude protein (>34%), crude fat (>5%), crude fiber (<7%), calcium (2.80%), phosphorus (1.22%), canthaxanthin 40 µg/kg of feed, metabolizable energy 2.49 Kcals/g. Ingredients include alfalfa meal, ground wheat, corn gluten meal, fish meal, bone meal and other ingredients similar to formula 2. Feeding directions are the same as for formula 2.

RESTRAINT, ANESTHESIA, SURGERY

Restraint

Flamingos usually may be slowly herded into a corner or a catch-pen, permitting handlers to enter the enclosure to capture them. They should be grasped by the neck with one hand while quickly grasping the body with the other, lifting the bird off its feet, and directing the legs slightly away from the handler. The body of the bird should be held next to the hip of the handler, leaving the legs to move freely. Flamingos should not be clutched by their lower legs or netted, for the long spindly legs may be injured. If it is necessary to restrict the movement of the limbs, do so by grasping the neck with one hand and the base of the legs with the other.[4] A cloth hood may help calm the bird. Manual restraint is adequate for physical examination, blood collection, and minor medical treatments and procedures.

Anesthesia

Injectable immobilization and anesthesia may be accomplished using ketamine hydrochloride (15–25 mg/kg body weight). Diagnostic and surgical procedures are best carried out using isoflurane inhalation anesthesia. Induction and recovery are both rapid. The bird is captured as described. The isoflurane is administered using a precision vaporizer and a face mask adapted from the various plastic containers used to contain pharmaceuticals.

If endotracheal intubation is contemplated, the insertion has to be blind, inasmuch as the mouth is unable to be opened sufficiently to allow observation of the glottis. More commonly, anesthesia is continued using the face mask. Anesthesia should be monitored using the techniques common to avian medicine.

Flamingos present special problems during recovery from anesthesia or immobilization. If left to their own devices while awakening, they are likely to stagger and fall, injuring themselves. A satisfactory means of controlling the bird during recovery is to place it in a cloth sack (100% cotton, light-weight pillowcase) with the head exposed. This prevents the bird from standing until the effects of the anesthetic are completely dissipated.[4]

Surgery

The procedures used for fracture repair are the same as in other birds. Pinioning, for deflighting, must be performed on flamingos that are maintained in outdoor enclosures. Removal of the digits and metacarpal bones of a single wing may be carried out in the new chick by simply crushing the tissue with a hemostat and cutting with scissors or a scalpel. This author tries to avoid handling chicks and prefers to wait until the bird is about to fledge. Anesthesia may be sedation with ketamine hydrogen chloride (HCl) (15 mg/kg) or, preferably, general anesthesia using isoflurane.

The site of amputation is determined by finding the false wing (alula) and cutting the bone just distal to the base of this structure. The feathers should be plucked, not clipped, including two or three of the primary flight feathers. A circumferential skin incision is made distally and the skin reflected back on the metacarpus. The major vessels in this area are between the metacarpal bones that are fused at either end. A figure-eight transfixion ligature is placed loosely around the proximal ends of the bone. The bones are amputated using a sharp chisel while the wing is lying on a wooden block. Once the distal end of the metacarpals are freed, the ligature is tightened. The skin is pulled into place to cover the end of the bones and sutured closed. A light pressure bandage is applied to assist in controlling hemorrhage. The bird usually removes the bandage in a day or two.

DIAGNOSIS

Clinical Examination

Physical examination is similar to that performed in other avian species. Blood may be collected from the jugular, medial metatarsal, or ulnar veins. Hematology and serum chemistry analysis is similar to other avian species. The plasma and serum from captive flamingos is typically orange.

Feces may be examined for cytology (Wright's or Diff Quick stain), gram stain, acid fast stain, occult blood, flotation, direct smear (parasites), and culture. Serum should be collected and banked as a standard for future disease concerns.

All the special diagnostic procedures employed in other avian species may be performed in flamingos, including laparoscopy, endoscopy, and radiology.

REPRODUCTION IN CAPTIVITY

Two important factors for successful reproduction are sufficient numbers of birds to allow proper social interaction and appropriate pigmentation of the feathers. The American and Chilean flamingos are now bred in captivity, but still not in sufficient numbers to supply the needs of all zoos.

Artificial incubation has not been routinely practiced because of the difficulty of hand rearing the chicks. It has not been possible to duplicate the crop milk supplied by the parents.

There are some common health problems of neonates. Chicks may become traumatized or develop metabolic bone diseases (described later). A unique condition occurred in lesser flamingos in Africa, with the formation of mineralized rings on the limbs of the chicks in response to lowering water levels and concentration of salts in the water. Rescue teams were able to save thousands of chicks, but not all, by breaking the rings.

DISEASES[1,3,5,6,8]

Infectious Diseases

Flamingos are not known to have any unique infectious diseases. They are susceptible to a number of specific avian viral, bacterial, and fungal diseases (Table 10.4). Opportunistic bacterial infections occur just as in other birds.

Omphalitis (incomplete closure of the umbilicus) is most commonly caused by gram-negative bacteria. It may be prevented by providing a clean environment and disinfecting the umbilicus at hatching with dilute povidone iodine solution. Incomplete closure of the umbilicus may be accompanied by a small yolk sac protuberance. If the protuberance turns black due to constriction, it should be left alone and will usually fall off in a few days.

Although infection with *Mycobacterium avium* complex is not a common problem in captive flamingos, it has been responsible for significant loss in free-ranging lesser flamingos in Kenya. Tuberculosis is a disease that should be considered in any bird necropsy, particularly in emaciated birds. The lesions found in birds may not always be the typical granuloma seen in mammals. Numerous cases of histiocytic infiltration of the lamina propria layer of the intestine with acid fast organisms have been identified.

Cutaneous avian pox may occur in flamingo chicks and cause death. Facilities that have experienced fatalities because of pox have used commercially available avian pox vaccine.

While herpesviruses are widespread in avian species, herpesvirus disease is rare in flamingos. Having said that, a herpesvirus was identified as being responsible for mortalities in captive Caribbean flamingo term embryos and neonatal chicks. Clinical signs included weakness, lethargy, dyspnea, and diarrhea. Lesions included enlarged liver, focal hepatic necrosis, cloacitis, enteritis, pneumonia, splenomegaly, renal gout, airsaculitis, and eosinophilic intranuclear inclusion bodies in the liver, cloaca, and kidney.

In addition to aspergillosis, which is a significant disease in newly captured and poorly adapted flamingos, other fungal infections include *Cladosporium herbarum* and *Geotrichum candidum*.

Parasitic Diseases

Like other birds, flamingos have parasites, but rarely is clinical parasitism a problem. Lice are often isolated from newly imported birds, but cause no problem except in the mind of the handler. The genera are the same as in Anseriformes. Internal parasites include *Tetrameres* spp., cestodes, trematodes, acanthocephalids, and *Sarcocystis* spp. Hemoparasites include *Hemoproteus* spp. and *Plasmodium* spp. See the chapter on penguins for a discussion of malaria. It is recommended that fecal flotation be performed twice a year to monitor parasite levels. Flamingos are not adversely affected by any of the anthelmintics.

Noninfectious Diseases

Nutritional metabolic bone disease is rare in captivity, but it may occur if the parents are on a calcium-deficient diet or are not exposed to ultraviolet irradiation. Many zoos have large tropical houses that may confine flamingos to no sunlight. The disease is similar to rickets in other birds.[8]

TABLE 10.4. Infectious diseases—flamingos

Disease (English)	Disease (Español)	Disease (Portuguese)	Etiology	Signs	Diagnosis	Management
Tuberculosis	Tuberculosis	Tuberculose	*Mycobacterium avium/ Mycobacterium intracellulare*	Emaciation, diarrhea	Culture, histopathology	Difficult, sanitation, quarantine
Salmonellosis	Salmonelosis	Salmonelose	*Salmonella* spp.	Diarrhea, septicemia	Culture	Sanitation, antibiotics
Colibacillosis	Colibacilosis	Colibacilose	*Escherichia coli*	Septicemia	Culture	Sanitation, antibiotics
Infectious pododermatitis (Bumblefoot)			Multiple bacteria, *Staphyloccocus aureus*, Gram-negative bacteria	Heat, swelling, redness of the foot, lameness, ulcers on foot pads	Signs, radiography, culture	Provide suitable substrate
Velogenic viscerotropic Newcastle disease	Enfermedad de Newcastle	Doença da Newcastle	Paramyxovirus	Central nervous system effects, tremors, paresis, paralysis	History, signs, viral culture, serology	Quarantine, sanitation
Aspergillosis	Aspergilosis	Aspergilose	*Aspergillus fumigatus*	Dyspnea, exercise intolerance	Culture, direct smear	Minimize stress
Malaria			*Plasmodium* spp.	Weakness, anemia	Direct smear	Mosquito control

Another metabolic bone disease is also similar to rickets, but is restricted to the tibio-tarsus. The etiology is unknown and perplexing because the deformities and bone lesions mimic rickets, but occur while the chicks are being fed by the parents. It is possible that the material ingested by the parents alters as the season advances and energy and protein intake increases. The condition in chickens is associated with rapid chick growth, as occurs in some broiler productions. It has also been reported in rheas. Signs include lameness, stunted growth, poor feather condition, leg bowing, and swelling of the proximal tibio-tarsus. As seen using radiography, there is cortical thinning and widening of the physis. Ossification of the cartilage is retarded.[8]

Tibiotarsal rotation occurs in flamingos but is not as common as in other long-legged birds, such as cranes. The condition and its management is not described fully in the literature, but cases should be managed as in cranes and storks. A decreased rate of growth by diet manipulation is necessary, along with increased exercise. Surgery may be necessary in advanced cases.

Traumas, particularly fractures of leg bones, are common clinical entities. Birds may become frightened by dogs or people and rush or fly into obstacles or fences. Injudicious physical restraint may cause fractures. Restraint of young chicks should be avoided to prevent physeal fracture of the tibiotarsal-tarsometatarsal bones.

Exertional myopathy may result from prolonged restriction of limb movement, such as confined space during transportation or recovery from anesthesia. The etiology is muscular hyperacidity with subsequent necrosis of muscle fibers. The signs are paresis or paralysis. Elevated muscle enzyme (CK, SGOT) levels are definitive. Treatment is usually fruitless.[3]

Poisoning, particularly lead poisoning, has been reported from Spain in greater flamingos and Mexico in American flamingos when they forage in mud in an area where lead shotgun shot is used for hunting waterfowl. The clinical signs include emaciation and mortality. At necropsy lead pellets are found. There is evidence of enteritis, and the ventriculus lining is stained an intense green. Therapy regimens have not been reported, but would be similar to those used in psittacine birds and waterfowl.[7]

Zinc toxicosis has been suspected in captive collections of flamingos. The source of the zinc was not found.

Frostbite can occur in flamingos. Flamingos must be housed indoors during the winter in northern North American and southern South American temperate climates, otherwise they may suffer from frostbite of the foot webs and digits. Signs include lameness, darkening of the interdigital web, and, ultimately, necrosis of the affected tissue. Therapy is ineffective once necrosis has occurred. General nursing care may allow return to partial function.[3]

Amyloidosis occurs in captive flamingos, particularly older birds. Both sexes are equally affected. Stress and concurrent chronic disease play major roles in the development of the disease. Signs vary with the organ system affected. The lesions are similar to other birds with amyloidosis.

Miscellaneous diseases include a variety of diseases that have been reported including heat stress, neoplasia, atherosclerosis, visceral gout, myocardial infarct, stress, and intestinal obstruction.[5]

PREVENTIVE MEDICINE

Newly acquired flamingos should be placed in a quarantine facility for at least 30 days, during which time standard testing should be done. Vaccinations are not necessary for flamingos and routine antiparasite medication is rarely used. Providing a proper substrate to minimize foot problems and sufficient space to minimize social stress will do much to aid the health and well-being of flamingos. Periodic physical examination including hematology, serum chemistry, and fecal analysis, evaluation of body condition, and evaluation of the plantar surface of the feet should be performed. Birds should be permanently identified using leg bands or microchips.

REFERENCES

1. Allen, R. 1956. The Flamingos: Their Life History and Survival. New York, National Audubon Society.
2. Bildstein, K.L.; Golden, C.B.; and McGraith, B.J. 1993. Feeding behavior, aggression, and the conservation biology of flamingos: Integrating studies of captive and free-ranging birds. American Zoologist 33:117–125.
3. Fowler, M.E. 1978. Phoenicopteriformes, In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 160–163.
4. Fowler, M.E. 1995. Restraint and Handling of Wild and Domestic Animals, 2nd Ed. Ames, Iowa, Iowa State University Press, pp. 322–324.
5. Humphreys, P.N. 1975. Pathological conditions. In J. Kear and N. Duplaix-Hall, eds.,. Flamingos. Berkhamsted, England, T. and A.D. Poyser.
6. Kear, J.; and Duplaix-Hall, N. Eds. 1975. Flamingos. Berkhamsted, England, T. and A.D. Poyser.
7. Mateo, R.; Dolz, J.C.; Aguilar Serrano, J.M.; Belliure, J.; and Guitart, R. 1997. An epizootic of lead poisoning in greater flamingos *Phoenicopterus ruber roseus* in Spain. Journal of Wildlife Diseases 33(1):131–134.
8. Ogilvie, M.; and Ogilvie C. 1986. Flamingos. Gloucester, England, Alan Sutton Publishing.
9. Zweers, G.; de Jong, F.; and Berkhoudt, H. 1995. Filter feeding in flamingos (*Phoenocopterus ruber ruber*). The Condor 97: 297–324.

11
Order Anseriformes (Ducks, Geese, Swans)

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BIOLOGY

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The order Anseriformes is a large one composed of three families—Anhimidae (screamers), Anseranatidae (Magpie goose, only in Australia and New Guinea), and Anatidae (ducks, swans, and geese).[4] Although everybody recognizes a duck or its relatives, few people recognize that the screamers are members of this order (see below). The short legs, webbed toes, broad and flat bill with many lamellae, dense plumage, and the rounded body proper to aquatic habits, seem sufficient to characterize ducks, but there are exceptions to this pattern when considering the whole order. The males have a phallus protrundens, an intromittent copulatory structure, nonhomologous to the penis of the mammals due to the lymphatic (not sanguineous) erection mechanism, the way in which sperm is conducted, and its exclusive use in reproduction; this structure is also found in Tinamiformes and Ratites. The Anseriformes possess a bony projection in the caudal part of the mandible, the processus retroarticularis, a structure shared with other bird orders (e.g., Galliformes, Ciconiiformes, and Phoenicopteriformes). The first fossil attributed to this order is *Presbyornis,* from the Paleocene in Utah and Mongolia. This interesting bird has a mosaic of characters suggesting a link between the shorebirds and ducks; in fact *Presbyornis* has a cranium and beak similar to the ducks (also possessing the processus retroarticularis), but the postcranial skeleton is like shorebirds, with their long legs.[2] Always living near the water, this order is, without doubt, one of the most important and well-studied among the birds. One of the best-conducted phylogenetic studies in the class Aves was made by Livezey, and those findings clarified many of the relationships among the Anseriformes' genera.

Family Anhimidae (Screamers)

There are three species in two genera. They are large birds (80–95 cm, 3.5–5 kg) found only in South America. They are the base in the phylogeny of the Anseriformes.[4] This family can be characterized by the absence of uncinate process, a well-developed system of air sacs beneath the skin giving a spongy aspect, exceptionally pneumatic bones, lack of feather tracts (similar to the penguins), small head and beak (resembling the Galliformes), sparse and few developed lamellae in the inner surface of the bill, strong and long legs and toes, webbed only in the basal portion (appropriate to walk through the marsh vegetation), rounded wings, two bony spurs at the carpus, the proximal longer and sharper than the distal. *Anhima cornuta* has a long corneous (like a horn) projection from the forehead, sometimes larger than the bill, and *Chauna* has a small crest. The colors are discrete, black or gray, with white marks on the wings, neck, and breast. *Anhima* has a white belly. Sexes are alike, but the male is usually larger than the female. The molt is gradual, different from the Anatidae, which lose all flight feathers at the same time.

The screamers can swim and are good fliers, but they are chiefly ground dwellers. They have loud voices and are one of the most characteristic voices of marshlands of South America. Herbivorous, they also can prey on some invertebrates when they are grazing. Inhabitants of open areas, the screamers live in groups or in pairs (in the breeding season) and prefer marshes and lakes, always with much vegetation around.

The pairs are territorial when breeding, and the nests are constructed by the couple with sticks and the vegetation that they find near the nesting place, always near the water; the female lays from two to seven eggs.[3] The chicks are covered with dense yellow down feathers and are precocial.

The species of screamers are common in many parts of their range, but they are particularly sensitive to alterations of their particular habitat; the drainage of the humid areas and the construction of dams have already eliminated the horned screamer from some parts of its range in southeastern Brazil. Due to the restricted range, the northern screamer, *Chauna chavaria,* is now considered to be near threatened, with small populations in the northwestern part of South America.[3]

Family Anatidae (Ducks, Swans, and Geese)

There are 38 species in 16 genera in this family. The rounded body, short legs and tail, webbed toes, and the broad and flat bill are, in general, sufficient to characterize the Anatidae. Inhabitants of marshes, shores, lakes, rivers, and streams, the South American ducks show a large variety of forms and colors.

The size and number of lamellae located in the inner surface of the bill (and its shape) reflect the adaptations imposed by the diet; the adaptations are drastic in the Merganser (*Mergus*), where the lamellae are modified in a set of small saws (serrated bill), which specialize in catching fish. Most species are essentially herbivorous, feeding on roots and tubers to leaves, flowers, and seeds. Some species, such as Mergansers or the steamer ducks (*Tachyeres*) prefer fish or aquatic invertebrates. We note three types of feeding behavior: grazing, dabbling, and diving. Their habit can be useful in identifying the species in the field or in captive breeding.

There is great variation in size and weight, with the largest species in South America, the black-necked swan, *Cygnus melanocorypha,* weighing 7.0 kg and being 120 cm long to the small ringed teal, *Callonetta leucophrys,* weighing 360 g and being 38 cm long.[3] Then there are the large *Tachyeres, Coscoroba, Chloephaga, Cairina, Sarkidiornis* and the small *Amazonetta* and *Anas* species. They have dense plumage to maintain their floatation and isolation from water.

The Anatidae have a unique type of molt, which occurs after the breeding season: All flight feathers are lost and the birds become flightless. Wings are strong, but short and pointed.[3] Many species have a large patch in the secondaries with a different color, forming a speculum. The colors are usually brilliant, such as blue or green, but can be pure white. The color of the speculum is very important in identifying the species of *Anas.* Some ducks show sexual differences in color of plumage (extreme in some *Chloephaga*), with the males being more brilliant and the females duller and less ornate; the male of *Sarkidiornis melanotos* has a fleshy comb in the bill, *C. melanocorypha* and *Cairina moschata* develop caruncles in the base of the bill and lores, more evident in males, during the breeding season. The *Anas* species have cryptic colors, but other South American ducks have colors with variations from black, white, gray, or brown (and combinations of these colors), with metallic sheen principally in the back.

The species of ducks that obtain their food by dabbling prefer plain waters, where the concentration of nutrients is larger than in open waters and it is easier to obtain food. Others, like the torrent duck (*Merganetta armata*)[3] and the Brazilian Merganser (*Mergus octosetaceus*)[6] prefer the clear, very oxygenated, and fast-flowing streams and rivers of the mountains, where they find invertebrates and fish, respectively, as their specialized food. *Dendrocygna, Cairina, Neochen,*[5] *Sarkidiornis,* and *Anas flavirostris*[5] can use perches to rest.

The voices of South American ducks are very simple, not elaborate, and the sexes have different songs. They are gregarious, and *Dendrocygna* can be found in large flocks. Some strongly territorial species, like the Brazilian Merganser, *Mergus octosetaceus,* live in pairs or in small family groups[6]. There are many migratory species, mainly on the southern part of the continent, where most of the species of the family concentrate. The Amazon is very poor in species of Anatidae, probably due to the competition with the great number of fish species of the rivers[5]. The ducks spend the day searching for food in the water during the warmer periods, resting on the water's surface or over stones that emerge from the water, and caring for their plumage in the hottest hours. *Dendrocygna* flies in groups at night, and its groups can be heard passing over large and polluted cities such as São Paulo. The courtship is characteristic to each specie and involves displays, which consist in a series of movements with the wings, tail, head, and neck. The ducks form a new pair in each breeding season; only territorial species have a permanent mate. *Cairina moschata* and *Sarkidironis melanotos* are polygamous.[3,5] They copulate only in the water and choose a variety of places to construct the nest: among the marsh vegetation, on the dry land, burrows, or cavities in trees.[3] The nests are constructed from the vegetation around the nest and feathers that the female picks up from her breast. The male can help the female, but only the latter incubates the eggs (except in *Cygnus*).[3] Hatching synchronously, the ducklings are precocial and follow their parents (or anything that moves—a process known as imprinting), being protected by them. Both male and female of *Cygnus melanocorypha* can carry their offspring on their backs. Only one species is a nest parasite, the black-headed duck, *Heteronetta atricapilla,* laying her eggs in the nests of other species.[5]

Some species of ducks (e.g., *Netta peposaca*) in South America can be hunted in certain seasons, and the ducks occupy an important place in the human life, as domestic species (*Cairina moschata, Anas platyrhynchos,* and *Anser anser,* the last two being species from the Northern Hemisphere).

Although many species may gain with the expansion of crops like rice and the construction of dams or artificial lakes, others have specific habitats and are dependent on undisturbed lakes or rivers with abundant natural vegetation, giving protection, food, and nesting places. The situation is dramatic to at least one species in South America, the Brazilian Merganser (*Mergus octosetaceus*). Originally found in mountain rivers in Argentina, Paraguay, and Brazil,[1] this duck is rare and very sensitive to human presence and only feeds off fish that prey in the rapids of clear and well-oxygenated water. This habitat is the prime area to construct dams that produce electric energy, and when the rapids disappear, the Merganser is no longer found. This species is almost extinct in Argentina and Paraguay; in Brazil it can be found only in two or three places. Certainly the best-studied and more protected site is in the Serra da Canastra National Park, in southwest Minas Gerais State, Brazil.[6] The population that still lives there is increasing, but the area is threatened by diamond miners and uncontrolled tourism. Many species of ducks breed easily in captivity, and it could be an interesting alternative to increase the population of these Mergansers with the establishment of a captive population, and, in the future, to try to reintroduce this bird in places where it occurred in the past.

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REFERENCES

1. Collar, N.J.; Gonzaga, L.A.P.; Krabbe, N.; Madroño Nieto, A.; Naranjo, L.G.; Parker, T.A.; and Wege, D.C. 1992. Threatened Birds of the Americas: The ICBP/IUCN Red Data Book. Cambridge, International Council for Bird Preservation, p. 1,150.
2. Feduccia, A. 1996. The Origin and Evolution of Birds. New York, Yale University Press, p. 420.
3. del Hoyo, J.; Elliot, A.; and Sargatal, J. Eds. 1992. Handbook of the Birds of the World, Vol. 1. Ostrich to Ducks. Barcelona, Spain, Lynx Editions, p. 696.
4. Livezey, B.C. 1986. A phylogenetic analysis of recent anseriform genera using morphological characters. Auk 103: 737–754.
5. Sick, H. 1997. Ornitologia Brasileira. Rio de Janeiro, Nova Fronteira, p.911.
6. Silveira, L.F. 1998. The Birds of Serra da Canastra National Park and Adjacent Areas, Minas Gerais, Brazil. Cotinga 10: 55–63.

CAPTIVE MANAGEMENT AND MEDICINE

Murray E. Fowler

MANAGEMENT IN CAPTIVITY

One hundred forty-seven species of ducks, geese, and swans inhabit the world.[17] In South America alone there are 18 genera and 50 species (one-third of the world total). They occupy varied habitats and have different feed preferences (Table 11.1). Ducks, geese, and swans were among the first birds to be domesticated, as early as 4500 years ago.[17] First they were kept for food, but eventually some were kept for their ornamental beauty. Private waterfowl collections are not as plentiful as psittacine and passerine cage birds, because of the more exacting requirements for managing an aquatic habitat; however, ponds in private parks and zoos abound.

Housing

Waterfowl are often maintained in large, mixed open exhibits containing a large pond and few to many species of birds. If propagation is an important aspect of

TABLE 11.1. Selected South American Anseriformes

Scientific Name	Common Name (English)	Common Name (Spanish)
Cairina moschata	Muscovey duck	Pato criollo
Sarkidiornis melanotas	Comb duck	Pato crestudo
Dendrocygna bicolor	Fulvus tree duck	Sirirí Colorado
Dendrocygna autumnalis	Black-bellied tree duck	Sirirí ala blanca
Dendrocygna viduata	White-bellied tree duck	Sirirí pampa
Anas discors	Blue-winged teal	Pato media-luna
Anas cyanoptera	Cinnamon teal	Pato colorado
Oxyura ferruginea	Andean rudy duck	Pato-zambullidar grande
Anas platyrhynchos	Mallard duck	Malard
Netta peposaca	Rosy-billed pochard	Pato picazo
Merganetta armata	Torrent duck	Pato de Torrente
Neochen jubatus	Orinoco goose	Ganso de monte
Chloephaga hybrida	Kelp goose	Caranca
Chloephaga melanoptera	Andean goose	Guayata
Coscoroba coscoroba	Coscoroba swan	Coscoroba
Cygnus melanconypha	Black-necked swan	Cisne cuello negro
Chauna torquata	Southern screamer	Chajá

the facility, some birds are usually kept as a pair in an enclosure with a small pond or flowing stream. Birds kept in open enclosures must be pinioned to avoid escape (see chapter on flamingos for details). Feather clipping is not a satisfactory method of de-flighting, because attention to reclipping lapses and soon the birds are capable of flight. Large ponds should contain islands to provide privacy and nesting sites for the birds. Maintaining water quality is a constant challenge. A stream flowing from one enclosure to another is a risk. Nonflowing water in ponds and lakes may become contaminated with feces and attendant overgrowth of aquatic plants. A pond depth of 0.6 m is adequate for most waterfowl, but for some of the swans and diving ducks the optimal depth is 0.9–1.2 m.[13] Waterfowl frequently nibble at the banks of a pond, necessitating placement of stones, concrete blocks, or other solid material to maintain the integrity of the pond.

Space does not permit a detailed discussion of water quality, but factors that should be considered include clarity, color, temperature, circulation, oxygenation, and nutrient load. Maintaining optimal water quality requires periodic cleaning, water exchange, filtration, aeration, and sediment removal.[1]

Marine species have highly developed nasal salt glands, and it is thought that the salt inhibits mycotic growth. If they are kept in freshwater, the salt gland may atrophy, which may affect the birds' resistance to mycotic infection. If those birds are then introduced into a saline water, they may die of hypernatremia.[13] Marine species may be kept in an enclosure restricted to marine species and supplemented with salt in the diet; however, this may not be practical.

Indoor housing may not be necessary if birds have access to open water, especially if a water circulation system is used to prevent solid ice formation. Some tropical species may require indoor housing during the winter in temperate climates. Birds kept on concrete surfaces during the winter are prone to foot abrasions and the development of bumblefoot. Rubber mats or other soft surfaces should be provided.[13]

Feeding and Nutrition[11,13]

The majority of waterfowl are herbivores, but a few species are fish eaters and others consume invertebrates. Successful maintenance is dependent upon knowing the diet of the free-ranging bird and adapting available feeds. Domestic ducks *Anas platyrhynchos* have been farmed for centuries. The nutrient requirements for that species are known, and whole grains, pellets, and crumbles are available commercially. Mashes should not be used for feeding waterfowl, because the ground feed tends to form a sticky mass when mixed with saliva, which inhibits the proper ingestion of the feed.[13] The commercial diets available for game birds and domestic waterfowl should not be used as the total diet for wild waterfowl. Commercial diets are designed to produce rapid growth and a heavy body, which is not the goal for wild waterfowl.

Most of the information available for feeding wild waterfowl has been gleaned from practical experience coupled with a basic understanding of poultry nutrition. Much is known about vitamin and mineral requirements for birds. Birds have different requirements for amino acids than do mammals.[11,13] Dog food is not appropriate for waterfowl, because the protein and fat level are too high and the amino acid composition is inappropriate. Neither should high-protein game-bird chow or other high-energy/high-protein diets for poultry be used as a total diet. When wild waterfowl are fed excessive energy diets they tend to develop hepatic lipidosis, particularly if they are deficient in choline chloride.

Geese are basically grass grazers. Grass provides a lower protein level than seeds and other vegetable matter. Geese are also able to deal with diets higher in fiber than other waterfowl. Feeding high-protein/high-energy diets may cause terminal renal failure and at best higher water intake to rid the body of excess nitrogen. That same diet may predispose certain species to angel wing in hatchlings (see later discussion).

New hatchling waterfowl may be offered a turkey pelleted ration containing 20% protein. At 2–3 weeks of age scratch grains should be added to the diet. When the birds are mature they may be fed a diet of 10% turkey grower pellets and 90% mixed grains until laying season begins.[13] Free-ranging migratory ducks and geese consume high-energy feeds in preparation for migration. If such diets are fed year round in captivity, birds will become obese.

Pellet size should be 3-4 mm in diameter for hatchlings and 10-12 mm for birds after a few weeks of age. Insoluble grit should be supplied as soon as scratch grain is offered. At first the grit should be sprinkled over the diet, but later it may be placed in a container by itself. Some breeders include 4% grit in the pelleted ration.[13]

Feed containers should be at least 20 cm deep and 30 cm square to provide space for their normal forward-shoveling prehension behavior. Waterfowl should always have free access to fresh, clean water. Poultry water units and nipples may be used for waterfowl.[13]

RESTRAINT, ANESTHESIA, AND SURGERY[3,7]

Ducks, geese, and swans are not innately aggressive; however, large angry geese and swans may attack and inflict significant injury by beating the handler vigorously with their wings.[3] This author recalls only too well being chased to the house from the barnyard as a child by an angry goose pecking at his backside and being beaten by its wings. Both spur-winged geese and screamers have sharp spurs at the carpus of the wing, which may be deliberately and effectively used against an animal handler. When cornered, a screamer may fly at a person, flailing its wings.

Waterfowl may be netted or grasped by the neck by hand or with a hook on a long handle. The wings must be contained to avoid being beaten. Some waterfowl have beaks with sharp tips, and some have sharp toenails and scratch.[3] Once extracted from the net, small birds may be held by grasping the wings at the base, keeping a finger between the wings. Larger waterfowl may be caught by grasping the neck and then quickly grasping the wings to avoid flailing. If the bird is to be carried, it should be tucked under the arm with its head directed rearward and the wings folded to its sides. A special restraint jacket may be constructed employing adjustable Velcro strips to restrict wing movement, or the bird may be placed in a cloth sack containing a hole through which the head and neck may protrude.[13]

Catching waterfowl at night may be effective by using a bright spotlight to temporarily blind the bird, allowing it to be approached quietly.

The capture of free-ranging waterfowl requires a detailed knowledge of the biology of the species to be captured and special equipment. Cannon nets (rocket nets) are commonly used by biologists in studying waterfowl populations.[3] The net is a rectangle that is set in a line near an area where the birds have become accustomed to feeding on grain provided. The net is gathered to the line. The forward edge of the net is periodically attached to a weighted object that fits into a pipe set at an angle over the area to be covered by the net. The number of pipes needed depends on the size of the net to be used. At the base of the pipe a charge is placed. All of the charges are connected to a remote detonation device located at some distance from the net and out of sight of the birds. When a group of birds is feeding, the charges are fired simultaneously, the weighted object is propelled out of the pipe and over the area, carrying the net with it.

Anesthesia

Local anesthesia with 2% lidocaine hydrochloride may be used for suturing lacerations and performing minimally invasive procedures. Isoflurane gas administered through a precision vaporizer is the safest and most effective form of general anesthesia. Birds may be masked for induction of anesthesia and performance of short diagnostic or surgical procedures. For longer procedures the bird should be intubated by looking at the glottis through the open mouth. Halothane and methoxyflurane may also be used, but induction and recovery times are much longer than with isoflurane. Many other anesthetic agents have been employed in waterfowl for clinical, surgical, and experimental procedures, but the foregoing are recommended by this author.

Free-ranging waterfowl present special problems in anesthesia. A number of anesthetic agents (tribromoethanol, alpha-chloralose, methoxymol, metomidate, pentobarbital sodium, secobarbital sodium and thiopental sodium) have been given in feed to immobilize waterfowl. Tribromoethanol was the safest and most effective of any of these agents in one study.[13] Field usage suggests a ratio of 3 g of tribromoethanol per cup of whole corn. The bait is prepared by dissolving the agent in water and pouring it on an appropriate amount of corn situated in a shallow container to allow rapid drying with a fan. The birds must be trained to take untreated corn before the bait is offered. Immobilization may take 30–60 minutes, and the operation should not be hurried lest the birds be startled and fly off and die. Birds that enter water must be observed carefully so that they are collected before they drown.

The author suggested a different technique to people attempting to capture an injured duck at a park pond in

Las Vegas, Nevada, USA. The duck had been given the name “Donna” by the local press. Donna had been seen a month previously with an arrow impaled across her chest, and she had become a local celebrity. Numerous attempts by personnel from the local Humane Society had been made to capture her to remove the arrow. People from near and far volunteered all kinds of novel methods for catching her, all to no avail.

A technique that had been successfully used by California’s Department of Fish and Game to capture ducks on a pond near a California zoo was suggested. Personnel were instructed to start baiting all the ducks inhabiting the pond with pieces of bread until individual birds could be fed by tossing each a piece of bread. Donna came for her share of the bread. When the training process was completed, a piece of bread was injected with a solution of secobarbital sodium (commonly used as a sleeping pill for humans). Donna took the baited bread, and in 30 minutes a boat picked her up as she began to become immobilized. The arrow was easily removed and after recovery from the anesthesia, she was released back to the wild.

Surgery

Common surgical procedures include pinioning (see flamingo chapter), suturing lacerations, fracture repair, and surgical correction of bumblefoot lesions. Less common procedures include air sac cannulation to correct airway obstruction, beak repair, and castration or caponization. These procedures are performed as in other birds.

DIAGNOSIS

Clinical Examination

Use the same techniques as those used in other birds. Heart beat rate and breathing rates vary considerably, even in the same species, depending on the time of day and the stress level experienced by the bird. Diving birds have control over the heart rate, as evidenced by the rate slowing dramatically when the bird dives. As in other animals, a knowledge of the behavior of the species helps to identify possible problems. Waterfowl lose heat by panting and through their webbed feet. During cold weather they squat down and cover their feet to avoid heat loss from the body.

Waterfowl are quite long-lived. Ducks may live in captivity for 10–12 years and geese and swans 25 years or more. Longevity in free-ranging species is much less; small birds live up to 6 years and larger birds 10–15 years. Some studies have recorded 60–70% mortality in the first year and as high as 90 or 95% before 3 years of age.

Collecting Laboratory Samples

Blood may be collected from the medial metatarsal vein, the right jugular vein, or the ulnar vein. Hematologic and serum chemistry parameters are not known for most species of waterfowl, but general extrapolation from parameters for Peking and Muscovey duck data is a starting point.[16]

Special Diagnostic Procedures

Radiography, ultrasonography, and endoscopy procedures are the same as for other birds.

DISEASES

Diseases in Free-Ranging Populations

Much is known about the diseases of waterfowl because the birds are hunted as game species in most countries of the world. Governmental organizations expend large sums of money to monitor, diagnose, and conduct research on the diseases of this valuable resource. Populations of waterfowl are managed more intensively than any other group of birds or mammals. The diseases of free-ranging waterfowl may be restricted locally by geographical isolation, ecological situations, or management practices, but basically all the diseases seen in captivity are also seen in free-ranging waterfowl. Free-ranging waterfowl from anywhere in the world may potentially have interaction with other waterfowl and other avian species throughout the world (Figure 11.1). Thus, when discussing diseases of waterfowl, a world-view should be employed.

The human propensity to transport exotic species all over the world increases the risk of spreading infectious and parasitic agents through carrier birds. Quarantine is a vital requirement when new birds are acquired by a captive facility. Another factor affecting the spread of disease is that captive waterfowl are most commonly kept in outdoor enclosures accessible to other birds, particularly fly-in free-ranging waterfowl.

Waterfowl species vary in their susceptibility to infectious and parasitic agents, so it is not appropriate to extrapolate disease data from one population to another. No attempt will be made to discuss diseases diagnosed only in South America. South American species of waterfowl are exhibited and maintained throughout the world and are therefore subject to potential infection by any of the microorganisms infecting Anseriformes. Furthermore, species not native to South America are imported for exhibition and may bring along infections and parasites.

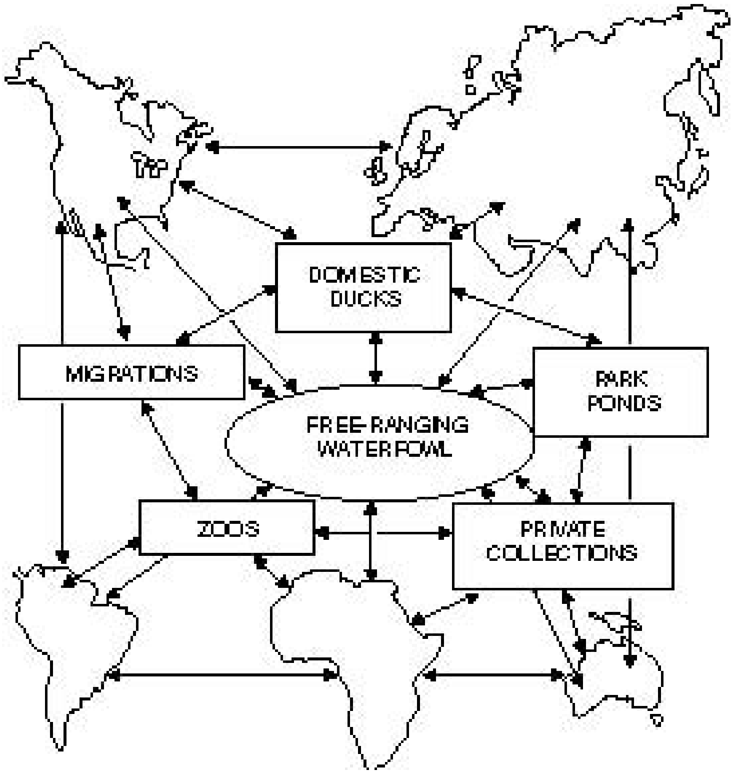


FIGURE 11.1. Interrelationships between domestic ducks and free-ranging water fowl.

Veterinarians who provide medical care for both free-ranging and captive waterfowl should be aware of local, state, province, or national regulations concerning these birds. Many countries have enacted legislation to safeguard their waterfowl populations. Because some species are migratory and cross international borders, the regulations may involve multiple countries. Permits for transporting waterfowl from one state, province, or country to another vary, and it is up to shippers and their veterinarians to ascertain the regulations and comply with any special tests required.

Infectious Diseases[2,8,10,13-15,18]

Fungal Diseases

Waterfowl are commonly affected by aspergillosis, especially birds that have been severely stressed, such as those being rehabilitated following an oil spill. Waterfowl may also become infected with *Candida albicans* and other fungal agents. The aquatic habitat is an ideal medium for fungal growth, and stressed birds are always at risk.

Bacterial Diseases

Waterfowl are subject to infection with numerous ubiquitous organisms including *Pasteurella multicida* (avian cholera), *Mycobacterium avium* complex (avian tuberculosis), *Salmonella* spp., *Erysipelothrix rhusiopathea* (erysipelas), *Yersinia* spp. (yersiniosis), *Mycoplasma* spp. (mycoplasmosis), and *Chlamydia* spp. (chlamydiosis) (Table 11.2).

AVIAN CHOLERA Avian cholera is an important cause of mortality in free-ranging populations of waterfowl throughout the world. In the wintering areas of central California, as many as 80,000 birds may die annually from this peracute disease. Although avian cholera in waterfowl has been studied intensively for over 100 years, it is still not known how and where the organism resides between epizootics and what triggering mechanisms initiate an epizootic. Neither is the mode of transmission in waterfowl well understood. Outbreaks of avian cholera are usually associated with dense concentrations of birds, such as in wintering areas. Not all species of waterfowl are affected equally during epizootics, and the vulnerability may change from year to year in the same location.[8,18]

The clinical signs in waterfowl are those of a peracute septicemia. Sudden death may be preceded by apathy, neuromuscular disorders, and diarrhea. The cause of death appears to be an endotoxin produced by the organism. Lesions may be minimal, but petechiae of the epicardium serosa of the gizzard and other serous membranes are seen on necropsy. If the bird survives for a few hours, necrotic foci will appear in the liver along with generalized petechiation. Pneumonia may be seen

TABLE 11.2. Selected diseases of South American Anseriformes

Disease (English)	Disease (Spanish)	Disease (Portuguese)	Etiology	Transmission	Signs	Lesions	Diagnosis	Management
Duck viral enteritis (Duck plague)			Herpes virus	Ingestion, inhalation, carrier birds	Nasal discharge, diarrhea, epiphora, photophobia, acute death, depression	Enteritis, hepatic foci, reddened annular bands or patches in the intestines	Signs, lesions	No therapy, live virus vaccination, isolation of sick birds
Duck viral hepatitis	Enteritis viral de los Patos		Picornovirus	Ingestion; if the bird lives it becomes a carrier	Depression, CNS signs, peracute death	Hepatitis	Signs, lesions	Hyperimmune serum, vaccination
Avian influenza	Influenza	Influenza	Orthomyxovirus	Ingestion, direct contact, highly contagious	Primarily ducklings, depression, spasms of legs, opisthotonus	Hemorrhage in the liver, spleen, and kidneys	Signs, lesions, virus isolation, serum neutralization test	Reduce stress and crowding, supportive care
Avian cholera (Pasteurellosis)	Pasteurelosis	Pasteurelose	*Pasteurella multicida*	Direct contact, inhalation	Dyspnea, apathy, diarrhea, sudden death	Hemorrhages on serosal surfaces, foci in liver	Virus isolation	Carcass collection, reduce concentration of birds
Colibacillosis	Colibacilosis	Colibacilose	*Escherichia coli*	Direct contact, ingestion	Anorexia, diarrhea, dyspnea, sudden death, omphalitis	Enlarged liver, hemorrhages of serosal surfaces	History, lesions, culture	Collect carcasses, reduce concentration of birds
Botulism	Botulismo	Botulismo	*Clostridium botulinum* (toxin)	Ingestion of fly larvae containing toxin	Muscle paralysis of neck, wings, legs, and muscles of respiration	None	History, signs, mouse protection test	Remove all carcasses daily, maintain steep sided pools
Erysipelas	Erisipelas	Erysipelose	*Erosipelothrix insidiosis*	Ingestion, wound infection	Depression, anorexia, diarrhea	Hemorrhages on serosal surfaces	Culture	Sanitation, vaccinate with bacterin
Salmonellosis	Salmonelosis	Salmonelose	*Salmonella* spp.	Ingestion of contaminated feed	Depression, diarrhea, sudden death	Enteritis, septicemia	Culture	Antibiotics, remove carriers
Aspergillosis	Aspergilosis	Aspergilose	*Aspergillus fumigatus*	Airborne spores, moldy feed	Dyspnea	Fungal plaques in air sacs and lungs	Direct smear, culture, lesions, endoscopy	Prevent exposure
Chlamydiosis (Ornithosis)	Clamidiosis, ornitosis, psittacosis	Chlamydiose	*Chlamydia psittaci*	Inhalation, ingestion of contaminated feed	Dyspnea, rhinitis, sinusitis, diarrhea, weakness	Enlarged spleen and liver, air sacculitis	Culture	Isolation, antibiotic (doxycycline)
Tuberculosis	Tuberculosis	Tuberculose	*Mycobacterium avium*	Inhalation, ingestion of contaminated feed	Emaciation, diarrhea	Granulomas, histiocytic infiltration of intestinal wall	Necropsy	Sanitation, isolate sick birds, depopulate
Newcastle disease	Enfermedad de Newcastle	Doença da Newcastle	Paramyxovirus, NDV 1	Direct contact, contaminated feed	Conjunctivitis, dyspnea, CNS signs, diarrhea	Multiple organ systems involved	Serology, embryonated egg inoculation	Isolate sick birds, depopulate

Note: CNS, central nervous system.

in some birds. The diagnosis is based on the isolation and identification of *Pasteurella multicida*.

Management of an outbreak is difficult, but the most useful measures are: (1) regular monitoring of large waterfowl concentrations so that mortality may be detected at an early stage; (2) rigorous collection and incineration of carcasses of all birds found dead; and (3) control of scavenging birds to prevent dispersal of the disease. The ultimate step in outbreak control is depopulation.[8,18]

AVIAN TUBERCULOSIS[2,13,18] Although not as lethal as avian cholera, tuberculosis is a difficult challenge to those maintaining captive collections of waterfowl. Avian tuberculosis is a chronic wasting disease and is difficult to diagnose antemortem.

Viral Diseases[2,13,15]

Table 11.2 lists some of the numerous viral diseases of waterfowl. Space does not permit discussion of the majority of these diseases. Textbooks have been written on the subject of waterfowl infectious and parasitic diseases, and viral diseases play a prominent part in those discussions.[2,13,15,18] Following is a list of known viral diseases of waterfowl: Duck plague (herpesvirus), influenza A, Newcastle disease (paramyxovirus), duck viral hepatitis (picornavirus, astrovirus, hepatitis B), eastern encephalitis (arbovirus), avian encephalomyelitis (picornavirus), goose virus hepatitis (parvovirus), reticuloendotheliosis (retrovirus), avian pox (poxvirus), and miscellaneous other viruses (reovirus, adenovirus).

Parasitic Diseases[2,5,13,18]

As in other taxa, parasitic agents have evolved with Anseriformes and in the free-ranging state don't usually cause serious clinical disease. In captivity the control of parasites is an ever-present challenge.

PROTOZOA *Leukocytozoon simondi* is a cause of mortality in free-ranging geese nestlings in North America. Other haematozoan parasites include *Haemaproteus* spp. and *Plasmodium* spp. The parasites may cause variable disease in geographical regions and in various species.[2,5]

Coccidiosis may occur as an enteritis (*Eimeria* spp., *Tyzzeria* spp., *Wenyonella* spp., and *Isospora* spp.), but also as renal coccidiosis (*Eimeria boschadis,* ducks; *Eimeria truncata,* geese; and *Eimeria christianseni, mute swans)*.[18] The life cycles, clinical signs, diagnosis, lesions, and management are the same as for other birds. Additional coccidian parasites include *Cryptosporidium* spp. and *Sarcocystis* spp.

Waterfowl may be infested by ectoparasitic arthropods, including lice (Mallophaga), mites and ticks (Acarina), biting mosquitoes and flies (Diptera), fleas (Siphonaptera, and bugs (Hemiptera).[18] Generally, ectoparasites do not cause clinical disease in waterfowl; however, fatal myiasis (*Wohlfahrtia opaca)* has been observed.[18] Leeches (*Theromyzon* spp.) affect both domestic and captive wild waterfowl and free-ranging birds. The leeches may be found attached within the nasal cavity, pharynx, trachea, or conjunctival spaces of waterfowl. Heavy infestation may be lethal, otherwise little disease is noted.

Internal parasitism is caused by trematodes, cestodes, nematodes, and acanthocephalids. Clinical signs, diagnosis, lesions, and management are as in other avian species.

Noninfectious Diseases[4,13]

Noninfectious diseases are numerous. Three major noninfectious diseases are botulism, lead poisoning, and oil contamination. Other heavy metal toxicities include mercury, copper, selenium, zinc, cadmium, and vanadium. Pesticides (organophosphates, chlorinated hydrocarbons, carbamates) contribute to mortality in free-ranging populations. Miscellaneous toxicities include mycotoxicosis (aflatoxicosis, ergotism), algal poisoning, marine dinoflagellates (red tide), petroleum (oil spills), fluoride, phosphorus, and poisonous plants (oleander, castor beans).

Nonpoisonous noninfectious diseases include malnutrition, angel wing, trauma (lacerations, fractures, hematomas), inclement weather, exertional myopathy (capture myopathy), gout, amyloidosis, neoplasia, and esophageal impaction.[18]

Several diseases of waterfowl are associated with nutritional deficiencies or excesses, including angel wing, perosis, and rickets.

ANGEL WING In angel wing (slipped, airplane, crooked, rotating, or dropped wing) the wing is twisted at the carpus under the excessive weight of the emerging primary flight feathers. The cause may be multifactorial, but certain species of waterfowl are prone to develop this disorder if fed high-energy/high-protein diets that cause rapid growth. Hypovitaminosis E may also be involved. Diagnosis is easy because the twisted wing is evident. Management should include reduction in feed intake and the splinting of the distal wing into its proper position during the period of pinfeather emergence. The wing should not be taped to the body. Although angel wing is primarily a disorder of captive waterfowl, it is known to occur in free-ranging birds (waterfowl and gulls).

PEROSIS Perosis (slipped tendon)is characterized by enlargement of the hock, bending deformities of the mediotarsal and tarsal metatarsal bones, and medial luxation of the Achilles tendon, which prevents the bird from bearing weight on the affected limb.[13] The etiology is thought to be a manganese deficiency in the diet of either the mother during egg laying or of the hatchling. Excess calcium may bind manganese and contribute to the deficiency. The prognosis for the affected bird is usually poor, but some success has followed surgical intervention to replace the tendon into its proper trochlear groove and suture the tendon sheath to the lateral periosteum.[13]

RICKETS Rickets ((nutritional secondary hyperparathyroidism, fibrous osteodystrophy, osteomalacia)may occur in birds fed a diet deficient in calcium or with an improper calcium/phosphorus ratio. Waterfowl deprived of sunlight, proper ultraviolet light exposure, or vitamin D_3 in the diet may also become rachitic or develop one of the other forms of metabolic bone disease. The clinical signs, diagnosis, and management of rickets in waterfowl are the same as for other avian species.

BOTULISM Botulism (western duck sickness in Western North America)[9] is caused by ingestion of a neuroparalytic toxin produced by *Clostridium botulinum,* a gram-positive, motile bacillus that is an obligate saprophytic anaerobe. A number of strains of the toxin affect different species, but waterfowl are affected by Type C toxin. Transmission is primarily by ingestion of fly larvae (maggots) infesting carcasses of birds that have died of botulism. Warm weather facilitates the growth of the organism and production of the toxin. Botulism causes considerable mortality during the warm months of the year in certain sections of North, Central, and South America, several European countries, South Africa, Australia, and New Zealand. Variation in the percentage of a given species affected may be a reflection of the feeding habits of the species, hence exposure to the toxin, but species also vary in susceptibility to the toxin.[18]

The spores of *C. botulinum* may persist in the soil beneath ponds or marshes for years. A change in the water level, hot weather, or change in the salinity may cause mortality in the invertebrate population, which in turn becomes a medium for the growth of the organism. Birds that die from any cause become an anaerobic medium for production of the toxin, and the fly larvae that infest the carcass become missiles laden with the toxin to be ingested by other birds.

The botulism toxin causes flaccid paralysis of the muscular system. Birds in the water are unable to keep their heads out of the water and drown. In the early stages birds are observed to be weak, unable to fly, the wings are drooped, and the bird may be unable to stand. A lethal ingestion results in paralysis of the respiratory centers of the brain and the muscles of respiration.

Diagnosis is based on the history (time of year and environmental situation) and clinical signs. It is possible to test for the toxin, but this is usually not necessary. Lead poisoning may cause some of the same signs, but its presentation is different.

The most important aspect of management of botulism is to quickly remove all carcasses from the marsh daily to prevent buildup of the toxin in fly larvae. The water level of ponds should be maintained to avoid killing the invertebrates. Botulism tends to occur most often in shallow ponds with sloping bottoms. Maintaining steeper banks and uniform depth of ponds are long-term goals with recurrent botulism areas.

Antitoxin is a specific therapy, but is usually not available in sufficient quantity to be of any value in an outbreak, but 75 IU of antitoxin intraperitoneally will substantially increase the survival rate.[18] Flushing the digestive tract with quantities of fresh water will save many birds with mild to moderate signs.

LEAD POISONING Lead shot was the pellet of choice for hunting waterfowl for decades. Lead shot can no longer be used in the United States, but tons of lead have been deposited in the mud of many bodies of water frequented by waterfowl. Other countries may still permit use of lead shot. Spent shot settles to the bottom of ponds, where it may lie inert for years. Waterfowl foraging in the mud pick up the pellets, which remain in the gizzard as grit. The acidic conditions of the gizzard dissolve elemental lead into an ionic form that is absorbed into the bloodstream. Lead accumulates in the bones, liver, and kidneys and a few other tissues, but when serum levels become elevated (>40 μg/dL), signs of acute lead toxicity develop. Clinical lead poisoning may not be the most damaging aspect of lead pollution to a population of wild waterfowl. The sublethal effects of lead may cause immunosuppression, leading to decreased resistance to infectious and parasitic agents, reduced coordination resulting in increased predation and collision with electrical transmission lines, and anemia.

Lead is a systemic poison that may affect most tissues and organ systems of the body, but effects on the nervous and digestive systems predominate. Signs are variable, but include lethargy, progressive weakness, anorexia, green liquid feces (hemorrhagic diarrhea is more common in psittacine birds), ataxia, weight loss, and emaciation. Proventricular and esophageal impactions are common. Anemia will be noted upon evaluation of the hemogram.

Diagnosis is based on analysis of blood (>40 μg/dL), liver (4 to >6 parts per million [ppm], wet weight) and kidney (15 to >20 ppm, dry weight) for lead levels, plus clinical signs and lesions. Radio-dense objects in the area of the gizzard upon radiographic evaluation are a good indication of exposure. Pellets that are in the muscles or other tissues are rarely cause for concern for lead poisoning because acidic conditions are necessary to ionize elemental lead.

Prevention and management of lead poisoning in waterfowl focuses primarily on decreasing the amount of lead shot available for ingestion. Cessation of the use of lead shot is practiced in the United States, Canada, Denmark, the Netherlands, and Australia. This has had considerable impact on the prevalence of clinical lead poisoning. Deep tillage to bury shot was a viable management option on a wetland with continuing problems of lead poisoning despite use of nontoxic shot.[18]

Systemic treatment with chelating agents such as calcium disodium versenate (calcium ethylenediaminetetraacetate [EDTA]) are of no practical use in treating clinically affected free-ranging waterfowl, but may be life saving for valuable breeding birds in zoo and private collections. The recommended dose is 10–40 mg/kg administered twice daily intramuscularly for 3 days. This must be accompanied by removal of shot from the gizzard by administration of a cathartic or by feeding the fowl peanut butter on bread. Repeated administration of calcium EDTA is needed if signs recur or if radiography indicates that lead pellets remain in the digestive tract.

OIL CONTAMINATION Oil contamination of oceans, rivers, and lakes has increased significantly in the last few decades from accidents occurring with the transport of crude and refined petroleum products. An additional source of oil contamination is birds landing on small ponds associated with land oil fields. Waterfowl are frequently the major group of animals affected by oil released into the environment.

Oil discharges may contaminate the birds' food sources, their nesting habitat, and even eggs in the nest, thus reducing hatchability. The external effects on the bird include the disruption of the interlocking mechanism of the barbicels of the feather, thus decreasing insulation and waterproofing. Hypothermia is a serious consequence of oil contamination. Other external effects include dermatitis, keratitis, and conjunctivitis. Internally, petroleum products produce variable toxic effects on the intestines, liver, kidney, and the hematopoietic system. Pneumonia is a common sequel to inhalation of lipid particles, producing a foreign-body pneumonia. Oiled birds are stressed severely, thus aspergillosis and other opportunistic diseases cause secondary infection.

The management of oiled waterfowl has been perfected over the years because of the frequency of oil spills and the need to manage from single birds up to hundreds of birds at a time.[12] As in any rehabilitation operation, each bird should be evaluated through triage. Those that are deemed capable of living must be stabilized by dealing with hypothermia, dehydration, traumatic injury, and concurrent disease. Once stabilized, cleaning may commence.

Each bird is cleaned by a team of 2–4 people. The bird's head should be held out of the water and cleaned later with a toothbrush or a cloth. The eyes should be irrigated with normal saline. Large tubs holding 40–80 L (10–20 gallons) of water at 40–45°C (103–105°F) are used. Approximately 400 L (100 gallons) of water are needed over a 30-minute period to clean one duck. The first tub should contain a 5% solution of the detergent. Experience has shown that Dawn (Joy in Canada and Central and South America; Fairy Liquid in England and South Africa [Proctor & Gamble, Cincinnati, Ohio, USA]), a dishwashing detergent, is most suitable.[12] Once the water becomes heavily contaminated with oil, the birds should be moved to another tub of 4% detergent. A succession of 8–10 changes (down to a 1% solution) may be necessary to clean heavily contaminated birds.

When cleaning is finished, the bird should be thoroughly rinsed with clean warm water spray at 40–60 psi pressure. Failure to rinse the bird is the most common mistake made. Access to water within 24 hours will start the process of restoring waterproofing. The bird enters the water, and at first will quickly get wet. It will come out of the water and begin preening, which is the means of reestablishing the interlocking mechanism of the feather. Keep in mind that ingested oil may pass with the feces and contaminate the bird if a filtration system or constant overflow is not used.[12]

Other factors in the rehabilitation process include stress reduction, nutrition, fluid support, housing, pools, saltwater, social grouping, temperature, ventilation, and prerelease examination.[12]

EXERTIONAL MYOPATHY[4,13] Prolonged struggling against restraint may cause lactic acid buildup in the muscles and subsequent myonecrosis. This condition can occur when waterfowl are captured with a cannon net. If the birds are left to struggle beneath a net for a prolonged period of time (lack of sufficient personnel to handle the number of birds captured), some of the released birds may be unable to walk away when released and may subsequently die from myopathy of various muscles. The condition is easily prevented by strategically locating a number of blocks in the area where the net is to be projected. The blocks prevent the net from falling directly on the birds, and allows them to move about under the net until they can be handled by the investigators.

REFERENCES

1. Cambre, R.C. 1999. Water quality control for a waterfowl collection. In M.E. Fowler and R.E. Miller, eds., Zoo and Wild Animal Medicine, 4th Ed. Philadelphia, W.B. Saunders, pp. 292–299.
2. Davis, J.W.; Anderson, R.C.; Karstad, L.; and Trainer, D.O. Eds. 1971. Infectious and Parasitic Diseases of Wild Birds. Ames, Iowa, Iowa State University Press.
3. Fowler, M.E. 1995. Restraint of water birds. In M.E. Fowler, ed., Restraint and Handling of Wild and Domestic Animals, Ames, Iowa, Iowa State Univ. Press, p. 316.
4. Hoff, G.L.; and Davis, J.W. 1982. Noninfectious Diseases of Wildlife, Ames, Iowa, Iowa State University Press.
5. Humphreys, P.N. 1986. Parasitic diseases. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 353–355.
6. Humphreys, P.N. 1986. Reproduction. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 356–357.
7. Humphreys, P.N. 1986. Restraint. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 341–342.
8. Jessup, D.A. 1986. Avian cholera. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 347–348.
9. Jessup, D.A. 1986. Botulism. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 349–353.
10. Jessup, D.A. 1986. Duck virus enteritis. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 342–345.
11. Kear, J. 1986. Feeding and nutrition. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 335–344.
12. Miller, E.A.; and Welte, S.C. 1999. Caring for oiled birds. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 4th Ed. Philadelphia, W.B. Saunders, pp. 300–308.
13. Olson, J.H. 1994. Anseriformes. In B. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Applications. Lakeworth, Florida, Wingers Press, pp. 1237–1275.
14. Redig, P.T. 1993. Avian aspergillosis. In Fowler, M.E., ed., Zoo and Wild Animal Medicine, 3rd Ed. Philadelphia, W.B. Saunders, pp. 178–181.
15. Ritchie, B.W. 1995. Avian Viruses: Function and Control. Lakeworth, Florida, Wingers Publishing.
16. Shave, H. 1986. Clinical pathology. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 357–362.
17. Todd, F.S. 1979. Waterfowl: Ducks, Geese and Swans of the World. New York, Harcourt Brace Jovanovich.
18. Wobeser, G.A. 1997. Diseases of Wild Waterfowl, 2nd Ed. New York, Plenum Press.

12
Order Falconiformes (Hawks, Eagles, Falcons, Vultures)

Juan Carlos Chebez
Roberto F. Aguilar

BIOLOGY

Juan Carlos Chebez

INTRODUCTION

Birds of prey (raptors), including carrion eating New World vultures, belong to the order Falconiformes. Their important ecological role and their domain of the heights, their elegant and calm flight, as well as their ability to chase prey at high speeds, have amazed humanity since ancient times, with some civilizations even worshiping certain species.

Birds of prey efficiently control many insect, bird, and rodent populations considered to be agricultural pests or disease reservoirs for humans, thus enabling people to avoid the use of expensive pesticides or toxic agents that may contaminate the environment. Unfortunately, the destruction of the rain forests is endangering many South American species and causing problems for migratory species such as Swainson's hawk (*Buteo swainsoni*) that nest in North America and migrate to the Argentine pampas, where a massive poisoning with pesticides occurred. Many raptors are shot, used as target practice, or because they are mistakenly considered to be dangerous to domestic bird species or cattle.

South America is refuge to approximately 92 species of birds of prey that occupy a wide variety of ecosystem niches. Much remains unknown about these birds, making their conservation and proper management a true challenge.

TAXONOMY

Table 12.1 lists the four suborders and five families that form the order Falconiformes, including all extant genera and species, both in South America and worldwide. Three of the four suborders are represented in South America. The only one not found in South America is represented by the secretary bird, an African bird of prey with a remarkable neck, a feather crest, and long legs. The Cathartae belong to the cathartid family, which includes turkey vultures and urubús (mistakenly called crows), as well as the large condors, known for their wide wings that make them efficient gliders-experts in soaring on rising warm air currents. These birds have featherless heads with occasional bright coloration, and a crest, which helps to differentiate the sexes. These traits are an adaptation to their necrophagic habits. The beaks are strong in order to open holes in the cadavers on which they feed.

TABLE 12.1. Taxonomy of the order Falconiformes

		Genus		Species	
Suborders	Families	World	South America	World	South America
Cathartae	Cathartidae	5	4	7	6
Accipitres	Pandionidae	1	1	1	1
	Accipitridae	64	25	237	61
Sagittarii	Sagittariidae	1	—	1	—
Falcones	Falconidae	10	8	61	24
	Total	81	38	307	92

They have strong legs, but their fine talons cannot be used to carry food in flight.

Their outward appearance is reminiscent of Old World vultures, but those are considered Accipitridae; birds of prey that eat carrion. New World vultures are related to the Ciconidae (storks, jabirus, marabus, etc.), proven by DNA analysis. Nevertheless, for the purposes of this discussion, they will be considered members of Falconiformes.[4]

The suborder Accipitres is composed of two families. The Family Pandionidae consists of a monotypic genus, represented by the osprey (*Pandion haliaetus*), a migratory fish-eating species of Palearctic and Pan-American distribution. It nests in North America, building large nests of sticks in trees near water, and migrates to South America in the winter, where it has been reported in every country. This bird is easily distinguished by its long, pointed wings, long tail, and white underside that contrasts with the black dorsum, and a thin, dark brow. It also has a distinctive feather crest.

The second family is composed of the Accipitridae, with 25 genera and 61 species in South America alone. The South American species represent approximately 25.7% of the birds in the family worldwide. The subcontinent species are varied, all adapted to different habitats and diets, and include eagles, hawks, sparrow hawks, goshawks, buzzards, and broad-winged hawks. They are good hunters, with long, relatively broad and rounded wings. The tails are short, with some exceptions observed in forest-dwelling species. The beaks are strong and hooklike and occasionally have notches. The eyes are large, and the overdeveloped supercilliary cranial arches give these birds a stern appearance. The legs are strong, and the feet have long and powerful talons that vary in size and shape by species. In most species, the female is larger than the male, and there is a marked difference between juvenile and adult plumage. Juvenile plumage is not replaced for a long time to avoid attacks from other members of the same species because of territorial disputes, mating-related aggression, or nesting competition.

The suborder Falconae contains a single family, the Falconidae, composed of two well-differentiated groups. The falcons (subfamily Falconinae) have an aerodynamic shape, long pointed wings, and short, notched beaks with a hooklike shape. They are adapted to capturing their prey in the air, developing high-velocity stoops for that purpose. The other subfamily, Polyborinae, includes the so-called robust species, which are mainly carrion-eating birds, though some of them hunt, circumstances permitting. They are good walkers and build poorly structured nests. Members of this group are the caracaras (*Polyborus plancus*), chimangos (*Milvago chimango*), yellow-headed caracaras (*Milvago chimachima*), and other caracaras (*Phalcoboenus* spp.), as well as some other species, such as the laughing falcon (*Herpetotheres cachinnans*), of ophidiophagus habits, and the forest falcons (*Micrastur* spp.). The forest falcons are characterized by a long tail and round wings. They specialize in the capture of forest-dwelling birds and rodents.

DISTRIBUTION

Birds of prey colonize almost every continent and region of the globe, with the exceptions of Antarctica and some oceanic islands. Table 12.2 lists the species, divided by families, and distribution throughout South America. The four countries with the greatest diversity of raptors are Colombia (76 species), Ecuador (70 species), Peru, and Brazil (69 species each). A large number of raptor species inhabit the length of the Andes mountain range.

The three countries with the least raptor biodiversity are Paraguay (51 species), Uruguay (28 species), and Chile (21 species). In Chile, the lack of variation in habitat diversity (puna and high Andes, Mediterranean xerophilic, austral or subantarctic forests) and a relatively narrow territory result in a lack of faunistic diversity. Uruguay is even smaller, but has subtropical species because of its gallery of rain forests and small rain for-

TABLE 12.2. Species diversity of Falconiformes in South American countries

	Families				
Country	Cathartidae	Pandionidae	Accipitridae	Falconidae	Total
Colombia	6	1	51	18	76
Ecuador	5	1	46	18	70
Perú	6	1	45	17	69
Brazil	6	1	45	17	69
Bolivia	6	1	44	15	66
Venezuela	6	1	44	15	66
Argentina	6	1	40	15	62
Guyanas[a]	5	1	33	15	54
Paraguay	4	1	34	12	51
Uruguay	3	1	18	6	28
Chile	3	1	10	7	21

Source: See reference 1.
[a]For practical reasons, Guyana, French Guiana, and Surinam are considered as a single country.

est islands in the marshy northern areas of the country. Of these three, Paraguay has the greatest raptor diversity thanks to its subtropical climate. The large eastern section forms part of the Parana rain forest, also known as the interior Atlantic rain forest.

HABITAT

The variety of natural habitats occupied by birds of prey is enormous. They have colonized all available habitats in South America, developing unique anatomical and behavioral characteristics in the process. There are many forest-dwelling genera distributed in tropical (i.e., Amazon) and subtropical (i.e., Parana) forests: *Cathartes melambrotus, Harpia, Morphnus, Spizaetus, Spizastur, Harpagus, Leucopternis, Daptrius,* and *Micrastur,* to name a few. Others prefer savannah and grassy plains, like the Llanos of the Orinoco, the Matogrosso Pantanal, and the Chaco regions. Genera include *Sarcoramphus, Elanoides, Ictinia, Buteogallus, Heterospizias, Chondrohierax, Leptodon, Buteo, Accipiter, Gampsonyx, Geranospiza, Harpyhaliaetus (H. solitarius), Milvago (M. chimachima), Herpetotheres, and Falco.* A few of the marshland and pampas grassland dwellers are *Cathartes (Cathartes burrovianus), Rostrhamus (R. sociabilis), Circus (Circus buffoni), Busarellus,* and *Pandion.*

Open arid areas are home to *Geranoaetus* (which may also be found in some mountain regions), *Harpyhaliaetus, Parabuteo* (sometimes also seen in the pampas grasslands and the humid Chaco), and *Circus cinereus, Buteo swainsoni* (which migrate en masse from North America), *Buteo polyosoma, Spiziapteryx,* as well as some species of *Falco.*

Species of the Andes include *Vultur gryphus* (with some sightings in southern Brazil and Paraguay), *Phaloboneus megalopterus, Buteo ventralis,* and *Buteo albigula.* The last two of these species are seen in the subantarctic forest and migrate north along the Andean mountain range in the winter.

Phalcoboenus albogularis is mainly found in the subantarctic forest. One species inhabits the subantarctic forest exclusively, *Accipiter chilensis.* Some authors consider it to be a subspecies of *Accipiter bicolor.* Other specialized species are *Phalcoboenus australis,* which is adapted to live near seabird and marine mammal colonies on the Falkland (Malvinas) coast and the archipielago of Tierra del Fuego; *Buteo galapagoensis,* exclusive to the Galapagos Islands, and its conspecific, that lives in the Juan Fernández Archipelago (Chile) in the South Pacific. This last raptor is considered to be a separate species and a subspecies of *Buteo polyosoma (Buteo polyosoma exsul)* by some authors.

Finally, some species may be observed in varied habitats, including regions altered by deforestation, which actually seems to favor their expansion. These genera include *Coragyps, Cathartes (Cathartes aura), Elanus, Polyborus, Milvago (Milvago chimango) and Falco (Falco sparverius).*

FIELD STUDIES

Birds of prey have always attracted humans. This has lead to their intensive study by ornithologists and other bird lovers, eager to know more about their ecology and habits. The habits of some species, mostly forest dwelling, have not yet been well described, but most of the species have been studied in detail. It would be impossible to summarize all the pertinent information in this chapter. A detailed, updated description of every species may be found in Del Hoyo[4] et al. Many field guides help in the identification of South American raptor species,[4,6] but a field guide to South American birds of prey with descriptions of plumage variation and stages is not yet available. Such a guide would help in identification of rare or problematic species. Sick[7] has published a detailed and complete guide to Brazilian birds of prey. Fjeldsa and Krabbe[5] have described Andean species. Collar[2] et al. discuss some endangered species in detail (particularly *Accipiter gundlachi, Leucopternis lacernulata, Leucopternis occidentalis, Harpyhaliaetus coronatus, Buteo ridgwayi, Micrastur buckleyi,* and *Micrastur plumbeus)*. Chebez[3] has described *Harpia harpyja, Morphnus guianensis, Leucopternis polionota,* and *Falco peregrinus* in an overview paper on the endangered wildlife of Argentina.

CONSERVATION PROGRAMS

Some birds of prey, such as the California condor (*Gymnogyps californianus*) and the Mauritius falcon (*Falco punctatus*), have become worldwide flagship species for the fight against extinction. In both cases, breeding in captivity was of vital importance to their recovery. Many threats hover over birds of prey. Natural habitat destruction has the most serious consequences, because it prevents the birds from procuring food and nesting areas. Hunting, because of the belief that they threaten domestic livestock, and indiscriminate target shooting contribute greatly to their depopulation.

Toxicity resulting from the use of pesticides may affect a raptor's reproductive behavior and may also impede calcification of the egg shells (as seen in the cases of the bald eagle, the white-tailed eagle, and the peregrine falcon). These same toxins may have a direct effect, leading to death by acute poisoning. Recent cases of mortality of *Buteo* caused by pesticides have been reported in the Argentine pampas.

The creation of natural wildlife reserves for the totality of the species and their populations would be the most advisable measure, followed by studies of the different species, especially of those considered to be most endangered. Breeding in captivity should be a last resort, left for those species that merit the economic and personal investment, counterbalanced with the real possibility of survival of wild populations.

Rehabilitation centers are of great help to birds (sometimes endangered or rare) brought in by well-meaning persons after the birds have been injured by cars, electrical cables, or firearms. In that case, the management in captivity of birds of prey is highly advisable.

REFERENCES

1. Altman, A.; and Swift, B. 1993. Checklist of the Birds of South America, 3rd Ed.
2. Collar, N.J.; Gonzaga, L.P.; Krabbe, N.; Madroño Nieto, A.; Naranjo, L.G.; Parker, T.A, III.; and Wege, D.C. 1992. Threatened Birds of the Americas: The ICOP/IUCN Red Data Book. Cambridge, IUCN.
3. Chebez, J.C. 1994. Los que se Van. Especies Argentinas en Peligro. Buenos Aires, Argentina, Albatros.
4. Del Hoyo, J.; Elliott, A.; and Sargatal, J. 1994. Handbook of the Birds of the World, Vol. 2. New World Vultures to Guineafowl. Barcelona, Spain, Lynx Editions.
5. Fjeldsa, J.; and Krabbe, N. 1990. Birds of the High Andes. Copenhagen, Zoological Museum of the University of Copenhagen.
6. Olrog, C. 1968. Las Aves Sudamericanas. Una Guía de Campo, Vol. 1. Tucumán, M. Lillo.
7. Sick, H. 1985. Ornitologia Brasileira, una Introducao, Vol. I. Brasilia, Editora Universidade de Brasilia.

RAPTOR MEDICINE AND SURGERY

Roberto F. Aguilar

Extensive clinical and research data dealing with raptors is available in the literature. The following information is presented as a guideline for the interested practitioner. The references listed at the end of this section provide further information.

PHYSICAL EXAMINATION

The physical examination of raptors follows the same guidelines as those recommended for most birds. It is necessary to maintain control of wings, talons, and head, so the assistance of an experienced restrainer is advised. A systematic approach in examination is useful to avoid omitting organs or systems. Full fundic ocular exams, though difficult with a direct ophthalmoscope, are possible in Strigiformes, but somewhat limited in Falconiformes. Careful examination and palpation of all accessible organs and systems may be invaluable to the trained practitioner. Most fractures are palpable to the practiced clinician, and the information gained may be critical in establishing a proper course of action. Skeletal and articular abnormalities, localized inflammation, abnormal limb posture, range of motion of limbs, and hydration may all be evaluated by careful palpation.

Radiography

Radiography of the entire body is recommended during an examination. Positioning is key, with ventro-dorsal and lateral views of the whole body being the most frequent diagnostic positions. Rare-earth film is recommended, along with high-intensity screens for table-top radiography. Bilateral symmetry is important for diagnostics, especially when working with unfamiliar species. With 300 mA of current output at 1/20 of a second exposure time, 52 kilovolt (peak) (kVp) is sufficient for adequate penetration and imaging in a Harris hawk (*Parabuteo unicinctus*), whereas up to 66 kVp may be needed for a harpy eagle (*Harpia harpyja*). Head studies for most species can be performed with 52 kVp at 300 mA in 1/26 of a second (12.5 mA-s). Small birds and the extremities of medium-sized birds may require as little as 48 kVp at 200 mA in 1/12 of a second (12.5 mA-s). Fractures and metal-density gastric foreign bodies are the most frequently radiographic diagnoses, although luxations, soft tissue swelling, air sac granulomas, and splenic enlargement can be observed by the experienced clinician and are valuable diagnostic indicators.

Fluid Therapy

Basic principles of fluid therapy are the same as those applied to most birds. It is important to establish the level of hydration upon examination of the animal. Simple manipulation of the taut skin over the keel will allow the clinician to evaluate hydration of most raptors. Loose, freely motile dermis over the keel is indicative of good hydration. When 1–3% dehydrated, the raptor's skin loses its pliability, and when 5–7% of dehydration is estimated, the skin is difficult to move over the keel and tends to "tent" on pinching. The eyes also become slightly sunken and the mucous membranes are evidently dry and "muddy" in color. It is critical that fluids be replaced when the animal is handled, because most raptors obtain their water from metabolic sources in their food (fat degradation) and may become further dehydrated if they fail to eat for a day or two after examination. Fluids may be supplied by intraosseous, intravenous, subcutaneous, or oral routes. Lactated Ringer's solution is usually used, although the use of colloids has been advocated for raptors with moderate to severe blood loss. Fluid deficit is calculated based on weight (i.e., 10% deficit would be 10% of the weight of the bird in kilograms) and 50 mL per kilogram are added for daily maintenance to be supplied over a 24-hour period. It is recommended that hydration be spread out over several doses in a day, if the animal can tolerate the manipulation. Intravenous administration of fluids may be tolerated as a bolus, but it is best delivered slowly over time, as a continuous drip.

NUTRITIONAL DISEASE

Nutritional deficiencies and deficits tend to be common in raptors kept in captivity by inexperienced or misinformed people. Proper nutrition may require supplementation, especially in birds of prey whose diet is fish based and who are fed frozen and thawed fish diets.

Hypovitaminosis A

Raptors are unable to convert carotenoid precursors into active vitamin A. In the wild, the prey's liver is the greatest source of vitamin A, so deficiencies are seen most often in birds fed exclusively meat diets without access to viscera. Vitamin A is indispensable in maintaining the integrity and function of epithelial tissues, and its deficiency is most often expressed as hyperkeratosis or metaplasia of squamous cell tissue. Oral, esophageal, and infraorbital gland hyperplasia, as well as syringeal, tracheal, bronchial and nasal, or lacrimal gland metaplasia account for the most obvious clinical signs. Hyperkeratotic oral plaques are frequently confused with lesions associated with oral trichomoniasis or candidiasis. Keratin accumulation may deform the infraorbital sinuses and conjunctival sacs, and may be mistaken for sinusitis or focal aspergillosis. Hyperkeratotic plugs in the trachea frequently cause inspiratory dyspnea. Visceral and articular gout associated with renal failure induced by hypovitaminosis A has been reported.

Adding liver, egg yolk, cod-liver oil, or commercial vitamin A supplements to the diet is usually sufficient to prevent and treat the problem. Liver levels of vitamin A in raptors should be between 9000 and 13,000 μg/g, so necropsy diagnostics can be performed if hepatic levels are measured.

Hypovitaminosis B

Diurnal raptors fed exclusively on day-old chicks, meat, or eviscerated prey, as well as piscivorous birds fed frozen fish diets that may be affected by thiaminase activity, tend to present neurological deficits; typically opisthotonos and ataxia, and may eventually develop axonal and neuronal degeneration. Clinical signs dramatically improve or disappear in response to parenteral thiamin supplementation. Dramatic improvement following thiamin supplement injection may lead to a diagnosis based on clinical response. Episodes of toxicity from insecticides, as well as viral, bacterial, or mycotic encephalitides may present with similar clinical signs and may obscure the diagnosis.

There are a few reports of riboflavin deficiency in raptors. The classic sign of "curled toe" seen in riboflavin-deficient domestic birds appears to be strongly suggestive of the same problem in birds of prey. Treatment with an injectable supplement at doses recommended for small mammals has reportedly led to dramatic improvement in a short time, and may be considered diagnostic.

Hypovitaminosis D and Mineral Imbalance

Raptors metabolize vitamin D_3 in the skin and excrete it through the uropygium or preen gland. Animals exposed to sunlight and fed balanced diets are able to ingest enough activated vitamin D_3 to meet their needs. Animals fed all meat diets or viscera, which have a poor calcium-to-phosphorous ratio (frequently the recommended ratio of 2:1 of calcium to phosphorous is reversed in unsupplemented viscera), may present with rickets when young or osteomalacia when subadult to adult. Diets high in fats tend to cause chronic vitamin D_3 deficiency by reducing the absorption of liposoluble vitamins from the intestine and by the saponification of calcium-containing compounds, which renders the mineral nonabsorbable at the enteric level.

Raptor chicks are extremely fast growing, sometimes achieving or exceeding adult weight in 20 days. Poor mineralization during this phase results in rickets, which are expressed as pathological fractures of debilitated long bones, as well as their deformity during growth. Onset of rickets may be as short as 5 days, whereas osteomalacia in adults may take months to develop and express itself.

Severe and rapid declines in calcium levels in the blood may lead to clinical hypocalcemic tetany and seizures. Spontaneous fractures of poorly mineralized bones in growing birds have been associated with seizures during hypocalcemic tetany. Administration of parenteral calcium borogluconate by slow intravenous or subcutaneous routes may alleviate hypocalcemic signs, but immediate and definitive diet correction is the only way to reverse the underlying pathology. Bone deformities are permanent and frequently impede full functional recovery.

Hypovitaminosis E and Selenium Deficiencies

Hypovitaminosis E and selenium deficiencies are extremely rare, but may occur in raptors fed exclusively meat. Diagnosis is usually made at necropsy, by histopathology, where characteristic lesions of skeletal muscle and hyaline degeneration are noted. The lesions are usually indicative of nutritional myopathy. The myocardium, fat, and liver are usually not affected. At present, no antemortem diagnostics or treatment are available. A complete and properly balanced diet is the only preventive.

PARASITISM

Endoparasites are frequently observed in wild raptors. Infestation by trematodes is frequent but generally considered of little clinical significance. Cestodes are generally innocuous, but massive infestations have been known to cause intestinal obstruction. Proglottids may be observed in feces or on feathers directly, and their detection is sufficient to establish a diagnosis. Nematodes of the genera *Prorrocaecum* and *Ascaridia* may be diagnosed through their eggs in a fecal flotation and may be found in a wide variety of species of raptor. Doses of 0.2 mg of ivermectin given orally are generally effective.

Birds of the genus *Falco,* notably prairie falcons (*Falco mexicanus*) and peregrine falcons (*F. peregrinus*) may develop severe infestations by *Serratospiculum amaculatta.* The nematodes are found in the air sacs, many times in high numbers. Severe infestation may lead to dyspnea, emaciation, and death. Occasionally, parasites breaking through the air sacs cause severe coelomitis in the host. Doses of up to 50 mg/kg of fenbendazole administered orally, repeated in 14 days, have been reported to be effective. *Capillaria, Syngamus, Physaloptera, Thelazia,* and *Habronema* are all infrequently seen, but occasionally reported. Treatment as mentioned above is effective.

Myasis is an important consideration when examining raptor nestlings. Ears and mucous membranes should be carefully examined for the presence of fly maggots of the genera *Calliphora* and *Callitroga.* Careful physical removal is necessary to resolve infestation. Hippoboscid flies, even though not directly pathogenic, may transmit hematogenous parasites. The presence of feather lice, though a nuisance, is rarely considered a problem of clinical significance. Treatment with topical insecticides safe in other birds, generally pyrethroids, is recommended.

Infection by *Trichomonas gallinae* is frequent in raptors fed pigeons, but may also be seen in birds of prey not eating columbiforms. It may be serious to lethal in immunosuppressed birds. Lesions produced are characterized by yellowish caseated plaques in the oral mucosa and by the associated anorexia and dyspnea they produce. Carnidazole at a dose of 20 mg/kg orally, once a day for 2 days, is extremely effective.

Haemoproteus, Leucocytozoon, and *Plasmodium* spp. are frequently observed in blood smears of raptors.

Plasmodium infestations may lead to severe depression, listlessness, and death. Chloroquine at an initial dose of 10 mg/kg orally, followed by 5 mg/kg at 6, 24, and 48 hours, along with primaquine at a dose of 0.3 mg/kg orally every 24 hours for 7 days, may be given to treat all sporozoan parasites. Combined therapy seems to be most effective in cases of severe *Haemoproteus* or *Leucocytozoon* infections. *Sarcocystis* is frequently seen in the muscle mass of raptors. There are a few reports of progressive paresis, neurological deficits, and death associated with the presence of the parasite in the central nervous system

INFECTIOUS DISEASES

Herpesvirus

Falcon herpesvirus infection is characterized by hepatitis and splenitis. The virus is presumed to be transmitted by pigeons consumed as food. Infection in falcons causes up to 100% mortality and is characterized by intranuclear inclusion bodies found by histopathology. At present no treatment seems to be effective. Prevention is limited to avoiding pigeons as a food source or feeding birds from known, closed, and tested pigeon colonies.

Owl herpesvirus and eagle herpesvirus cause signs in these birds similar to those seen in falcons. Infection by herpesvirus may resemble salmonellosis on a macroscopic level, so culture and histopathology are recommended in suspect cases.

Avian Tuberculosis

Infection by *Mycobacterium avium* is the most common bacterial disease of raptors. The disease may be chronic and is usually expressed as a slowly progressive emaciation ending in death. Yellowish caseated granulomas measuring 1–3 mm are found primarily in the liver, but may also be observed in the spleen, lung, air sacs, gastrointestinal tract, bone marrow, and skin.

Infected prey is the origin of infection, and at present treatment is ineffective and ill advised because of its possible zoonotic potential toward immunosuppressed humans. Diagnosis is based on fecal culture, acid-fast fecal staining, or hepatic biopsy histopathology.

Bumblefoot

Pododermatitis, primarily affecting the metatarsal pads of raptors, is a common problem of birds kept in captivity. Causes of the condition are multiple, but generally include trauma caused by smooth or inadequate perches, large or heavy birds, talon-inflicted punctures of the pads during gripping, foreign bodies, uneven wear of the skin of the pads, and hyperkeratosis of the pad. Hypovitaminosis A has occasionally been associated with chronic conditions.

Staphylococcus aureus is generally cultured from infected pads. Lesions have been graded according to severity, with pathogenicity varying from simple and minor epithelial erosion of the pad to severe and ascending bacterial tendosynovitis. Chronic and severe infections may lead to bacterial endocarditis.

Simple uninfected erosions of the feet may be treated by correcting inadequate perching materials and design and by the use of light, padded bandaging. Chronic and infected pads may require careful surgical debridement of abscessed tissue before antibiotic therapy can be effective. Once one foot is affected, the opposite foot becomes eroded and infected because of increased weight bearing over long periods of time. Topical localized treatment with combined penicillin-based antibiotics and dimethyl sulfoxide (DMSO) may alleviate localized processes with minor infections.

Miscellaneous Infections

Other infectious diseases that are common to many species of birds include pasteurellosis, candidiasis, and aspergillosis.

TRAUMA

Trauma is the most common reason wild raptors are presented for treatment. Cephalic trauma is frequently seen in wild birds, and neurologic sequelae may impair full functional recovery. Ocular trauma may lead to corneal laceration, lenticular proptosis, and partial or total retinal detachment. The large size of the eye in comparison with the proportionate size of the skull predisposes raptors to trauma and intraocular hemorrhage. Treatment with antibiotics and parenteral anti-inflammatory drugs may be effective, but vision is difficult to evaluate once the animal has recovered. Vision in a single eye may impair the ability of diurnal raptors to hunt prey. Their release following successful rehabilitation should be evaluated carefully.

Fractures of the long bones are also a common occurrence. The techniques for repair have been extensively described, but proper triage and prompt surgical intervention increase the likelihood of a successful repair. At present, external fixation, or the combined use of external and internal fixation techniques, seems to be effective. New lightweight plastic materials have made external devices effective, strong, and relatively easy to apply. Postoperative care must include a structured and consistent exercise program for full return to function before release.

REINTRODUCTION AND MONITORING

Raptor rehabilitation has provided numerous means of monitoring wild or released birds of prey. Several techniques have been tried over the years, and the experience gained has benefited programs that study their biology and migration. Aggressive trapping, banding, and release programs over the last decade have produced excellent information regarding transcontinental migratory routes as well as nesting habits. Wildlife monitoring and understanding of population dynamics remain key to the success of raptors in the wild.

REFERENCES

1. Ackermann, J.; and Redig, P. 1997. Surgical repair of elbow luxation in raptors. Journal of Avian Medicine and Surgery 11(4):247–254.
2. Aguilar, R.F.; and Redig, P.T. 1997. Diagnosis and treatment of avian aspergillosis. In J.D. Bonagura, ed., Kirk's Current Veterinary Therapy, Vol. 7. Small Animal Practice. Philadelphia. W.B. Saunders, pp. 1294–1299.
3. Aguilar, R.F.; Smith, V.E.; Ogburn, P.; and Redig, P.T. 1995. Arrhythmias associated with isoflurane anesthesia in bald eagles (*Haliaeetus leucocephalus*). Journal of Zoo and Wildlife Medicine 26(4):508–516.
4. Aguilar, R.F.; Johnston, G.R.; Robinson, T.; and Redig, P.T. 1993. Osseous-venous and central circulatory transit times of technetium-99m pertechnetate in anesthetized raptors. Journal of Zoo and Wildlife Medicine 24(4):488–497.
5. Aguilar, R.F.; Stiles, J.; Bistner, S.I.; and Redig, P.T. 1993. Surgical synechiotomy in an adult bald eagle (*Haliaeetus leucocephalus*). Journal of Zoo and Wildlife Medicine 24(1):63–67.
6. Aguilar, R.F.; Shaw, D.P.; Dubey, J.P.; and Redig, P.T. 1991. Sarcocystis-associated encephalitis in an immature northern Goshawk (*Accipiter gentilis atricapillus*). Journal of Zoo and Wildlife Medicine 22(4):466–469.
7. Baker, D.G.; Morishita, T.Y.; Bartlett, J.L.; and Brooks, D.L. 1996. Coprologic survey of internal parasites of northern California raptors. Journal of Zoo and Wildlife Medicine 27(3):358–363.
8. Battisti, A.; di Guardo, G.; Agrimi, U.; Bozzano, A.I.; and Di Guardo, G. 1998. Embryonic an neonatal mortality from salmonellosis in captive bred raptors. Journal of Wildlife Diseases 34(1):64–72.
9. Blus, L.J. 1996. Effects of pesticides on owls in North America. Journal of Raptor Research 30(4):198–206.
10. Buffin, D. 1997. Action to halt hawk deaths. Pesticide News 35:6.
11. Clark, F.D. 1986. Mycobacteriosis in a red-tailed hawk (*Buteo jamaicensis*). Southwest Veterinary 37:200–201.
12. Cooper, J.E. 1968. Tuberculosis in birds of prey. Veterinary Record 100:61.
13. Cooper, J.E. 1993. Avian pox in birds of prey (order Falconiformes). Veterinary Record 132:343–345.
14. Cray, C.; and Tatum, L.M. 1998. Applications of protein electrophoresis in avian diagnostics. Journal of Avian Medicine and Surgery 12(1):4–10.
15. Deem, S.L.; Terrell, S.P.; and Forrester, D.J. 1998. A retrospective study of morbidity and mortality of raptors in Florida: 1988–1994. Journal of Zoo and Wildlife Medicine 29(2):160–164.
16. Elliott, J.E.; and Norstrom, R.J. 1998. Chlorinated hydrocarbon contaminants and productivity of bald eagle populations on the pacific coast of Canada. Environmental Toxicology and Chemistry 17(6):1142–1153.
17. Fairbrother, A.; Locke, L.N.; and Hoff, G.L. 1996. Non-Infectious Diseases of Wildlife, 2nd Ed. London, Manson Publishing.
18. Forbes, N.A. 1997. PTFE toxicity in birds. Veterinary Record 140(19):512.
19. Forbes, N.A. 1996. Differential diagnosis and treatment of fitting in raptors with particular attention to the previously unreported condition of stress induced hyperglycemia in northern goshawks (*Accipiter gentilis*). Israel Journal of Veterinary Medicine 51(3–4):183–188.
20. Forbes, N.A. 1996. Non-ocular conditions of the head of raptors. Israel Journal of Veterinary Medicine 51(3–4):177–182.
21. Forbes, N.A.; and Simpson, G.N. 1997. A review of viruses affecting raptors. Veterinary Record 141(5):123–126.
22. Foster, G.W.; Morrison, J.L.; Hartless, C.S.; and Forrester, D.J. 1998. Haemoproteus tinnunculi in crested caracaras (*Caracara plancus audubonii*) from southcentral Florida. Journal of Raptor Research 32(2):159–162.
23. Fowler, M.E.; Schulz, T.; Ardans, A.; Reynolds, B.; and Behymer, D. 1990. Chlamydiosis in captive raptors. Avian Disease 34:657–662.
24. Garner, M.M. 1989. Bumblefoot associated with pox virus in a wild golden eagle (*Aquila chrysaetos*). Companion Animal Practice 19:17–20.
25. Gentz, E.J. 1996. *Fusobacterium necrophorum* associated with bumblefoot in a wild great horned owl. Journal of Avian Medicine and Surgery 10(4):258–261.
26. Graham, J.E.; Larocca, R.D.; and McLaughlin, S.A. 1999. Implantation of intraocular silicone prosthesis in a great horned owl (*Bubo virginianus*). Journal of Avian Medicine and Surgery 13(2):98–103.
27. Harper, F.D.W.; Humphreys, P.N.; Beynon, P.H.; Forbes, N.A.; and Harcourt-Brown, N.H. Eds. 1996. Manual of Raptors, Pigeons and Waterfowl. Cheltenham, U.K., British Small Animal Veterinary Association.
28. Hawks, S.; and Klann, R. 1997. Helminth ova recovered from raptors admitted for rehabilitation. Journal of the Iowa Academy of Science 104(2):47–49.
29. Heidenreich, M. 1997. Birds of Prey: Medicine and Management. Oxford, Blackwell Science, pp. 107–113, 120–125.

30. Jarman, W.M.; Burns, S.A.; Bacon, C.E.; Rechtin, J.; DeBenedetti, S.; Linthicum, J.L.; and Walton, B.J. 1996. High levels of HCB and DDE associated with reproductive failure in prairie falcons (*Falco mexicanus*) from California. Bulletin of Environmental Contamination and Toxicology 57(1):8–15.
31. Kaleta, E.F. 1990. Herpesviruses of birds: A review. Avian Pathology 19:193–211.
32. Keith, J.O.; and Bruggers, R.L. 1998. Review of hazards to raptors from pest control in Sahelian Africa. Journal of Raptor Research 32(2):151–158.
33. Kinsella, J.M.; Cole, R.A.; Forrester, D.J.; and Roderick, C.L. 1996. Helminth parasites of the osprey, *Pandion haliaetus,* in North America. Journal of the Helminthological Society of Washington 63(2):262–265.
34. Kramer, J.L.; and Redig, P.T. 1997. Sixteen years of lead poisoning in eagles, 1980–95: An epizootiologic view. Journal of Raptor Research 31(4):327–332.
35. Lindsay, D.S.; and Blagburn, B.L. 1999. Prevalence of encysted apicomplexes in muscles of raptors. Veterinary Parasitology 80(4):341–344.
36. Mama, K.R.; Phillips, L.G.; and Pascoe, P.J. 1996. Use of propofol for induction and maintenance of anesthesia in a barn owl (*Tyto alba*) undergoing tracheal resection. Journal of Zoo and Wildlife Medicine 27(3):397–401.
37. Mirande, L.A.; Howerth, E.W.; and Poston, R.P. 1992. Chlamydiosis in a red-tailed hawk (*Buteo jamaicensis*). Journal of Wildlife Diseases 28:284–287.
38. Morishita, T.Y.; Aye, P.P.; and Brooks, D.L. 1997. A survey of diseases of raptorial birds. Journal of Avian Medicine and Surgery 11(2):77–92.
39. Morishita, T.Y.; Fullerton, A.T.; Lowenstine, L.J.; Gardner, I.A.; and Brooks, D.L. 1998. Morbidity and mortality in free living raptorial birds of Northern California: A retrospective study, 1983–1994. Journal of Avian Medicine and Surgery 12(2):78–81.
40. Morishita, T.Y.; Lowenstine, L.J.; Hirsh, D.C.; and Brooks, D.L. 1997. Lesions associated with *Pasteurella multocida* infection in raptors. Avian Diseases 41(1):203–213.
41. Morishita, T.Y.; Lowenstine, L.J.; Hirsh, D.C.; and Brooks, D.L. 1996. *Pasteurella multocida* in raptors: Prevalence and characterization. Avian Diseases 40(4):908–918.
42. Morishita, T.Y.; McFadzen, M.E.; Mohan, R.; Aye, P.P.; and Brooks, D.L. 1998. Serologic survey of free living nestling prairie falcons (*Falco mexicanus*) for selected pathogens. Journal of Zoo and Wildlife Medicine 29(1):18–20.
43. Mozos, E.; Hervas, J.; Moyano, T.; Diaz, J.; and Gomez-Villa-Mandos, J.C. 1994. Inclusion body disease in a peregrine falcon (*Falco peregrinus*): Histological and ultrastructural study. Avian Pathology 23:175–181.
44. Nayar, J.K.; Knight, J.W.; and Telford, S.R., Jr. 1998. Vector ability of mosquitoes for isolates of *Plasmodium elongatum* from raptors in Florida. Journal of Parasitology 84(3):542–546.
45. Nicholson, M.G.G. 1996. The raptor as a patient. Irish Veterinary Journal 49(2):87–90.
46. Okoh, A.E.J. 1979. Newcastle disease in falcons. Journal of Wildlife Diseases 15:479–480.
47. Poveda, J.B.; Carranza, J.; Miranda, A.; Garrido, A.; Hermoso, M.; Hernandez, A.; and Domenech, J. 1990. An epizootiological study of avian mycoplasmas in southern Spain. Avian Pathology 19:627–633.
48. Poveda, J.B.; Giebel, J.; Flossdorf, J.; Meier, J; and Kirchhoff, H. 1994. *Mycoplasma buteonis* sp. nov., *Mycoplasma falconis sp. nov., and Mycoplasma gypis* sp. nov., three species from birds of prey. International Journal of Systemic Bacteriology 44:94–98.
49. Poveda, J.B.; Giebel, J.; Kiechoff, H.; and Fernandez, A. 1990. Isolation of mycoplasmas from a buzzard, falcons and vultures. Avian Pathology 19:779–783.
50. Potgieter, L.N.D.; Kocan, A.A.; and Kocan, K.M. 1979. Isolation of a herpesvirus from an American kestrel with inclusion body disease. Journal of Wildlife Diseases 15:143–149.
51. Ramis, A.; Majo, N.; Pumarola, M.; Fondevila, D.; and Ferrer, L. 1994. Herpesvirus hepatitis in two eagles in Spain. Avian Diseases 38:197–200.
52. Redig, P.T.; Cooper, J.E.; Remple, J.D.; and Hunter, D.B. 1993. Raptor Biomedicine. Minneapolis, University of Minnesota Press.
53. Redig, P.T.; Marx, K.L.; and Roston, M.A. 1998. Methods for management of forelimb fractures. In Proceedings of the 19th Annual Conference on Avian Medicine and Surgery Mid-Atlantic States Association of Avian Veterinarians, Lancaster, Pennsylvania. Association of Avian Veterinarians, pp. 26–28.
54. Redig, P.T. 1993. Medical Management of Birds of Prey, 3rd Ed. St. Paul, Minnesota, The Raptor Center at the University of Minnesota.
55. Ritchie, B.W.; Harrison, G.J.; and Harrison, L.R. 1994. Avian medicine: Principles and application. Lakeworth, Florida, Wingers Publishing.
56. Rodriguez-Lainz, A.J.; Hird, D.W.; Kass, P.H.; and Brooks, D.L. 1997. Incidence and risk factors for bumblefoot (pododermatitis) in rehabilitated raptors. Preventive Veterinary Medicine 31(3–4):175–184.
57. Samour, J.H.; and Cooper, J.E. 1993. Avian pox in birds of prey (order Falconiformes). Veterinary Record 132:343–345.
58. Shannon, L.M.; Poulton, J.L.; Emmons, R.W.; Woodie, J.D.; and Fowler, M.E. 1988. Serological survey for rabies antibodies in raptors from California. Journal of Wildlife Diseases 24:264–267.
59. Stein, R.W.; Yamamoto, J.T.; Fry, D.M.; and Wilson, B.W. 1998. Comparative hematology and plasma biochemistry of red-tailed hawks and American kestrels wintering in California. Journal of Raptor Research 32(2):163–169.
60. Sykes, G.P. 1982. Tuberculosis in a red-tailed hawk (*Buteo jamaicensis*). Journal of Wildlife Diseases 18:495–499.
61. Taft, S.J.; Rosenfield, R.N.; and Evans, D.L. 1996. Hematozoa in autumnal migrant raptors from the

Hawk Ridge Nature Reserve, Duluth, Minnesota. Journal of the Helminthological Society of Washington 63(1):141–143.
62. Ward, F.P. 1971. Inclusion body hepatitis in a prairie falcon. Journal of Wildlife Diseases 7:120–124.
63. Wernery, U.; Remple, J.D.; Neumann, U.; Alexander, D.J.; Manvell, R.J.; and Kaaden, O.R. 1992. Avian paramyxovirus serotype I (Newcastle disease virus) infectious in falcons. Journal of Veterinary Medicine 39:153–158.
64. Wheeldon, E.B.; Sedgwick, C.J.; and Shultz, T.A. 1993. Epornitic of avian pox in a raptor rehabilitation center. Journal of American Veterinary Medical Association 187:1202.
65. Wheler, C.L. 1993. Herpesvirus disease in raptors: A review of the literature. In J.E Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Keighley, West Yorkshire, England, Circon Publishing, pp. 103–107.

13
Order Strigiformes (Owls)

Murray E. Fowler

BIOLOGY, MEDICINE, AND SURGERY

INTRODUCTION

Owls inhabit every continent of the world except the Antarctic.[1] Owls are distinctive, such that most people recognize them by their huge head, large, forward-looking eyes, concentrated expression, chunky body, and sober habit. Children the world over hear about them in legends and read about them in storybooks. The owl signifies solemn wisdom.[1] Owls are a major factor in controlling rodent populations.

BIOLOGY

Owls are generally nocturnal or crepuscular, but the burrowing owl *Athene (Speotyto) cunicularis* is diurnal. The soft feathers of owls allow soundless flight, which avoids alerting prey being approached. Owls have excellent eyesight, with the eyeball enclosed within a sclerotic ring (bone). An arrangement of feathers on the face form a parabola (facial disk) that concentrates sound to the external ear canal, allowing some species to locate and capture prey in total darkness.

Taxonomy

Eleven to 13 genera and 29 species or subspecies of owls (order Strigiformes, families Strigidae and Tytonidae) inhabit South America.[8,11] Table 13.1 lists selected owl species with broad distribution in Central and South America, along with information on their general diets. Many of the broadly distributed species of South American owls are also found in North America, and the barn owl *Tyto alba,* is widely distributed throughout the world.

Habitat Requirements

Owls occupy a variety of habitats, but most will be found hunting near forests or woodlands. The burrowing owl is a ground nester, preferring open fields to see small mammals and insects clearly.

Food Habits

Small owls consume small mammals, reptiles, amphibians, insects, and other invertebrates. Large owls eat rodents, lagomorphs, and some small birds.

TABLE 13.1. South American owls with broad distribution—biological data (Class: Aves; order: Strigiformes; families: Tytonidae and Strigidae)

Scientific Name	Common Name (English)	Common Name (Spanish)	Common Name (Portuguese)	Distribution	Diet
Tyto alba	Barn owl	Lechuza de campanario	Suindara, Coruja-das-igrejas	Argentina, Bolivia, Paraguay, Uruguay, eastern and central Brazil, Chile	Various vertebrates, especially small nocturnal rodents, shrews, small birds
Bubo virginianus	Great-horned owl	Ñacurutú	Murucutu, Mocho-orelhudo, Jacurutu	Equador, Peru, Bolivia, Chile, Brazil	Various vertebrates, including hares and rabbits
Ciccaba virgata	Mottled owl	Lechuza estriada	Coruja-de-cauda-longa	Brazil	Small rodents, birds, reptiles, insects
Athene (Speotyto) cunicularia	Burrowing owl	Lechucita pampa	Coruja-buraqueira	Brazil	Insects, especially large beetles, small rodents, frogs, small birds
Asio stygius	Stygian owl	Lechuzón negruzco	Mocho-do-diablo	Argentina, Paraguay, Brazil	Various vertebrates
Asio flammeus	Short-eared owl	Lechuzón campestre		Argentina, Bolivia, Paraguay, Uruguay, Brazil, with the exception of the eastern Amazon basin	Various vertebrates
Glaucidium brasilianum	Ferruginous pygmy owl	Caburé grande	Caburezinho-do-sol	Argentina, Bolivia, Paraguay, Uruguay, Brazil	Insects, small mammals, amphibians
Otus choliba	Tropical screech owl	Alicuco grande	Cabure comum	Argentina, Bolivia, Paraguay, Uruguay, Brazil	Insects, small mammals, amphibians

Exploitation

Owls do not have the unfortunate reputation of hawks and eagles for preying on domestic poultry, but they are still shot by ignorant people in the name of sport. Their nocturnal behavior protects them from most hunters, but a few species hunt during the early evening and are flying when hunters are about.

Studies on Free-Ranging Populations

Owls have been studied intensively both in the wild and captivity. Interested readers may consult the references provided. The primary concern for conservation is the loss of habitat.[1,11]

MANAGEMENT OF OWLS IN CAPTIVITY

Housing

No single enclosure is suitable for all species of owls. The type and size of the enclosure should be based on the species of owl and whether or not the owl is being held for medical purposes; for instance, is the owl being exhibited in a zoo? Is breeding contemplated? The criteria for an appropriate enclosure are as follows:

The cage for medical and surgical treatment should be large enough for the bird to stand upright and stretch its wings. It should contain a perch suitable for the species and be constructed of material that is easily cleaned and sanitized (fiberglass, metal, wood covered with epoxy paint). It should also have a curtain (paper or cloth) to give privacy and darkness.

Zoo housing enclosures should provide space for the bird to fly, even if only for a short distance. In most climates it is not necessary to provide indoor caging, but the owl should be able to find shade and escape from rain, snow, and heavy winds. The walls of the enclosure may be constructed of wire mesh, wood, fiberglass, gunite concrete, high-tensile wire, glass, or various combinations of these. A variety of perching sites should be provided at various heights within the enclosure.

When considering enclosures for breeding, indoor flight cages should have space necessary for the birds to fly and have exposure to sunlight (skylights, windows). They should provide privacy from people and other birds and provide nesting sites and nesting material suitable for the species (platforms, hollow tree trunks). Perching sites suitable for the species should be provided, as well.

Once medical and surgical aftercare are completed, the bird should be provided with space sufficient to exercise and regain muscle strength and agility. Ultimately, the owl should be placed in a large flight cage to fine tune muscle coordination and flexibility of articulations. Owls hospitalized before fledglings must be taught to capture prey. In the raptor center at the University of California (Davis, California, USA), this is done by constructing an open-topped rodent pen (2×6 m) in the center of a flight cage (20×30 m). The walls of the rodent pen are 0.5 m high and constructed of smooth metal to prevent the rodents from climbing out. Live mice are placed in the rodent pen. The owl to be taught to hunt is placed in the flight cage along with an owl of the same species that is an experienced hunter.

Feeding

Owls maintained in captivity are fed a variety of feeds in accordance with their natural prey, if possible. Insectivores are fed mealworms *Tenebrio* spp. and crickets *Acheta domestica*. Many owls are fed whole laboratory rodents or day-old chicks, *Gallus domesticus*. The rodents and chicks are killed humanely and stored frozen until fed. Chicks and immature mice and rats are calcium deficient because the bones are primarily cartilage at this early stage of life. Supplementary calcium must be supplied. All owls should have exposure to sunlight or be fed a supplement containing vitamin D_3. Some owls will eat only live prey, and birds destined for release soon should be fed live prey to accustom the bird to capturing its own prey.

Additional food items fed to captive owls include, ground or chunk red meat, chicken necks, meat mixes, and fish. Great care must be exercised to balance the diet for energy, calcium, phosphorous, and vitamin D_3. Chicken necks provide sufficient calcium, but because over half of the chicken neck is ash (mineral), there is insufficient meat to provide energy and protein.

Owls may become adapted to eating commercial carnivore diets, which are balanced for calcium and phosphorous. In North America, a commercial raptor diet is available. Unfortunately, the diet is supplied only in frozen plastic packets (2.25 kg [5 lbs]), so having to purchase a large quantity may create freezer storage problems.

RESTRAINT, ANESTHESIA, AND SURGERY

Restraint and Handling[6]

The talons are the primary means of obtaining food and are also used for defense. Owls in small cages may be captured with an appropriate-sized net. If the bird is on the ground, a towel or small blanket may be thrown over it, and then it is grasped from behind. Once the owl is in hand, it may be controlled by grasping the legs and head. Although gloves are desirable for initial capture, they should be removed when holding the bird to better sense the degree of pressure being applied. It is advisable to wear heavy leather gloves when working with large owls, even though gloves do not afford full protection.

To grasp a bird standing upright in a cardboard box, place both hands on the back over the wings and legs. Press the owl to the floor of the box, and direct the fingers of both hands around and underneath the body to grasp the legs and control the feet before lifting the owl.

Owls often throw themselves on their backs and direct a flailing set of formidable talons toward anyone attempting to pick them up. A great-horned owl can drive a talon completely through the heaviest leather glove, so this bird should be approached with caution. Dangling an empty glove above the owl invites attack, and while the talons are attached to the glove, the legs may be grabbed with the other hand. Young owls may sometimes be captured from this prone position by presenting a small towel or piece of cloth for the talons to grasp.

If an owl impales a handler with a talon during the capturing process, the bird should be released and allowed to move away. An impaled talon may be released manually by straightening the leg at the tarsal-metatarsal articulation, which relaxes the tendon-tightening mechanism that operates to maintain the grip when the leg is flexed.

Small to medium-sized owls may be effectively restrained after initial capture by placing them in a cotton stockinette tube or a nylon hose. This tube may also be used as an initial drape for surgery. A hole my be cut in the stockinette to withdraw a wing or a limb. Nylon hose will retain body heat, thus the handler should be cognizant of excessive ambient heat.

Chemical restraint should be used when painful procedures are to be performed. Numerous drugs and drug combinations have been employed successfully. A mixture of ketamine and xylazine at 10 mg/kg ketamine and 2 mg/kg (xylazine) provides good immobilization for carrying out most clinical procedures, such as radiography, endoscopy, and physical examination.[6] Dosages of 15 mg/kg of ketamine and 0.15 mg/kg xylazine have also been recommended. Tiletamine/zolazepam (Telazol, Zolotil) 10 mg/kg has also been used.[7]

Anesthesia

Minor surgical procedures may be performed using chemical immobilization. The owl should be placed under general anesthesia for orthopedic surgery and procedures that may require considerable time. The most satisfactory anesthetic agent is isoflurane, but both fluothane and methoxyflurane have been used.[5]

Surgery

Space does not permit an in-depth discussion of surgical disorders. All the various avian orthopedic procedures have been applied to owl surgery. A recent innovative approach has been the use of a polypropylene rod for insertion into the intramedullary cavity of the fractured long bone.[4] Then liquid methyl methacrylate (bone cement) is injected around the pin to fill the space between the rod and the cortex. The advantages of this procedure are rigid internal stability, ability to stabilize fractures near joints, and rapid return to functional use of the limb. This procedure should not be used on the pneumatized radius or the tarsometatarsus, which has no marrow cavity.

The surgical protocol for owl orthopedics is the same as for other raptors. A few basic principles should be followed.

1. If the bird is to be rehabilitated to the free-ranging state, fracture healing must be as perfect as possible.
2. Fractures near an articulation have a poor prognosis because frequently the articulation sustains damage at the same time the fracture occurs. Often there is no initial radiographic evidence of trauma, but subsequent radiographs chronicle the development of arthritic changes.
3. Feathers should not be clipped to provide a surgical field and only the minimum number of feathers should be plucked. Avoid pulling primary and secondary flight feathers, because these may not replace themselves until the next molt, and in some cases for years.
4. The areas contiguous to the surgical field should be wrapped with aluminum foil and covered with a stockinette. Ultimately, paper or cloth drapes may be used to establish an antiseptic surgical field.
5. The bird should be placed on a circulating water heating pad if the surgery requires more than one-half hour.

DIAGNOSIS

Clinical Examination

Owls are generally not difficult to examine. The same protocol as used for all birds should be followed. Owls have a tendency to bite as a defense mechanism, so the head should always be restrained. Make certain that the mouth is opened for inspection. A number of local and systemic conditions produce oral lesions, including trichomoniasis, aspergillosis, candidiasis capillariasis, hypovitaminosis A, and pox. Blood may be collected from the medial tarsal vein or the brachial vein as it crosses the elbow. The right jugular vein is also accessible and is especially useful when a large quantity of blood is required. The toenail of an owl should not be clipped to obtain a blood sample. Sharp talons are necessary to obtain food. Hematology and blood chemistry values are known for a number of the more common species of owls, but space precludes listing the values here. The literature may be consulted to obtain these. Special procedures, such as radiography, ultrasonography, and endoscopy, are performed routinely, as in other birds.

DISEASES

Diseases in Free-Ranging Populations

Most disease studies have been concerned with owls in captivity. The diseases seen in diagnostic laboratories dealing with injured and ill birds coming from the wild include hepatosplenitis, trichomoniasis, avian cholera, miscellaneous bacterial infections, and pesticide toxicity. Free-ranging owls have the usual array of external and internal parasites, most of which live in harmony with their host. Many owls coming directly from the wild have lice (order Mallophaga), which are much larger than mammalian lice (2–10 mm).

Owls are at the top of the food chain, so they may ingest prey species that have accumulated a variety of chlorinated hydrocarbon, carbamate, or organophosphate pesticides. Prey species suffering from exposure to rodenticides may be less able to evade owls.

NONINFECTIOUS DISEASES IN FREE-RANGING OWLS Trauma is a major cause of mortality in free-ranging owls. They may be shot, struck by automobiles, injured in fights with other owls, or injured by their prey. Veterinarians are frequently called upon to provide care for injured owls. Barn owls in the author's area have become scavengers of road-killed rodents and other small mammals. The birds often underestimate the speed of approaching vehicles on motorways and do not fly off fast enough to avoid being struck. Refer to the noninfectious disease section for a discussion of details of care and management.

High mortality of juvenile owls occurs when inclement weather prevents birds from foraging or during cyclic rodent population decline. Some birds simply do not learn sufficient predator skills to compete when food is scarce. In any stable wild animal population as many animals must die each year as are born or hatched. This is normal mortality associated with maximum utilization of food resources and survival of the fittest.

ELECTROCUTION Owls are not as likely to be electrocuted as are the large hawks or eagles, but large owls may alight on an electrical transmission line and with outstretched wings contact a second line to complete an electrical circuit. The electrical shock may kill the owl or produce primary signs as follows: skin burns; necrotic wings, mouth, beak, legs; paralysis; shock; and apathy. Secondary effects include head and limb trauma from the fall, mummification of the extremities, and renal gout. The bird will often be found beneath an electrical transmission line, unable to fly, and brought to a veterinarian. The bird may be presented as a trauma victim, as the fall from the wire perch may fracture wing bones or other bones. Depending on the length of time that has elapsed between the injury and being found, the bird may be in an advanced state of dehydration and or starvation.

The prognosis for most electrocution victims is unfavorable. If the bird is paralyzed, has mummification of the limbs, or extensive burns, it should be euthanized. Otherwise, the owl, should be hydrated and dexamethasone administered at 2 mg/kg, along with wound management.

Infectious Diseases in Captivity[2,3,10]

FUNGAL DISEASES The major fungal disease seen in captive owls is aspergillosis (see Table 13.2). The spores of *Aspergillus fumigatus* are ubiquitous in the environment. The disease is generally found in birds that are immunodeficient through stress. Aspergillosis is frequently a secondary complication of a primary condition. Oral candidiasis may be observed in owls maintained in unsanitary conditions or in malnourished birds.

BACTERIAL DISEASES Birds of prey are subject to an array of opportunistic bacterial infections and are also exposed to the organisms that may be present in their prey. Usually owls take only live prey, but as already stated, the barn owl in certain locations has become adapted to using road-killed birds and small mammals. Captive owls may contract tuberculosis, coliform septicemia (*E. coli*), salmonellosis, or avian cholera (*Pasteurella multicida)*. A few selected infections are listed in Table 13.2.

VIRAL DISEASES Viral diseases reported in South American owl species include herpesviruses (hepatosplenitis and Marek's disease) and rabies (in the great-horned owl).[2,3,10]

TABLE 13.2. Infectious diseases—owls

Disease (English)	Disease (Spanish)	Disease (Portuguese)	Etiology	Signs	Diagnosis	Management
Infectious hepatosplenitis			Owl herpesvirus, Strigid HV 1	Sudden death, anorexia, depression, inability to perch, diarrhea	Intranuclear inclusion bodies in hepatocytes and spleen at necropsy	Isolation of sick birds, insect control.
Tuberculosis	Tuberculosis	Tuberculose	*Mycobacterium avium/ Mycobacterium intracellulare*	Emaciation, diarrhea	Granulomas (3–25 mm) in lungs and other viscera. Acid-fast rods from impression smears. Culture, histopathology	Difficult, sanitation, quarantine. Treatment has been unsuccessful.
Salmonellosis	Salmonelosis	Salmonelose	*Salmonella* spp.	Diarrhea, septicemia	Enteritis, culture	Sanitation, antibiotics, fluids
Colibacillosis	Colibacilosis	Colibacilose	*Escherichia coli*	Septicemia	Culture	Sanitation, antibiotics
Pseudomoniasis	Pseudomoniosis	Pseudomonose	*Pseudomonas* spp.	Variable depending on the organs involved. General septicemia.	Culture of blood, and at necropsy from the liver, spleen and lung.	Antibiotics based on sensitivity
Infectious pododermatitis (Bumblefoot)			*Staphylococcus aureus,* miscellaneous gram-negative rods	Swelling and ulceration of foot pads, difficulty in perching	Radiography, culture, and sensitivity	Provide suitable perches for the species exhibited, antibiotics, surgery, intensive wound care.
Velogenic viscerotropic Newcastle disease	Enfermedad de Newcastle	Doença da Newcastle	Paramyxovirus	Central nervous system effects, tremors, paresis, paralysis	History, signs, viral culture, serology	Quarantine, sanitation
Aspergillosis	Aspergilosis	Aspergilose	*Aspergillus fumigatus*	Dyspnea, exercise intolerance, weakness, depression	Tracheal culture, endoscopic examination, radiography, serology	No therapeutic regimen has been found to be effective particularly in advanced cases. Aspergillosis is most common in immunocompromised individuals, so minimizing stress is important. Avoid exposure to inhalation of decomposing organic material.
Candidiasis	Moneliasis	Moneliose	*Candida albicans*	Anorexia, diarrhea, diptheritic plaques in mouth	Lesions, direct smear, culture	Imidazole antifungal agents, such as itraconizole, fluconizole

PARASITIC DISEASES[12] Parasites are listed in Table 13.3.

Coccidial parasites are commonly observed on fecal flotations and include the genera, *Caryospora, Eimeria, Frenkelia, Sarcocystis,* and *Toxoplasma.* Except for *Toxoplasma,* these parasites are host specific and rarely cause clinical disease.

Trichomoniasis occurs in owls, but is not as common as it is in hawks and eagles. Signs include salivation and diphtheritic plaques in the oral cavity. The diagnosis is easily made by direct smears from the mouth. Protozoa infecting blood cells include *Haemoproteus* spp. and *Plasmodium* spp.

Most owls in the free-ranging state have feather lice, which are observed upon first examination.[2] Avian lice are host specific and much larger than mammalian lice (5–10 mm long). Owls may also host the hippoboscid fly *Pseudolynchia* spp. Dusting powder containing a carbamate or pyrethrin insecticide readily rids the bird of these external parasites. Numerous anthelmintics are used to deparasitize owls (see Table 13.4).

TABLE 13.3. Parasites from owls in North America, but in species also found in South America

Parasite	Free-Ranging $n = 72$ (%)	Captive $n = 30$ (%)
Nematoda		
Capillaria	56 (78)	2 (7)
Ascarids	22 (31)	2 (7)
Spirurids	9 (13)	4 (13)
Syngamous	4 (6)	0 (0)
Trematoda	26 (36)	7 (30)
Cestoda	5 (7)	1 (3)
Acanthocephala	4 (6)	0 (0)

TABLE 13.4. Anthelmintics used for treatment of helminth parasites in owls

Anthelmintic	Dosage & Regimen
For nematodes	
Thiabendazole	100 mg/kg orally
Levamisole	10–20 mg/kg orally
Ivermectin	0.4 mg/kg intramuscularly
Mebendazole	200 mg/kg orally
Fenbendazole	25 mg/kg orally
For trematodes	
Rafoxanide	10–15 mg/kg orally
Praziquantel	50 mg/kg orally
For cestodes	
Praziquantel	50 mg/kg orally

Source: See reference 12.

Noninfectious Diseases

The most common reason for a veterinarian to deal with an owl is traumatic injury. The principles of dealing with such cases are standard for the rehabilitation of any injured animal. First, properly triage the owl. The general public and rehabilitators are eternal optimists and want to do anything possible to keep an animal alive. Many of the owls presented are injured so severely that rehabilitation is extremely doubtful, and the veterinarian should make an evaluation based on basic veterinary medical principles. In order to capture prey in the wild, it is vital that the bird be able to fly. Orthopedic conditions involving the wings require near-perfect healing and return to function. The basic protocol for dealing with an injured owl or any other animal, for that matter is as follows: (1) Eliminate life-threatening conditions (hemorrhage, impairment of respiration); (2) establish a normal body temperature; (3) hydrate the animal; (4) treat injuries or disease; (5) nourish the individual; and (6) place the animal into the hands of a rehabilitator who can follow through on aftercare.

Most injured owls will be hypothermic as evidenced by dullness, slowness to respond, depressed cardiac function, shivering (may not be present in nestlings), coma. Body temperature is below normal, but rectal temperatures are rarely taken on birds. An effective means of reestablishing normal body temperature is to place the owl in a plastic bag with its head out. Immerse the owl in the bag into warm 43.3–44.4°C (110–112°F) water.

Do not attempt to nourish an owl until it has been hydrated and is warm, because digestive enzyme function may be inhibited and food may decompose in the gastrointestinal tract rather than digest. Signs of dehydration include dry mucous membranes, sunken eyes, loss of skin elasticity, lethargy, dull feathers, and shock. The rule of thumb is to administer fluids at 10% of the body weight and then reassess hydration. If it is not possible to cannulate a vein, administer intraosseously.

Blood loss may be critical in owls weighing less than 100 g. Blood volume is generally around 8–10% of body weight, and 30% of the blood may be lost before there is a risk of death. However, in a 50-g bird, that may amount to 15–20 drops of blood.

Wound management follows the basic pattern for wounds in all species. Stabilize the patient, and remove a minimum number of feathers to allow for cleanliness during suturing or proper drainage. Debride any necrotic tissue. Flush the wound with sterile saline to remove all dirt and debris. Suture the wound if appropriate, and protect the wound from further contamination or self-mutilation by the victim by using bandages or Elizabethan collars.

Metabolic bone disease (rickets) is a common sequel when a private individual collects and tries to rear an injured owl that has fallen out of a nest. The bird is usually fed ground meat or other food items without proper supplementation, and within 2 or 3 weeks the bird develops bowed legs, sustains pathologic bone fractures, and fails to grow properly. Long bone cortices lack density because they have not been adequately mineralized. It is important to understand that the epiphysis of avian bones is cartilaginous rather than a center of ossification (as in mammals), until full growth is reached, after which the cartilage is converted to bone. The clinician may not be able to see the articulations in an initial assessment.

Hypovitaminosis A may occur because owls are unable to convert carotene to vitamin A and must ingest preformed vitamin A. Normally, they would obtain vitamin A when consuming the liver of their prey. The time lapse necessary before clinical signs of hypovitaminosis A appear depends on the amount of vitamin A stored in the owl's liver and how much is ingested in the diet.

Clinical signs include hyperkeratosis of keratinizing epithelium, squamous metaplasia of lacrimal and salivary glands and glands of the respiratory and urogenital tracts. It is also felt that hypovitaminosis A is a predisposing factor in the development of bumblefoot. Squamous metaplasia produces caseous plugs or plaques in the oral cavity. Obstructive renal gout may occur from squamous metaplasia in the kidney.

REPRODUCTION

Success in captive owl reproduction depends on the following factors: appropriate housing, nesting locations and materials for building a nest, photoperiod appropriate for the species, proper nutrition, sexual maturity, correct assessment of behavioral patterns, and identification of sexes.

South American owls have monomorphic color patterns, so other methods must be employed to identify sex. In raptors, the female is usually larger than the male, but it requires some experience to compare them. Methods used to determine sex include surgical sexing (otoscope, laparoscope), genetic sexing using a karyotype or DNA, and endocrine sexing using fecal/urate steroids or plasma hormones. Some of these techniques have not been validated for use in owls.

Propagation techniques include

1. pairing—must have compatible pairs or a compatible social situation;
2. natural incubation;
3. artificial incubation—techniques are known for a few species. It is necessary to know the length of incubation for the species and the proper incubation temperature and humidity,
4. hand-reared–owl chicks are altricial, thus they need special ambient temperature and humidity. Special attention must be given to providing a diet balanced for calcium and phosphorus.

REFERENCES

1. Burton, J.A. Ed. 1973. Owls of the World. New York, E.P. Dutton.
2. Cooper, J.E. 1972. Veterinary Aspects of Captive Birds of Prey. Newent, England, The Hawk Trust.
3. Cooper, J.E.; and Greenwood, A.G. Eds. 1981. Recent Advances in the Study of Raptor Diseases. Keighley, England, Chiron Publications.
4. Degernes, L.A.; Lind, P.J.; and Redig, P.T. 1993. Raptor orthopedics using methyl merthacralate and polypropylene rods. In P.T. Redig, J.E. Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Minneapolis, University of Minnesota Press, pp. 122–127.
5. Fitzgerald, G.; and Blais, D. 1993. Inhalation anesthesia in birds of prey. In P.T. Redig, J.E. Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Minneapolis, University of Minnesota Press, pp. 128–135.
6. Fowler, M.E. 1995. Restraint and Handling of Wild and Domestic Animals, 2nd Ed. Ames, Iowa, Iowa State University Press, pp. 317–321.
7. Kreeger, T.J.; Degernes, L.A.; Kreeger, J.S.; and Redig, P.T. 1993. Immobilization with tiletamine/zolazepam (Telazol). In P.T. Redig, J.E. Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Minneapolis, University of Minnesota Press, pp. 141–149.
8. Narosky, T.; and Yzurieta, D. 1989. Guia para la Identificacion de las Aves de Argentina y Uruguay [Guide to the identification of the birds of Argentina an Uruguay], 3rd Ed. Buenos Aires, Vazques Mazzini Editores.
9. Raffe, M.R.; Mammel, M.; Gordon, G.; Duke, G.; Redig, P.T.; and Boros, S. 1993. Cardiorespiratory effects of ketamine-xylazine in the great-horned owl. In P.T. Redig, J.E. Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Minneapolis, University of Minnesota Press, pp. 150–153.
10. Redig, P.T.; Cooper, J.E.; Remple, J.D.; and Hunter, D.B. Eds. 1993. Raptor Biomedicine. Minneapolis, University of Minnesota Press.
11. Ruschi A. 1979. Aves do Brasil (Birds of Brazil). São Paulo, Editora Rios.
12. Smith, S.A. 1993. Diagnosis and treatment of helminths in birds of prey. In P.T. Redig, J.E. Cooper, J.D. Remple, and D.B. Hunter, eds., Raptor Biomedicine. Minneapolis, University of Minnesota Press, pp. 21–27.

14
Order Gruiformes (Sun Bitterns, Trumpeters, Rails)

BIOLOGY
Marcia Cziulik

INTRODUCTION

This order is comprised of eight suborders, 12 families, and more than 200 species worldwide. Only the 6 families found in South America are described in this chapter.

Family Eurypygidae

Eurypyga helias (sun bittern, 46–48 cm) is the single species in the family and is exclusively Neotropical, ranging from Colombia, Venezuela, and the Guianas south; west of the Andes to northwestern Peru; and east of the Andes through eastern Ecuador and eastern Peru to northeastern Bolivia, and Brazil to southern Goiás, Mato Grosso, and Amazonas. They have thin, S-shaped necks and long, sharp beaks. The legs are slender, the wings long and wide. They live along shady streams, small forested pools, and damp, dense second-growth thickets near water, and feed on insects and other small animals. Solitary birds or pairs walk along stream banks or damp places in the forest. They fly to low branches if disturbed. Nests are built in low trees near rocky streams and are globular mounds, constructed of vegetation and mud with a shallow depression. Two or three eggs are in a clutch. Genders are similar.

Family Aramidae

Aramus guarauna (limpkin, 61–71 cm) is confined to the warmer areas of the New World, from Colombia, Venezuela, Trinidad, and the Guianas south; west of the Andes to western Ecuador; and east of the Andes through eastern Ecuador, eastern Peru, Brazil, Bolivia, and Paraguay to Uruguay and northern Argentina. They have a long halux, and the trachea in adult males is convoluted, serving as a voice volume amplifier. They are usually seen in freshwater swamps or marshes, marshy river banks, mudflats, and mangroves. They feed mainly on large *Pomacea* snails. Partially nocturnal, they are not shy, but rather conspicuous. They are often alone, but also may be seen in small groups or with other aquatic birds. The nest is a platform of twigs and leaves less than 3 m high in marsh vegetation, in which three to six eggs are laid. Genders are similar, but females are smaller.

Family Heliornithidae

Heliornis fulica (Sungrebe, 28–30 cm) is the only species in this family found in the New World, ranging from Colombia, Venezuela, and the Guianas south; west of the Andes to northwestern Peru; and east of the Andes through eastern Ecuador, eastern Peru, northeastern Bolivia and Brazil to Paraguay and northeastern Argentina. They have slender necks, sharp beaks, long wings, long and wide tails, and short, strong legs. The

TABLE 14.1. Biological data on selected gruiforms

Scientific Name	Common Name	Size (cm)	Distribution	Habitat
Posophia crepitans	Grey-winged trumpeter	48–56	East of Andes from southeastern Colombia to Brazil, Ecuador, Peru	Humid forest
Psophia leucoptera	Pale-winged trumpeter	55	Eastern Peru, northeastern Bolivia and western Amazonian Brazil	Humid forest
Psophia viridis	Dark-winged trumpeter	55	Lowlands of Amazon basin	Humid forest
Cariama cristata	Red-legged seriema		Lowlands of southeastern South America to northern Argentina	
Chunga burmeisteri	Black-legged seriema		Lowlands of south central South America to northwestern Argentina	
Genera *Micropygia, Anurolimnas, Laterallus, Porzana, Neocrex*	Crakes	14–20	Various locations in northern South America	Marshes, swamps, damp thickets, boggy meadows
Genera *Coturnicops, Rallus, Aramides, Paradirallus*	Rails and woodrails	15–40	Various locations in South America	
Porphyrio martinicus	Purple gallinule	25	Northern Chile and Argentina	Freshwater, marshes, pools, lagoons
Gallinula chloropus	Common moorhen	28	Most of South America to northern Chile and Argentina	Marshes, swampy riparian bushes, reed beds
Fulica americana	American coot	33–36	Colombia and Ecuador	Ponds, marshes, rivers, estuaries, bays
Fulica spp.	Coots	33–60	Various locations in South America	Ponds, marshes, rivers, estuaries, bays

thorax is flattened dorso-ventrally, which produces the overall flat form of the body. Sungrebes live on freshwater ponds and slow-moving streams. They swim along shady, secluded stream banks and dive for food. They may swim partly submerged, only the head and neck showing above water. When alarmed, they make a pattering run across water to take flight, staying low and seldom flying far. Sungrebes eat a variety of small aquatic life and some seeds. They nest in bushes, constructing a platform lined with leaves about 1 m above water. Two eggs are laid.

Family Psophiidae

Trumpeters are a small group of terrestrial birds largely confined to Amazonia. They are gregarious, sometimes forming flocks of as many as 100 birds. They have small heads and arched necks covered with short feathers. The beak is short and strong, the wings wide and drooping, which produces an appearance of strength. The tail is short and soft, the legs long with short toes. They are terrestrial, but perch in trees at night and are known to swim across rivers. They eat plants and animals, including reptiles and amphibians. Trumpeters build nests of leaves in natural cavities in trees. A clutch may contain as many as six eggs. Genders are similar, but males are slightly smaller than females. There is no information about their courtship, breeding, or social behavior. Species are listed in Table 14.1.

Family Cariamidae

These birds are distinguished from other birds by their hawklike heads and long necks, tails, and legs. They live on the pampas, feeding on small reptiles and mammals. They are reputed to devour snakes, but this is exaggerated. They hold their prey in the beak and dash it violently against the ground to kill it. They are mainly terrestrial and fly little. They nest in trees. Species are listed in Table 14.1.

Family Rallidae

This cosmopolitan family inhabits coastal and inland swamps, marshy woods, and moist meadows. They may be divided into two large groups: rails, which habitually walk, although they are good swimmers, and gallinules, which are commonly seen swimming, although they walk well. Many species of this family are secretive and rarely seen; some are also crepuscular and nocturnal in habit, preferring to run rather than fly from danger. Typical rails are characterized by narrow, laterally compressed bodies. Their size varies. They have long legs and toes. They have short tails and long colorful beaks. Females are usually considerably smaller than males. During the mating season, males and females caress each other's head and sleep together in the nest, which is made of leaves on bushes or, sometimes, in holes. They feed on insects and other small animals, seeds, vegetation, and fruit. Species are listed in Table 14.1.

CAPTIVE MANAGEMENT

Recommendations for breeding gruiform birds in captivity are discussed next.

The recommended size of an enclosure for small to medium-sized birds is 1 bird/3–5 m^2; for Cariamidae, 1 bird/10 m^2. The minimum height should be 2 m, and the enclosure should be at least 1 m away from the public.

The substrate may be earth, grass, or marsh, covered with sand or leaves. Trees, bushes, and herbs are necessary to produce shadows and provide material for the construction of nests. Many species build their nests in or under bushes. Seriemas may use basketlike nests made of fabric. It is important that the birds have free access to sticks and leaves with which to build and pad their nests.

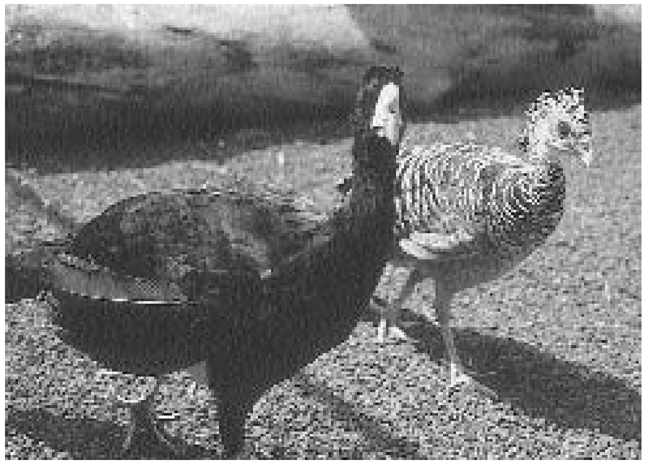
Most species require renewable water sources for bathing. The size of the pool will depend on the area available in the enclosure. In hot climates, sprinklers may be installed over the enclosures. They should spray in 30-minute intervals during the hottest period of the day.

Cariamidae require roosts to sleep. Ramphastidae and many other families of birds may share enclosures with Gruiforms in harmony. However, attention is necessary during the mating season. Also, in a collective aviary there must be a protected area for new birds to become adapted to the environment and to the other species.

In captivity, birds should be fed twice a day, once in the morning and once in the afternoon, according to the basic requirements of each species. Fruits, seeds, roots, and meat should be included in the diet of these birds. When nestlings are present, the diet should contain a higher protein level. In Rallidae, this is achieved by feeding them eggs and dog pellets. Seriemas should be offered medium-sized rats or domestic chicks, if parents are not able to find insects in the enclosure. There is little published information available related to the feeding, breeding, or behavior of South American bird species.

REFERENCES

1. De Schauensee, R.M. 1970. A Guide to the Birds of South America. Pennsylvania, Livingston Publishing.
2. De Schauensee, R.M.; and Phelps, W.H., Jr. 1978. A Guide to the Birds of Venezuela. Princeton, New Jersey, Princeton University Press.
3. Dunning, J.S. 1982. South American Land Birds. Newtown Square, Pennsylvania, Harrowood Books.
4. Hilt, S.L.; and Brown, W.L. 1986. A Guide to the Birds of Colombia. Princeton, New Jersey, Princeton University Press.
5. Mödlinger, B.A.; and Holman, G.M. 1986. Guia de Campo de Las Aves de Chile, 3rd Ed. Chile, Editorial Universitaria.
6. Rutgers, A.; and Norris, K.A. 1979. Encyclopaedia of Aviculture. Dorset, England, Blandford Press.
7. Sibley, C.G.; and Monroe, B.L., Jr. 1990. Distribution and Taxonomy of Birds of the World. New Haven, Connecticut, Yale University Press.
8. Sick, H. 1988. Ornitologia Brasileira: Uma Introdução, 3rd Ed. Brasilia, Editora Universidade.



15
Order Galliformes, Family Cracidae

CRAX BLUMENBACHII PRESERVATION PROJECT

Roberto Motta de Avelar Azeredo
James G.P. Simpson
Lúcia Paolinelli Barros

INTRODUCTION

Crax blumenbachii is one of the most endangered cracids, according to all official lists. Motivated by that fact, a field study was begun in 1975 by Roberto Motta de Avelar Azeredo. Since 1987, this research project has become a scientific nonprofit entity entitled CRAX—Wild Fauna Research Society (Sociedade de Pesquisa do Manejo e da Reprodução da Fauna Silvestre). Here we report on what has been achieved.

BIOLOGY

C. blumenbachii (red-billed curassow; southeast curassow) belongs to the *Crax* genus, of the Cracidae family, Galliformes order. It is a bird of dignified bearing, 84 cm (33 in.) tall, weighing 3.5 kg, (7.7 lbs)[6] and spends most of the day on the ground and feeding on seeds, fruits, leaves, sprouts, and insects.[4,8] It is sexually dimorphic[6] and found mostly in pairs.[6] The male has a red caruncle, black back, and white belly. The female also has a black back, but a rusty-colored belly.

Observations of free-ranging birds indicate that females are fertile at 2 or 3 years of age and remain fertile for at least 11 years.[8] The size of the brood is usually two chicks,[4,8] sometimes one.[4] In captive or semi-captive conditions, sexual maturity is attained at around 2.5 years of age, and females may normally breed for 21 years. Usually two eggs are laid, rarely one or three. The breeding season in the natural distribution area of the species is from September to February. Laying generally occurs at the end of the afternoon, with an interval of 48 hours between two eggs.

The male chooses a tree and builds the nest on a high and heavily forked branch, using small branches pulled from the same tree. The female incubates the eggs, and the pair raise the brood. Under normal temperature conditions, the incubation period lasts 30.5 days. With artificial incubation, the temperature must remain between 37.2 to 37.8°C (99–100°F), with humidity between 55 and 60%. The eggs must be turned at least every 12 hours.

During incubation in the wild, the eggs soon lose the initial white color and display the earth-colored camouflage brought in by the female's feet. The female leaves the nest during the hottest hours of the day, not rarely more than once a day, with the exception of the last week. The chicks may remain with the parents for as long as 8 months. Only one laying occurs per year. In captivity, if the eggs are gathered for artificial incubation, the female may lay as many as eight times.

In the wild, chicks usually hatch in the early morning, remain approximately 6 hours in the nest, and then jump to the ground, whatever the height of the nest may be. In the existing climate conditions in the natural distribution area, chicks do not require warmth (nor do hatchlings from artificial incubation) and roost apart from the parents beginning the first day, at 1–1.5 m above the ground, in dense branches, while the parents roost in the treetops. Within a few days, the chicks roost in the treetops, as well.

When raised by the parents, chicks immediately receive, through physical contact, an oily protection that keeps them dry (they are born in the rainy season). When the feathers develop, they acquire their own protection.

Newly hatched chicks almost always walk under their parents' tails, spread open for that purpose. The parents deliver food to chicks' bills, teaching them how and what to eat. Leaves and insects are the chicks' basic food early in life. In captivity, the food for birds of all ages may be similar to pheasant feed, supplemented with leaves, fruit, and tubers. In the breeding season, the protein level must be raised from 10–15% to 20% for adult pairs.

Predators of curassows include cats, foxes, and large hawks, which attack both adults and chicks. Large lizards eat eggs and chicks, and primates, coatis, skunks, and carrion crows disrupt the nests.

PRESENT STATUS

These birds are endemic in southeastern Brazil and belong to the rain forest biome. Their populations are in decline because of the strong deforestation pressure occurring in the last decades, which is disrupting their habitat. That is the greatest menace. With reduction of the habitat, hunting began to be a serious problem. Since the species became scarce, trading has become a constant additional menace. Formerly abundant, it is now one of the most threatened of Brazilian birds[2,5] and one of the three most endangered Brazilian cracid species. Reproduction in captivity may be the only solution to save it from extinction.[6] Species counts report fewer than 300 individuals in the wild[9] and populations are restricted to a few areas in southern Bahia, Monte Pascoal National Park[6,7] in northern Espírito Santo State, and Sooretama Reserve in eastern Minas Gerais State in the Rio Doce State Park. Birds have been reintroduced on a site chosen at Macedônia Farm (Cenibra) with the Fechos (Copasa), and Peti (Cemig) reserves, the three of them owned by the companies indicated; all of them are in the Minas Gerais State in southeastern Brazil. It was estimated in 1995 that the captive population consisted of no more than 500 individuals, of which about 30 were outside Brazil (CAMP-IUCN/SSC-1995). Today, there are approximately 1000 individuals in captivity, of which, it is estimated, 130 are abroad.

CONSERVATION STRATEGIES

Reproduction and study in captivity, protection of natural areas where the species still occur, rehabilitation of areas where the species once existed, and reintroduction of birds into these areas are the primary conservation strategies currently being pursued.

CONSERVATION PROJECT

The first captive hatching occurred in 1981. Since then, 1000 births have been recorded, confirming the success of the techniques for handling and reproduction compiled by CRAX to develop a basic methodology for the Wild Species Conservation Integrated Program, composed of the following eight stages.

1. Definition of species to be studied.
2. Acquisition of breeding pairs or groups.
3. Development of reproduction and handling techniques under captivity.
4. Multiplication of the number of individuals in captivity.
5. Identification of natural reserves for reintroduction of the species.
6. Reintroduction and selective distribution.
7. Postreintroduction monitoring.
8. Promotion of environmental education and scientific cooperation.

C. blumenbachii was the first, among the 50 endangered species under study at CRAX, for which the eight stages of the methodology have been completed, and it is now capable of fighting the extinction menace. In 1991 CRAX was able to reintroduce this species back to nature, within the Preservation Project discussed here.

On January 9, 1991, after an 8.5-month adaptation period in an aviary 20 m (66 ft) long, 14 m (46 ft) wide, and with a height of about 13 m (43 ft), built inside the forest on a site chosen at Macedônia Farm with the participation of the environmental authority, 15 pairs of the species averaging 2.5 years of age were released. On February 17, 1993, to complete the original aim and replace seven deaths, 21 males and 16 females were released, with the mean age of 2 years. CRAX has been reducing little by little the releasing age, always with good results—the younger the birds, the better they adapt to natural life. Both releases were supported by food and water strategically distributed close to the aviary, especially in the first weeks, to make a gradual and safe dispersion possible for the birds. Since the first release, a monitoring program was set up, which continues until today. At least 26 chicks (15 males and 11 females) have hatched, and individuals of the third generation are already present in the group, which represents a very favorable rate with regard to the developed initiative. Based on this accumulated experience, reintroductions of *C. blumenbachii* into two other natural areas are under way.

Further work at the CRAX Research Center includes selection of breeding pairs or groups of different genetic lineages; combined research on natural or artificial incubation of the eggs and raising of the chicks; identification of chicks with closed bands at approximately 60 days of age; preadaptation in semicaptivity in forested areas; selection of individuals suitable for reintroduction; health tests, weighing, and measuring; and postrelease monitoring.

CONCLUSIONS

Experience has shown that the proposed and applied methodology developed by CRAX may be adapted for other species to avert the extinction menace that approaches in different degrees for a great number of the world's wild fauna species. An important aspect is to point out that projects of reintroducing animals to nature, together with public or private enterprise owners of natural reserves and resources, fit extraordinarily well in the modern concept of sustainable development. These conditions become classic cases that may serve as a reference to the environmental, scientific, and business publics, among others.

REFERENCES

1. Collar, N.J.; Gonzaga, L.P; Krabbe, N.; Madroño Nieto, A.; Naranjo, L.G.; Parker, T.A., III; and Wege, D.C. 1992. Threatened Birds of the Americas: The ICBP/IUCN Red Data Book, 3rd Ed. Washington DC, Smithsonian Institution Press.
2. Delacour, J.; and Amadon, D. 1973. Curassows and Related Birds. New York, The American Museum of Natural History and Chanticleer Press.
3. Schubart, O.; Aguirre, C.A.; and Sick, H. 1965. Contribuição para o conhecimento da alimentação das aves brasileiras. Arquivos de Zoologia 12.
4. Sick, H. 1970. Notes on Brazilian Cracidae. Condor 72(1):106–108.
5. Sick, H. 1972. A ameaça da avifauna brasileira. In Espécies da Fauna Brasileira Ameaçada de Extinção. Academia Brasileira de Ciências, Rio de Janeiro, Brazil, pp. 99–153.
6. Sick, H. 1985. Ornitologia Brasileira: Uma Introdução, Vols. 1–2. Brasília, Editora Universidade de Brasília.
7. Sick, H.; and Teixeira, D.M. 1979. Notas sobre aves brasileiras raras ou ameaçadas de extinção. Publiçacães Avulsas do Museu Nacional 62:39.
8. Teixeira, D.M.; and Antas, P.T.Z. 1982. Notes on endangered Brazilian Cracidae. In Proceedings of the 1° Simpósio Internacional sobre la Familia Cracidae. Mexico.
9. Strahl, S.; Ellis, S.; Byers, O.; and Plasse, C. Eds. 1995. Conservation Assessment and Management Plan for Neotropical Guans, Curassows and Chachalacas. Working Draft. Apple Valley, Minnesota, IUCN/SSC, Conservation Breeding Specialist Group.

16
Order Columbiformes (Pigeons, Doves)

Luiz Francisco Sanfilippo
Karin Werther

BIOLOGY

Luiz Francisco Sanfilippo

INTRODUCTION

There are nine genera and 47 species of Columbiformes in South America (Table 16.1).

HABITAT

Pigeons and doves are found worldwide, except in the Arctic and Antarctic. Most of them are powerful flyers, which has enabled them to fly across great expanses of water to colonize oceanic islands. The ancestors of the pigeons found in the Galapagos Islands crossed almost 1000 km of ocean.

Pigeons occupy varying habitats from dense forests to large open fields. They may be arboreal (*Columba, Leptotila*), partially terrestrial (*Columbina, Scardafella*), or exclusively terrestrial (*Geotrygon*). *Columba corensis* may be found in arid regions as well as mangrove swamps. *Metriopelia ceciliae* rest and make their nests in rocky shelters. *Metriopelia aymara* may be found at elevations of 3000-5000 m.[1]

Columbiforms may be divided by their feeding habits into two groups, one that feeds mainly on seeds and the other primarily on fruits. Many species may also eat small invertebrates. Some species (e.g., *Zenaida auriculata*) have benefited from human activity, with increased populations and expanded geographical distribution.[2]

The rock dove, *Columba livia,* has a worldwide association with people. Inhabiting mountain regions, originally native to Europe, North Africa, and Southwest Asia, it is now found in both rural and urban habitats. There are 13 subspecies of *C. livia* and more than 100 races or breeds have been developed through a selection of desired features.[1]

Many species are hunted for food. Some populations are considered to be pests, feeding on crops. Some die from ingestion of seeds poisoned by pesticides. Specialized species are the most vulnerable and susceptible to environmental changes. *Claravis godefrida,* for example, were abundant when bamboo forests flourished, but their habitat disappeared and they became endangered; they are now near extinction. *Columba oenops* and *Leptotila ochraceiventris* are vulnerable species, and *Leptotila conoveri* is considered to be at risk of extinction. *Columbina cyanopis* is also critically endangered.[1]

TABLE 16.1. Columbiformes distribution in South America

Genera	Species	Distribution
Claravis	*C. pretiosa*	Southern USA to central Brazil
	C. godefrida	Southeastern Brazil, eastern Paraguay, and northeastern Argentina
	C. mondetoura	Southeastern Mexico to west-central Bolivia
Columba	*C. livia*	Worldwide
	C. speciosa	Southern Mexico and southern Brazil
	C. picazuro	Northeastern Brazil, southeastern Bolivia, and southern Argentina
	C. corensis	Venezuela and Colombia coast and adjacent islands
	C. maculosa	Peru, Bolivia, Argentina, Paraguay, Brazil, and Uruguay
	C. fasciata	Southwestern North American coast, Central America, and northwestern South American coast
	C. araucana	Central and southern Chile and southern Argentina
	C. cayennensis	Southern Mexico, northern Argentina, and Uruguay
	C. oenops	Southeastern Equator and northern Peru
	C. plumbea	Eastern Colombia, southern Venezuela, and northeastern Paraguay
	C. subvinacea	Costa Rica, western Colombia Andes, and southern Brazilian Amazon forest
	C. nigrirostris	Southeastern Mexico, eastern Panama to northeastern Colombia
	C. goodsoni	Western Colombia and northwestern Equador
Columbina	*C. passerina*	Southern USA to central Brazil
	C. minuta	Southern Mexico to central Amazon forest, southwestern Equador and Peru, central Brazil, southern Paraguay, and northeastern Argentina
	C. buckleyi	Northwestern Equador and Peru
	C. talpacoti	Southern Mexico to northern Argentina
	C. picui	Northeastern Brazil to southern Argentina
	C. cruziana	Northern Equador to northern Chile
	C. cyanopis	South-central Brazil
Geotrygon	*G. goldmani*	Eastern Panama to northwestern Colombia
	G. saphirina	Northwestern Colombia to southeastern Peru
	G. veraguensis	Costa Rica to northwestern Equador
	G. linearis	Northeast Colombia to northwestern Venezuela
	G. frenata	Western Colombia to northwestern Argentina
	G. violacea	Nicaragua to south-central Venezuela, Brazilian coast, southern Paraguay, northeastern Argentina, northern and eastern Bolivia
	G. montana	Mexico to northern Argentina
Leptotila	*L. verreauxi*	West-central Mexico to north-central Argentina
	L. megalura	Andes from northern Bolivia to northwestern Argentina
	L. rufaxilla	Eastern Colombia to northeastern Argentina
	L. plumbeiceps	Eastern Mexico and western Colombia
	L. pallida	West Colombia to Equador
	L. cassini	Southern Guatemala to northern Colombia
	L. ochraceiventris	Southwestern Equador to northwestern Peru
	L. conoveri	Central region of Andes in Colombia
Metriopelia	*M. ceciliae*	Western Peru to northwestern Argentina
	M. morenoi	Northwestern Argentina
	M. melanoptera	Andes from southwestern Colombia to southern Chile and Argentina
	M. aymara	South-central Peru and northwestern Argentina
Scardafella	*S. squammata*	Colombia and Venezuela coast, east-central Brazil, Paraguay, and northeastern Argentina
Uropelia	*U. campestris*	Amapa state and Marajo Island (Brazil), west-central Brazil, northern and eastern Bolivia
Zenaida	*Z. auriculata*	South America
	Z. galapagoensis	Galapagos Islands
	Z. meloda	Southwest Equador to Northern Chile

Pigeons and doves are commonly propagated by aviculturists and zoos. The reproductive management of *C. livia* is well understood, whereas little is known about other species. Generally, pigeons and doves are monogamous. They make oval-shaped nests of leaves and twigs in shrubs or in the tops of trees.

REFERENCES

1. del Hoyo, J.; Elliott, A.; and Sargatal, J., Eds. 1997. Handbook of the birds of the world, Vol. 4. Barcelona, Lynx Editions, pp. 60–245.
2. Sick, H. 1997. Ornitologia Brasileira. Rio de Janeiro, Nova Fronteira, pp. 341–350.

MEDICINE

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PARASITISM

Helminths

A wild pigeon, *Z. auriculata,* that originated from São Paulo State, Brazil, was diagnosed with a severe nematode parasitism of the proventriculus. The female nematodes were located in the submucosa of the proventriculus, which was dark and swollen. It was possible to see the nematodes through the wall of the proventriculus (Figure 16.1). The white and elongated male nematodes were found in the mucus of the proventriculus lumen. The parasites were not identified as to species, but were similar to *Tetrameres* sp.

Some of these pigeons also had intestinal nematodes from the family Ascaridea. Although the parasitic infestation was high, the birds had normal body condition.

Protozoan

An important protozoan parasite of both free-ranging and captive columbiforms is the flagellate *Trichomonas* sp. Infested birds are usually in poor condition. Yellowish plaques are found on the palate, beak commissures, and the mucosa of the oral cavity. The plaques adhere closely to the mucosa and are difficult to remove. A putrid odor may be emitted from the oral lesions, and the bird is likely to be anorectic. The crop and esophagus may also be affected.[1] A diagnosis is made by direct microscopic examination of the organism on swabs taken from the lesions. Trichomonads survive outside the host for only 30–60 minutes and are not easily identifiable at necropsy.[1]

Insects

The pigeon louse fly (*Pseudolynchia* sp.) is frequently observed in columbiforms. The fly feeds on blood and is often responsible for transmission of the hemoparasite *Haemoproteus* sp. These flies have short wings and are not good fliers; rather, they hide under the feathers of the host.

INFECTIOUS DISEASES

Bacterial

Only a few bacterial infections are of clinical importance in pigeons. The most common are *Chlamidia psittaci, Salmonella typhimurium, Escherichia coli, Streptococcus bovis,* and *Clostridium* sp. Other bacteria may be identified, but are mostly restricted to isolated disease problems.[2]

C. psittaci infection in pigeons is usually a respiratory disorder, but certain strains cause serious diarrhea and weight loss in young birds.[3] Salmonellosis causes yellow diarrhea, anorexia, droopiness, and death after 2 to 3 days in young birds. In a flock, signs include swollen joints, wing paralysis, central nervous system signs, weight loss, thin, slimy diarrhea, and death. *E. coli* infections also cause diarrhea in young pigeons (2–6 months old), often appearing after the birds fledge. Infection with *S. bovis* causes sudden death in pigeons of all ages. Clinical signs are inability to fly, lameness, emaciation, polyuria, and green slimy droppings.[2]

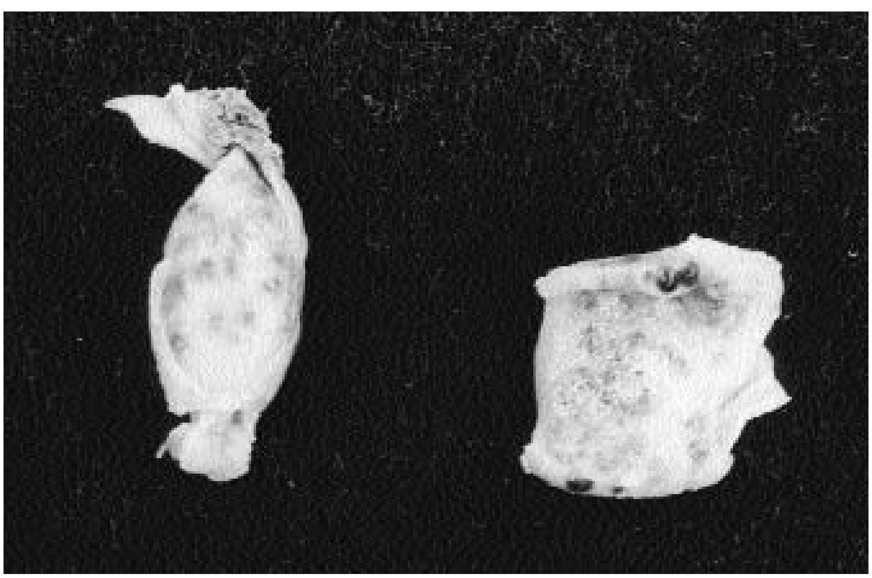

FIGURE 16.1. Proventriculus from *Zenaida auriculata* pigeon parasitized by *Trematodes* sp.

Viral

ADENOVIRUS INFECTION

Epizootiology. Birds from hatchling to 5 years of age are susceptible to adenovirus infection. Young birds may die within 48 hours after developing clinical signs. Mortality is highest 3–4 days after infection. Transmission is by the fecal or oral route. Virus is shed in high concentration in the feces. Free-ranging pigeons may be a source of virus for captive birds.[4]

Signs include acute death without premonitory signs, but more commonly, 2- to 4-month-old pigeons develop depression, anorexia, dyspnea, a crouched stance, polydipsia, polyuria, and slimy green diarrhea.

At necropsy, there is an enlarged liver and spleen, yellowish to copper gold patchy discoloration of the liver, hyperemia of the intestinal mucosa, and greenish mucoid fluid in the crop and intestinal tract. Microscopically, there are intranuclear inclusion bodies in the hepatocytes and epithelium of the intestines, similar to those caused by pigeon herpesvirus.[4]

Differential Diagnosis. Diagnosis should consider bacterial hepatitis or enteritis, chlamydiosis, salmonellosis, reovirus, and pigeon herpesvirus. In the living bird the virus may be seen in the feces using electron microscopy, or the virus may be isolated from feces. Birds with antibodies are considered to be latently infected. At necropsy, detection of suggestive microscopic changes, virus isolation from liver and intestines, and viral-specific DNA probes may confirm the diagnosis.[4]

Management. For prevention, vaccines are available for pigeons. The virus is stable outside the host and resistant to many disinfectants, but is inactivated by 1-hour exposure to formalin, aldehydes, and iodophors.

Therapy should include supportive care, administration of fluids, assisted feeding, and administration of broad spectrum antibiotics to prevent secondary infections.[4]

POX

Etiology. Poxvirus is among the largest and most complex of all animal viruses. The diameter is around 400 nm.

Epizootiology. A break in the epithelium is necessary for infection to occur upon direct contact with an infected bird or indirect contact with a contaminated object or insect. Mosquitoes and mites serve as the primary mechanical vectors. Epornitics are common in the spring and fall when mosquitoes are most prevalent. Affected birds are most infectious when lesions or scabs are present. Affected free-ranging pigeons and doves may serve as a source of the virus for captive birds. The incubation period is from 7 to 9 days.[1]

Signs. The cutaneous form is characterized by small blisterlike areas that progressively enlarge, ulcerate, and scab. There may be large wartlike proliferative growth on the skin and eyelids, and commissures of the beak, feet, and legs. Lesions are common around the cloaca and the umbilicus of unfeathered young. Uncomplicated lesions resolve in 3–4 weeks.

The diphtheritic form is characterized by thickened, yellowish, necrotic plaques in the mouth, esophagus, crop, and trachea. Accumulation of necrotic debris in the trachea may cause asphyxiation. Mortality is highest in young pigeons and may approach 50%.

At necropsy, gross lesions may be seen on the skin and oral mucosa. Fibronecrotic lesions in the gastrointestinal tract and ulcerations of the nasal, tracheal, and bronchial mucosa may be seen. Necrosis of the cells lining the mouth, esophagus, crop, and trachea may be observed using a microscope. Bollinger bodies (inclusion bodies) occur in the skin and on the mucosa of the sinuses, trachea, crop, and esophagus.[4]

In São Paulo State, Brazil, there was an occurrence of poxvirus in captive *Z. auriculata* captured from the wild and transferred to an enclosure. Some weeks later the majority of the birds died. At necropsy, suggestive macroscopic lesions were seen on the feet (Figure 16.2) and at the periocular skin (Figure 16.3). Microscopy showed Bollinger intracytoplasmic inclusion bodies, confirming the diagnosis of poxvirus.

Differential Diagnosis. Differential diagnosis should include (in the cutaneous form), trauma, *Trichophyton* sp. infection, and bacterial dermatitis. In the diphtheritic form, candidiasis, hypovitaminosis A, aspergillosis, trichomoniasis, or herpesvirus should be considered.

In the living bird, the virus may be isolated from vesicles, necrotic mucosa, pharyngeal swabs, or feces, or diagnosis may be made by demonstrating poxvirus in lesions using cytology or electron microscopy. Serologic tests may detect antibodies using enzyme-linked immunosorbent assay (ELISA), hemagglutination inhibition (HI), agar-gel immune diffusion (AGID), or virus neutralization (VN) assays.

Diagnosis at necropsy includes microscopic examination of tissues for intracytoplasmic inclusions and virus isolation from skin lesions, mucosal lesions, or the liver.[4]

Management. Attenuated-live virus vaccines are available. Vaccinations of pigeons in high-risk areas

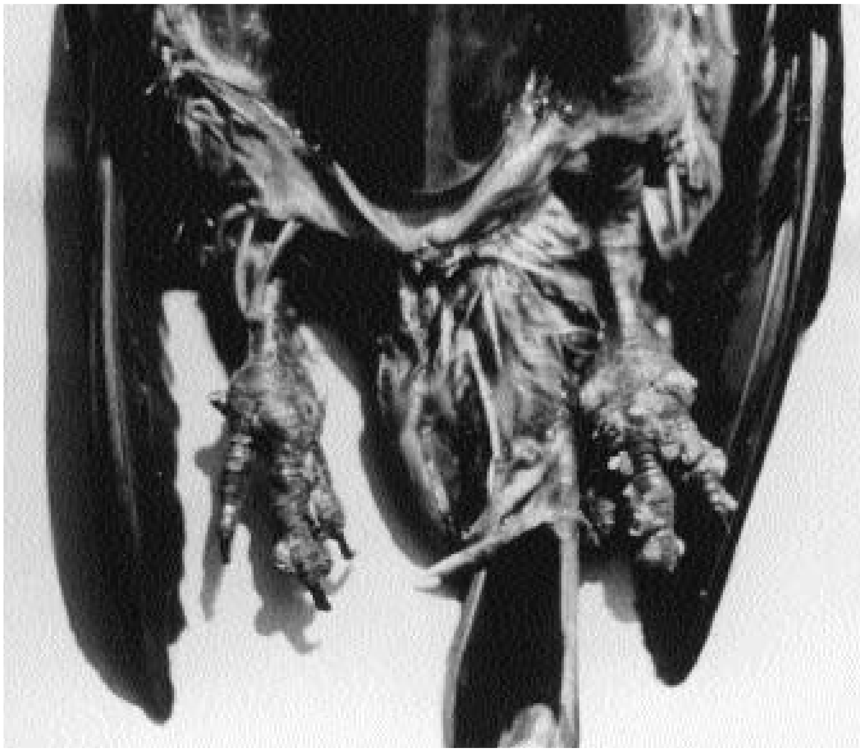

FIGURE 16.2. Feet of *Zenaida auriculata* pigeon with typical skin lesions caused by poxvirus.

FIGURE 16.3. Poxvirus lesions around the eyes of a *Zenaida auriculata* pigeon.

may be done at 4–6 weeks of age and 1 month before the race or show season.[4]

Virions are stable outside the birds, but may be inactivated with 1% potassium hydroxide at a temperature of 50°C for 30 minutes or 60°C for 8 minutes, 2% sodium hydroxide (NaOH), and 5% phenol. Infected birds and directly exposed birds should be isolated. Contaminated wooden perches or nest boxes should be destroyed, and contaminated nets, towels, clothing, holding containers, and other equipment and supplies should be sterilized.[4]

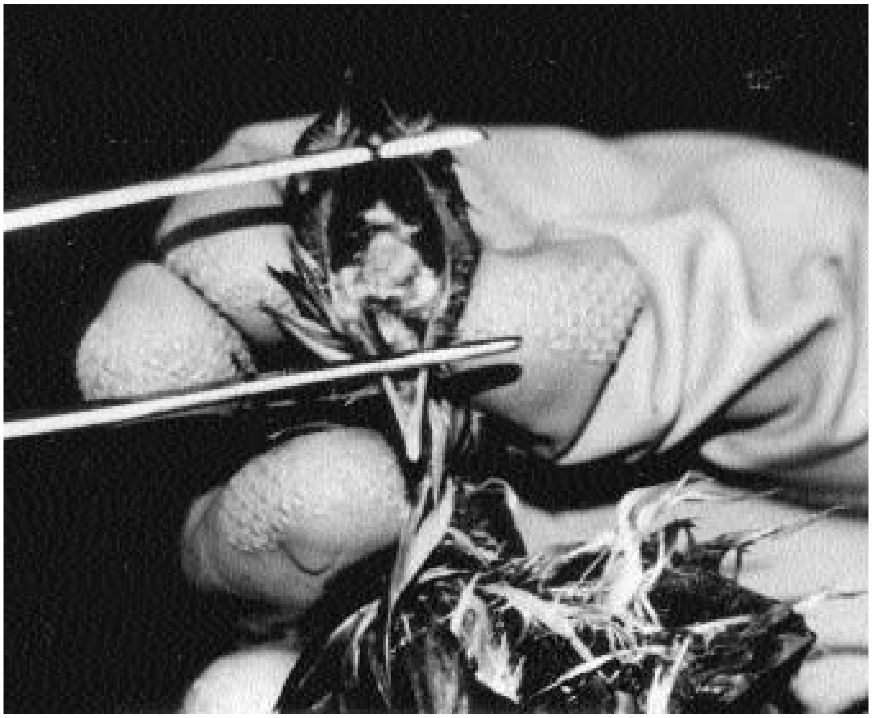

FIGURE 16.4. Oral cavity of a *Zenaida auriculata* pigeon with typical plaques caused by *Trichomonas* sp.

Therapy. Infected tissue should be gently removed and open wounds cleaned. Topical antibiotics should be applied. Systemic antibiotics should be administered to birds with lesions in the respiratory and gastrointestinal tracts. Secondary candidiasis or trichomoniasis should be treated and birds supplemented with vitamin A (10,000–25,000 IU/300 g body weight, intramuscularly), and vitamin C.[4] (See Figure 16.4.)

HERPESVIRUS INFECTION The virus is shed in feces, in respiratory secretions, and in contaminated secretions fed to squabs. Egg transmission is unproven. The incubation period is 1–3 days.

Signs. Signs include conjunctivitis; rhinitis; dyspnea; ulcerative lesions on the mucosa of the larynx, pharynx, or oral cavity; diarrhea; vomiting; and neurologic signs.

Lesions include ulcers on the mucosa of the alimentary tract, larynx, and pharynx;, hepatomegaly; splenomegaly; congestion of intestines; and formation of sialoliths. Microscopically, there is necrosis of the liver, spleen, pancreas, kidneys, and lungs. Intranuclear inclusion bodies occur in the pharyngeal mucosa, liver, spleen, pancreas, and tracheal mucosa.[4]

Differential Diagnosis. Diagnosis should include chlamydiosis, salmonellosis, trichomoniasis, poxvirus (diphtheritic form), paramyxovirus (PMV-1), and adenovirus. In the living bird virus-neutralizing bodies are detectable within a week of infection. The virus may be isolated from feces or respiratory secretions (pharyngeal swabs). Inclusion bodies may be observed in impression smears from ulcerative lesions. At necropsy, diagnosis is confirmed by microscopic examination of tissues for intranuclear inclusions and virus isolation from the pharynx, liver, spleen or pancreas.[4]

Management. Experimental vaccines prevent clinical disease, but not latent infections. The virus is unstable outside infected pigeons and is considered susceptible to most disinfectants. No therapy exists for this disease.[4]

PARAMYXOVIRUS-1 INFECTION

Epizootiology. Transmission is by ingestion or inhalation of contaminated respiratory secretions or feces. The virus is excreted in laryngeal secretions 2–9 days after infection and in feces 2–14 days after infection. Shedding may continue for as long as a month.[4] The natural incubation period is 5 days to more than a month. The experimental incubation period is 5–18 days.

Signs. Signs include incoordination, tremors of the head and wings, circling, paralysis, head shaking, torticollis, polydipsia, polyuria, diarrhea (may be watery or hemorrhagic), and coma. Clinical signs appear in 20–80% of exposed pigeons. Mortality may be as high as 90% of young pigeons; older birds typically recover in 3–4 weeks.

Lesions include hyperemia of the brain and abdominal organs, catarrhal enteritis, enlarged kidneys, and hemorrhage of the pancreas. Microscopically, there is inflammation of the intestines during early infection, hepatitis, and accumulation of lymphocytes and plasma cells in the kidneys. Other lesions include nonsuppurative encephalitis, hepatic necrosis, pancreatitis, necrosis of the liver, spleen, pancreas, kidneys, and lungs. Intranuclear inclusion bodies may be found in the pharyngeal mucosa, liver, spleen, pancreas, and tracheal mucosa.[4]

Differential Diagnosis. Diagnosis should include chlamydiosis, salmonellosis, Newcastle disease virus, pigeon herpesvirus, heavy metal toxicity, organophosphate poisoning, bacterial hepatitis, and bacterial enteritis. In the living bird, the virus can be detected in the feces using electron microscopy, and by a fourfold increase in the antibody titer using paired serum samples. At necropsy the virus may be isolated from the intestines, trachea, lung, spleen, liver, and brain.

Vaccination prevents overt disease, but not infection or shedding. Both attenuated live-virus and inactivated vaccines have been used. Adults should be vaccinated for a month before the breeding, racing, or show season; squabs at 3–4 weeks of age, and new pigeons during the quarantine period.

Control. The virus is relatively stable outside the bird, at 50° C for 19 weeks and 27° C or 40° C for 4 weeks. The virus is inactivated by high temperature (56°C), sunlight, detergents, chloramine (1%), sodium hypochlorite, Lysol, phenol, and 2% formalin. Pigeons with clinical signs should be isolated. New birds or those that leave the aviary for 6–8 weeks should be quarantined for 6–8 weeks. Free-ranging pigeons should be prevented from entering the loft.

Therapy. Therapy includes supportive care, administration of fluids, and administration of antibiotics to prevent secondary infections.[4]

REFERENCES

1. Vogel, C; Gerlach, H; and Loffler, M. 1994. Columbiformes. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 1200–1217.
2. Hooimeijer, J.; and Dorrestein, G.M. 1997. Pigeons and doves. In R.B. Altmann, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 886–909.
3. Pennycott, T.W. 1994. Pigeon disease—Results from a Scottish diagnostic laboratory. In Proceedings of the Association of Avian Veterinarians, Orlando, Florida, Association of Avian Veterinarians Publications Office, pp. 231–239.
4. Ritchie, B.W. 1995. Avian Viruses: Function and Control. Lakeworth, Florida, Wingers Publishing, p. 515.

17
Order Psittaciformes (Parrots, Macaws, Conures)

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BIOLOGY

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TAXONOMY AND NATURAL HISTORY

The parrots are an ancient group; the oldest evidence of a parrotlike bird dates from a fossil found in Middle Eocene deposits, with an age of about 30 million years.

The classification of Psittaciformes is in a state of flux; recently the order has been divided into one family with six subfamilies and, later, into three families with six subfamilies. This classification lists the families (with subfamilies) as Loriidae, Cacatuidae (Cacatuinae and Nymphicinae), and Psittacidae (Nestorinae, Micropsittinae, Psittacinae, and Strigopinae).[4,10,18]

In South America there are approximately 118 parrot species, some of them close to extinction, for example, Spix's macaw *Cyanopsitta spixi,* endemic to Brazil. Brazil is the country richest in psittacines, with 72 taxa, followed by Colombia (52), Venezuela (49), and Peru and Bolivia (47) (Table 17.1).

Parrots and their relatives are easy to recognize, despite the variety of forms. They have a unique bill, broad based with a fleshy cere and a chisel-shaped cutting edge, large heads, short necks and legs, and zygodactyl feet. The tibia is short and feathered or bared. In South America, the general color is green, with short or long wings and tails, but in the Pacific region most of the cockatoos are white with a crest.

With a few exceptions, there is no external sexual dimorphism within South American parrots. One exception is the purple-bellied parrot *Triclaria malachitacea;* other exceptions are found in the genera *Pionopsitta, Touit, Psilopsiagon,* and *Forpus.*

Nests are often built in natural holes found in cliffs, termite mounds, live and dead trees, or other plants. The monk parakeet *Myiopsitta monachus* builds communal nests of sticks. Young are altricial, remaining in the nest for a long period, especially nestlings of the large macaws. Eggs are normally white, and the size of the clutch varies among species, ranging from two to seven. The incubation period is around 20–28 days. The female incubates and is fed by the male during incubation.[8]

Many species of parrots pair for life. The bond is reinforced by allopreening, feeding, and male courtship displays during the breeding season.

Most parrots dwell in forest habitats, but some live in open habitats, such as Lear's macaw that inhabits the

TABLE 17.1. Distribution of parrot species in the countries of South America

Genus	Country
Anodorhynchus hyacinthinus	Brazil, Bolivia, Paraguay
Anodorhynchus leari	Brazil
Anodorhynchus glaucous	Argentina, Uruguay, Brazil, Paraguay
Cyanopsitta spixii	Brazil
Ara ararauna	Venezuela, Bolivia, Brazil, Peru, Paraguay, Colombia, Ecuador, Guiana, Surinam
Ara glaucogularis	Bolivia
Ara macao	Venezuela, Colombia, Bolivia, Brazil, Guiana, Ecuador, Peru
Ara chloroptera	Brazil, Peru, Colombia, Venezuela, Guianas, Argentina, Ecuador, Paraguay
Ara militaris	Colombia, Ecuador, Venezuela, Peru, Bolivia, Argentina
Ara ambigua	Colombia, Ecuador
Ara rubrogenys	Bolivia
Ara severa	Colombia, Bolivia, Ecuador, Peru, Brazil, Guiana
Orthopsittaca manilata	Colombia, Guiana, Bolivia, Brazil, Ecuador, Venezuela, Trinidad, Peru
Propyrrhura couloni	Brazil, Peru, Bolivia
Propyrrhura maracana	Brazil, Paraguay, Argentina
Propyrhura auricollis	Argentina, Bolivia, Paraguay, Brazil
Diopsittaca nobilis	Venezuela, Brazil, Bolivia, Peru, Guianas
Aratinga acuticaudata	Venezuela, Colombia, Brazil, Bolivia, Paraguay, Argentina, Uruguay
Guaruba guarouba	Brazil
Aratinga wagleri	Venezuela, Peru, Colombia, Ecuador
Aratinga mitrata	Peru, Argentina, Bolivia
Aratinga erythrogenys	Ecuador, Peru
Aratinga leucophthalmus	Argentina, Guiana, Venezuela, Colombia, Ecuador, Peru, Brazil, Bolivia, Paraguay, Uruguay
Aratinga solstitialis	Guiana, Brazil, Surinam
Aratinga jandaya	Brazil
Aratinga auricapilla	Brazil
Aratinga aurea	Guianas, Bolivia, Argentina
Aratinga weddellii	Colombia, Bolivia, Ecuador, Peru, Brazil
Aratinga cactorum	Brazil
Nandayus nenday	Bolivia, Brazil, Paraguay, Argentina
Leptosittaca branickii	Colombia, Peru, Ecuador
Ognorhynchus icterotis	Ecuador, Colombia
Enicognathus ferrugineus	Chile, Argentina
Enicognathus leptorhynchus	Chile
Cyanoliseus patagonus	Argentina, Uruguay, Chile
Myiopsitta monachus	Bolivia, Argentina, Paraguay, Brazil, Uruguay
Pyrrhura cruentata	Brazil
Pyrrhura frontalis	Brazil, Uruguay, Argentina, Paraguay, Bolívia
Pyrrhura molinae	Brazil, Argentina, Bolivia, Peru
Pyrrhura leucotis	Venezuela, Brazil
Pyrrhura picta	Colombia, Venezuela, Guiana, Brazil, Ecuador, Peru, Bolivia
Pyrrhura perlata	Brazil, Bolivia
Pyrrhura lepida	Brazil
Pyrrhura viridicata	Colombia
Pyrrhura egregia	Brazil, Venezuela
Pyrrhura melanura	Colombia, Ecuador
Pyrrhura orcesi	Ecuador
Pyrrhura albipectus	Ecuador
Pyrrhura rupicola	Peru, Bolivia, Brazil
Pyrrhura calliptera	Colombia
Pyrhura hoematotis	Venezuela
Pyrrhura rhodocephala	Venezuela
Psilopsiagon aymara	Bolivia, Argentina, Chile
Psilopsiagon aurifrons	Peru, Argentina, Chile, Bolivia
Bolborhynchus lineola	Venezuela, Peru, Colombia, Ecuador
Bolborhynchus orbygnesius	Peru, Bolivia
Bolborhynchus ferrugineifrons	Colombia
Forpus conspicillatus	Colombia, Venezuela

(continued)

TABLE 17.1. Distribution of parrot species in the countries of South America—*continued*

Genus	Country
Forpus coelestis	Ecuador, Peru
Forpus xanthops	Peru
Forpus sclateri	Brazil, Bolivia, Ecuador
Forpus passerinus	Guiana, Colombia, Brazil, Trinidad
Forpus crassirostris	Colombia, Argentina, Brazil
Brotogeris tirica	Brazil
Brotogeris versicolurus	Guiana, Brazil, Paraguay, Bolivia, Argentina
Brotogeris pyrrhopterus	Ecuador, Peru
Brotogeris jugularis	Colombia, Venezuela
Brotogeris cyanoptera	Colombia, Ecuador, Brazil, Peru, Bolivia, Venezuela
Brotogeris chrysopterus	Guiana, Brazil
Brotogeris sanctithomae	Colombia, Bolivia
Nannopsittaca panychlora	Venezuela
Nannopsittaca dachilleae	Peru, Bolivia
Pionites melanocephala	Peru, Colombia, Guiana, Ecuador, Venezuela, Brazil
Pionites leucogaster	Brazil, Bolivia
Touit batavica	Guiana, Venezuela, Trinidad
Touit huetii	Colombia, Venezuela, Guiana, Ecuador, Bolivia
Touit dilectissima	Colombia, Ecuador, Venezuela
Touit purpurata	Colombia, Ecuador, Peru, Brazil, Venezuela, Guiana
Touit melanonotus	Brazil
Touit surda	Brazil
Touit stictoptera	Colombia, Peru
Pionopsitta pyrilia	Colombia, Venezuela, Ecuador
Pionopsitta haematotis	Colombia
Pionopsitta pulchra	Colombia, Ecuador
Pionopsitta barrabandi	Venezuela, Colombia, Ecuador, Peru, Bolivia, Brazil
Pionopsitta vulturina	Brazil
Pionopsitta caica	Venezuela, Guiana, Brazil
Pionopsitta pileata	Brazil, Paraguay, Argentina
Graydidascalus brachyurus	Brazil, Ecuador, Guiana, Colombia, Ecuador, Peru
Hapalopsittaca melanotis	Peru, Bolivia
Hapalopsittaca amazonina	Colombia, Venezuela
Hapalopsittaca fuertesi	Colombia
Hapalopsittaca pyrrhops	Ecuador, Peru
Pionus menstruus	Bolivia, Brazil, Colombia, Venezuela, Trinidad, Guiana, Ecuador, Peru
Pionus fuscus	Venezuela, Guiana, Brazil, Colombia
Pionus maximiliani	Brazil, Bolivia, Argentina
Pionus tumultuosus	Venezuela, Colombia, Ecuador, Peru, Bolivia
Pionus sordidus	Venezuela, Bolivia, Colombia, Ecuador, Peru
Pionus chalcopterus	Venezuela, Guiana, Brazil
Amazona rhodocorytha	Brazil
Amazona tucumana	Argentina,Bolívia
Amazona pretrei	Brazil
Amazona autumnalis	Colômbia,Ecuador,Venezuela,Brazil
Amazona dufresniana	Venezuela, Guianas,Brazil
Amazona brasiliensis	Brazil
Amazona festiva	Venezuela, Colombia, Ecuador, Brazil, Peru
Amazona vinacea	Brazil, Paraguay, Argentina
Amazona xanthops	Brazil, Bolivia
Amazona barbadensis	Venezuela
Amazona aestiva	Brazil, Bolivia, Paraguay, Argentina
Amazona ochrocephala	Brazil, Bolivia
Amazona amazonica	Brazil, Colombia, Ecuador, Peru, Bolivia, Venezuela, Guiana, Trinidad
Amazona mercenaria	Venezuela, Bolivia
Amazona kawalli	Brazil
Amazona farinosa	Brazil, Ecuador, Guiana, Peru, Bolivia

"caatinga" in northern Brazil, and *Cyanoliseus patagonus* in open areas of Chile, Uruguay, and Argentina. Some species are found in close association with certain plants or types of forests, such as the red-spectacled Amazon *Amazona pretrei* with the araucaria tree and the red-tailed Amazon *Amazona brasiliensis* with the Atlantic forest in southern Brazil. In South America most species are diurnal; some are active at dawn and after sunset, an example being Lear's macaw that arrives in its overnight roost after sunset.

The diet of the majority of parrots is comprised of seeds and fruits of various kinds, which are procured in the treetops or on the ground.[4] They also feed on flowers, nectar, leaves, and buds, depending on individual's adaptations (e.g., the size of the bill and tongue). A wide range of parrots also visits clay banks, known as "barreiros", to ingest mineral elements that neutralize the toxic effects of poisons ingested with plant foods. Coconuts are consumed by many species of parrots and macaws.

South American parrots do not migrate, but some look for food far from their roost sites. Parrots are awake before sunrise, feeding during the first hours of the morning. They spend most of the day in social interaction or resting, feeding again in the late afternoon just before returning to the roosting area.

DISTRIBUTION

Most parrots are distributed throughout the tropical zones of the earth, but some species are found in cold areas of Patagonia, Argentina, and Chile. The largest number of species is in the Southern Hemisphere, with the most marked speciation in the New World with 45 taxa and 25 genera. In South America, the richest zone is the Amazon forests of Brazil, Colombia, Venezuela, and Peru with 72, 52, 49, and 47 taxa, respectively.[4]

EXPLOITATION AND TRADE

During past centuries, many species of parrots have suffered from human activities such as capturing live birds or destroying their habitats. Parrots are one of the most attractive birds to humans; many of them are kept as pets and develop mutual affection. Because of this and their function as a status symbol among many aviculturists, many species are threatened and some are close to extinction. Parrots from many regions are found in zoos and private collections, often with no attempt being made for comprehensive study of methods of conservation or increasing the populations of wild species in severe decline.

The estimated volume of international trade in Neotropical parrots represents only a fraction of the total number of birds removed from the wild. From 1982 to 1988 about 1.8 million parrots were taken from the neotropics, most being exported to the United States and Europe.[16]

STUDIES OF SOUTH AMERICAN PARROTS

Many studies of South American parrots have been conducted during past decades to determine their distribution, biology, and ecology and also to discover the real status of species in the wild. Many studies within the family Psittacidae are continuing today.

For the genus *Amazona,* surveys done in the field are the basis for the strategy for conservation. The red-tailed Amazon parrot has been studied intensively from 1982 to 1999.[12] The survey has determined its current distribution, general behavior, movements, reproduction, and diet. During the field work, daily migration from the continent to overnight communal roosting sites on islands of Paranaguá Bay was discovered. The population can be estimated through a regular census of the birds at those sites and can be monitored to evaluate increase or decline.

Another study of the red-spectacled Amazon parrot in southern Brazil involved many censuses in order to estimate the size of populations in the state of Rio Grande do Sul and Santa Catarina. The first studies were conducted by a biologist, Flávio Silva,[15] with a team of students and birdwatchers, and later by Nigel Varty,[17] with a team of Brazilian ornithologists. This parrot congregates in large flocks of about 15,000 birds.

The vinaceous Amazon parrot *Amazona vinacea* is a widely distributed species in Brazil, Argentina, and Paraguay. Threatened by habitat loss and capture, little is known about the biology and ecology of this species, although a survey to determine distribution and some other aspects was conducted in the state of Parana. Ligia Mieko Abe, a biologist of the Museum of Natural History Capão da Imbuia, is currently studying this Amazon parrot, after a large flock was discovered in the metropolitan area of the city of Curitiba. Nests were found from September to February, when nestlings fledged (L.M. Abe, personal communication, 1999). The usual habitat of the vinaceous Amazon parrot is the araucaria forests, where it eats the seeds of this tree, and the coastal Atlantic forest.

The yellow-shouldered *Amazona barbadensis* was studied on Margarita Island in Venezuela by Franklin Rojas-Suárez and colleagues,[11] seeking information about its population and reproductive biology. The natural history of the golden conure *Aratinga guarouba,* endemic to Brazil, is being studied by the Brazilian ornithologist Carlos Yamashita (personal communication).

Glayson Benke[1] conducted research on the purple-bellied parrot *Triclaria malachitacea* in Rio Grande do Sul in southern Brazil. He discovered a small group in a remnant of araucaria forest and studied their movements and ecology. This parrot is found deep inside the forests and is not easy to see, but may be recognized by its singular voice. The diet of the scaly-headed parrot *Pionus maximiliani* was studied by Mauro Galletti[5] in the semideciduous forests of southern Brazil. The blue-fronted *Amazona aestiva* has been studied for years in Argentina, and in Brazil a reintroduction program is in development in the state of Mato Grosso by biologist Glaucia Seixas (personal communication).

Field studies are the basis for conservation programs in situ and ex situ. Environmental management techniques developed by such programs may help avoid extinction of parrots all over the world.

REFERENCES

1. Benke, G. 1999. Ecology and Conservation of the Purple-bellied Parrot *Triclaria malachitacea* in Remaining Forests in Rio Grande do Sul. Masters thesis, Universidade Federal de São Carlos.
2. Bianchi, C.A.C. 1998. Biologia reprodutiva da arara-canindé (*Ara ararauna,* Psittacidae) no Parque Nacional das Emas, Goiás. Masters thesis, Brasilia, Universidade de Brasilia.
3. Brandt, A.; and Machado, R. 1990. Área de alimentação e comportamento alimentar de *Anodorhynchus leari.* Ararajuba, Rio de Janeiro, 1:57–63.
4. Forshaw, J.M. 1977. Parrots of the World. Neptune, New Jersey, T.F.H. Publications.
5. Galletti, M. 1993. Diet of the scaly-headed parrot (*Pionus maximiliani*) in a semidecidual forest in southern Brazil. Biotropica 25:419–425.
6. Guedes, N.M.R. 1993. Biologia reprodutiva da arara-azul (*Anodorhynchus hyacinthinus*) no Panatanal—MS, Brasil. Masters thesis, São Paulo, Universidade de São Paulo.
7. Guedes, N.M.R.; and Harper, L.H. 1995. The hyacinth macaw in the pantanal. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, Raintree Pub., pp. 394–421.
8. Juniper, T.; and Parr, M. 1998. Parrots: A Guide to Parrots of the World. New Haven, Yale University Press.
9. Munn, C.A.; Thomsen, J.B.; and Yamashita, C. 1990. The hyacinth macaw. In W.J. Chadler, ed., Audubon Wildlife Report. New York, Academic Press, pp. 404–419.
10. Peters, J.L. 1937. Check List of Birds of the World, Vol. 3. Cambridge, Massachusetts, Harvard University Press.
11. Rojas-Suárez, F. 1994. Reproductive biology of yellow-shouldered parrot *Amazona barbadensis* (Aves: Psittaciformes) in Macanao peninsula, Nueva Sparta State. In G. Morales, I. Novo, D. Bigio, A. Luy, and F. Rojas-Suarez, eds., Biología y Conservación de los Psitácidos de Venezuela, Caracas, pp. 73–81.
12. Scherer-Neto, P. 1989. Contribuição à Biologia do Papagaio-de-cara-roxa *Amazona brasliensis* (Linnaeus, 1758) (Psittacidae, Aves). Masters thesis, Universidade Federal do Paraná.
13. Sick, H. 1979. Découverte de la patrie de l'Ara de Lear Anodorhynchus leari. Alaud. 47(1):59–63.
14. Sick, H. 1985. Ornitologia brasileira: uma introdução. Brasilia, Universidade de Brasília, 2 Vol., p.828.
15. Silva, F. 1981 . Contribuição ao conhecimento da biologia do papagaio-charão *Amazona pretrei* (Temminck, 1830) (Psittacidae,Aves). Iheringia (Zoology) 58:79–85.
16. Thomsen, J.D.; and Brautigam, A. 1991. Sustainable use of neotropical parrots. In J.G. Robinson and K.H. Redford, eds., Neotropical Wildlife Use and Conservation. Chicago, University of Chicago Press, pp. 359–379.
17. Varty, N.; Benke, G.; Bernadini, L.M.; Cunha, A.S.; Dias, E.V.; Fontana, D.L.; Guadagnin, A.K.; Kindel, A.; Kindal, E.; Raymundo, M.M.; Richter, M.; Rosa, A.O.; Tostes, C.A.S. 1994. Conservação do Papagaio-charão *Amazona pretrei* no Sul do Brasil: Um Plano de Ação Preliminar. Divulgações do Museu de Ciências e Tecnologia. UBEA/PUCRS no. 1. Porto Alegre, EDIPUCRS.
18. Verheyen, R. 1956. Analise du potentiel morphologique et project d'une nouvelle classification des Psittaciformes. Bulletin Institute Royal Science Natural, Belgium 32(55):1–54.

THE LARGE MACAWS

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Within the family of the Psittacidae there are three genera of macaws: *Anodorhynchus* (3 species), *Cyanopsitta* (1 species) and *Ara* (15).[7] Macaws are beautifully colored, exuberant, and highly sought after for trade and breeding as pets, which is precisely the principal factor for the declining natural population, together with destruction of its natural habitat.

The genus *Anodorhynchus* is comprised of three species of large macaws that have fusiform bodies, a gradated tail, and a predominantly blue color, with yellow coloring around the mandible and periorbital region. In contrast to the other large macaws, the lores and facial area are not bare. The jaw is movable, articulated in the skull, which increases the strength of the heavy curved beak that is used to crack hard seeds.[7,19]

The hyacinth macaw *A. hyacinthinus* is the largest member of the Psittacidae family. The species' situation is the best in the genera although there are more individuals in captivity today than in the natural state. In 1987, the population in the wild was estimated at 3,000 individuals.[14] The situation of the species is particularly worrying because of the reduced population and limited distribution and because it is highly specialized in feeding and reproduction sites.

The first long-term work with free-living hyacinth macaws began in 1990, by Neiva Maria Robaldo Guedes in the UNIDERP Hyacinth Macaw Project (Projeto Arara Azul/UNIDERP). These studies are continuing with the objective of obtaining information on the biology and ecology of the species and of developing management alternatives. In the southern pantanal region of Mato Grosso do Sul State, the area in which the population density is highest, 95% of the nests were located in a single species of tree, the manduvi (*Sterculia apetala*), which has a soft core that is easily hollowed out. These trees are generally old and separated from the surrounding vegetation on the wooded edge of rivers or in isolated wooded clumps of forest. In the mountain chains surrounding the pantanal plain, hyacinth macaws make nests in crevices in the rocky walls.

The hyacinth macaw lays from one to three eggs, on average two, at intervals. The female incubates the eggs, and is fed by the male. Incubation takes from 28 to 30 days. Predators take 14–37% of the eggs, and the rate of hatching is 90%, depending on climatic and environmental conditions. Reproductive potential has grown each year and reached two eggs per female in 1997 and 1998.

The average weight of chicks at hatching is 31.6 g and average length 82.7 mm. Chicks grow rapidly, remaining in the nest for approximately 107 days. Parents continue to feed fledglings. In 1997, 44 chicks from 36 pairs fledged, and in 1998, 35 chicks from 36 pairs. Mortality of chicks is 7–35%. Chicks are preyed upon by ants *(Solenopis* spp.), ticks (*Ornithonyssus* spp.), fly larvae (*Philornis* spp.), birds (toucans, hawks, and owls), and mammals (*Tayra barbara*). Up to 50% of hyacinth macaw chicks that hatch second die. When the second chick hatches more than 5 days after the first, it may be unable to compete for food with the first and dies of starvation.

Before leaving the nest on their first flight, a stainless steel ring is attached to the chicks and a blood sample is collected for DNA analysis and sexing. According to Miyaki et al. (1999, in preparation), of 152 chicks that were sexed in the period from 1992 to 1998, the numbers of males and females were almost equal, with a small, insignificant deviation favoring females in some years. Using the DNA fingerprinting technique, the authors determined that the genetic variability in the hyacinth macaw population of the pantanal region is approximately 65%.

Hyacinth macaws are highly conspicuous, sedentary, and show a certain fidelity to nesting sites. Nonreproducing individuals are highly social and gather in flocks in both the feeding and dormitory areas.

During the breeding season there is great interspecific competition for tree cavities. This competition, together with the destruction of potential nest sites through deforestation or burning of the forest, which can reach 5% per annum, becomes a factor limiting hyacinth macaw reproduction in the pantanal region. Between 1997 and 1998, 105 artificial nests were installed in the southern pantanal. The hyacinth macaws used more than 50% of the nests each year, although only four were successful in raising chicks to fledging. Indirectly, a positive management success resulted from occupation of the artificial nests by other species that normally compete with macaws for natural nest sites, leaving more natural sites available for hyacinth macaws, which were consequently more successful in the regions in which the artificial nests were installed.

Anodorhynchys leari, without known distribution for over a century, was rediscovered in 1978 in the Raso da Catarina,[18] northeastern area of the State of Bahia. Endemic to this region, it is threatened with extinction as a result of being hunted for the domestic and international pet bird trade and the disappearance of its natural habitat, areas of small thorny stunted trees, the caatinga.

Studies on the feeding area habits of *A. leari* concluded that the availability of food sources was the principal factor limiting population growth.[5] The Conservation Committee and Brazilian Institute for the Environment and Natural Renewable Resources (IBAMA), coordinated by Luiz Sanfilippo of the São Paulo Zoological Gardens, have been recently carrying out studies that include a population count, monitoring nests, behavior observation, food supplies, both long and short term, and information about *A. Leari's* feeding area. The principal food source of Leari's macaw is nuts from the licuri palm tree (*Syagrus coronata*). Although this palm tree once was common on the high plains of the State of Bahia, it is becoming rare as a result of land clearance, burning, and young plants being trampled by cattle (Luiz Sanfilippo, unpublished work, 1999). As a result, the macaws must fly long distances[5] in search of food and sporadically use other sources such as corn (*Zea mays*), flowers of the sisal plant (*Agave* spp.), umbu (*Spondias tuberosa*) and mucumã (*Dioclea* spp.), and seeds of another tree (*Jatropha pohliana*) (Luiz Sanfilippo, unpublished work, 1999).

According to Barros (unpublished work, 1999), Spix's macaw *Cyanopsitta spixii* is the most endangered species in the world. With only one example known in the wild and around 40 individuals in captivity around the world, the future for Spix's macaw is bleak. IBAMA coordinates an international committee for the conservation of Spix's macaw, conducted in the field by Yara Barros. This work aims to monitor the last wild specimen that lives along the streams and groves of caraiba trees (*Tabebuia caraiba)* in the Curaça region of the State of Bahia. This male is paired with a female of the *Propyrrhura maracana* species. According to Barros (personal communication, 1999), who also monitors nesting sites of *P. maracana,* in the breeding season the mixed pair try to nest and even lay eggs. This is one of the most critical conservation programs in Brazil, because it seeks to establish strategies for capturing birds and studies the possibilities for future reintroduction.

REFERENCES

1. Bianchi, C.A.C. 1998. Biologia Reprodutiva da Arara-Canindé (*Ara ararauna,* Psittacidae) no Parque Nacional das Emas, Goiás. Master thesis, Universidade de Brasília, p 69.
2. Bjork, R.; and Powell, G. 1995. Buffon's macaw: Some observations on the Costa Rican population, its lowland forest habitat and conservation. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, California, Raintree Publications, pp. 387–392.
3. Boussekey, M.; Saint-Pie, J.; and Morvan, O. 1995. Food and feeding of the red-fronted macaw in the Rio Caine Valley, Bolívia. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, California, Raintree Publications, p. 386.
4. Boussekey, M.; Saint-Pie, J.; and Morvan, O. 1997. Preliminary observation of the blue-throated macaw *Ara glaucogularis* in the department of Beni (Bolivia). Papageienkunde, Bretten, (1):S151–156.
5. Brandt, A.; and Machado, R. 1990. Área de alimentação e comportamento alimentar de *Anodorhynchus leari.* Ararajuba 1:57–63.
6. Collar, N.J.; Gonzaga, L.P.; Krabbe, N.; Madroño Nieto, A.; Naranjo, L.G.; Parker, T.A., III; and Wege, D.C. 1992. *Anodorhynchus.* In Threatened Birds of the Americas: The ICBP/IUCN Red Data Book, 3rd Ed. Cambridge, England, pp. 241–65.
7. Forshaw, J.M. 1977. Parrots of the World, 2nd Ed. Melbourne, TFH Publications, p. 584.
8. Guedes, N.M.R. 1993. Biologia reprodutiva da arara-azul (*Anodorhynchus hyacinthinus*) no Pantanal—MS, Brasil. Master thesis, Universidade de São Paulo, p. 122.
9. Guedes, N.M.R.; and Harper, L.H. 1995. The hyacinth macaw in the pantanal. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, California, Raintree Publications, pp. 394–421.
10. Jordan, O.C.; and Munn, C.A. 1993. The first observations of the blue-throated macaw in Bolívia. Wilson Bulletin 105(4):694–695.
11. Lanning, D.V. 1991. Distribution and breeding biology of the red-fronted macaw. Wilson Bulletin 103(3):357–365.
12. Marineros, L.; and Vaughan, C. 1995. Scarlet macaws in Carara. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, California, Raintree Publications, pp. 445–467.
13. Munn, C.A. 1992. Macaw biology and ecoturism, or "When a bird in the bush is worth two in the hand." In S.R. Beissinger and N.F.R. Snyder, eds., New World Parrots in Crisis: Solutions from Conservation Biology. Washington, D.C., Smithsonian Institution Press, pp. 47–72.
14. Munn, C.A.; Thomsen, J.B.; and Yamashita, C. 1990. The hyacinth macaw. In W.J. Chadler, ed., Audubon Wildlife Report. New York, Academic Press, pp. 404–419.
15. Nycander, E.M.; Blanco, D.H.Z.; Holle, K.M.F.; Campo, A.; Munn, C.A.; Moscoso, J.L.G.; and Ricalde, D.R. 1995. Manu and tambopata. In J. Abramson, B.L. Speer, and J.B. Thomsen, eds., The Large Macaws: Their Care, Breeding and Conservation. Fort Bragg, California, Raintree Publications, pp. 423–443.
16. Pinho, J.B. 1998. Aspectos comportamentais da Arara-Azul (*Anodorhynchus hyacinthinus*) na localidade de Pirizal, Município de Nossa Senhora do Livramento — Pantanal de Poconé. Master thesis, Universidade Federal de Mato Grosso, p. 78.
17. Reintjes, N.; Kunz, B.; and Blomenkamp, A. 1997. Situation of the great green macaw *Ara ambigua guayaquilensis* in West Ecuador. Papageienkunde, Bretten (1):S141–150.
18. Sick, H. 1979. Découverte de la patrie de l'Ara de Lear *Anodorhynchus leari.* Alauda 47(1):59–63.
19. Sick, H. 1985. Ornitologia Brasileira: Uma Introdução. Brasília, Universidade de Brasília, p. 828.
20. Yamashita, C.; and Barros, Y.M. 1997. The blue-throated macaw *Ara glaucogularis:* Characterization of its distinctive habitats in savannahs of the Beni, Bolivia. Ararajuba 5(2):141–150.

NUTRITION

Aulus Cavalieri Carciofi

There is great diversity of form and natural feeding habits among the more than 344 species that constitute the Psittacidae family. In this chapter, nutritional needs will be discussed in a general manner, but it is important to consider the particular requirements of each species and make the necessary adaptations.

Free-living psittacines consume berries and other fruit, flowers, plant sprouts, legumes, insects, larvae, and seeds. Pollen and nectar are also a significant part

FIGURE 17.1. Offspring of blue-fronted Amazon (*Amizona aestiva*) in poor nutritional condition. Because owners generally do not have the knowledge of correct feeding of these animals, nutritional disorders in psittacines are very common. (Courtesy of Karin Werther).

of the diet of species that have anatomical adaptations that facilitate prehension and use of these foods. In captivity, psittacines were initially classified as granivores, which led to the false belief that seeds could totally satisfy their nutritional needs.

Much of what is known about feeding psittacines in captivity results from diets and menus that have proved successful more by empirical use than by data resulting from scientific studies.[11] The lack of knowledge of their nutritional needs[11,14,15] and of the efficiency with which they use the foods offered has led to many management mistakes and, therefore, to a high incidence of nutritional deficiencies,[4,14,16] currently one of the most prevalent problems seen in bird clinics[8,10,15] (Figure 17.1).

NUTRITIONAL DISEASES

Undernourished birds lack immunological capacity and are more susceptible to infection and systemic diseases[14,15] than those that are well nourished, and reproductive capacity is reduced. In 466 necropsies of psittacines, 85% of the aspergillosis cases were correlated to squamous metaplasia of the salivary glands, caused by chronic vitamin A deficiency.[4]

It is important to understand that birds suffering from the same nutritional deficiency may present different signs. For example, riboflavin deficiency in chickens is manifested by curled-toe paralysis, whereas in cockatiels (*Nymphicus hollandicus*) achromatosis of the primary feathers is seen, which, in turn, is caused by lysine deficiency in the chicken.[10]

One of the major nutritional diseases in adult psittacines is caused by vitamin A deficiency. In histological studies involving 112 psittacines, 55% of the cockatoos, 48% of the African gray parrots, and 51% of the Amazons presented alterations of the salivary glands, typical of deficiency of this vitamin.[4] This great prevalence results from the fact that these birds are often fed with large quantities of seeds, which are generally poor in vitamin A, particularly sunflower seeds, which are eagerly consumed. Infections by such parasites as *Giardia* spp., *Capillaria* spp., and coccidia predispose birds to vitamin A deficiency, as the capacity of enterocytes to biotransform ingested β-carotene into vitamin A is decreased. Clinical signs of vitamin A deficiency include reduction of cellular and humoral immunity (making the bird more susceptible to infections of the respiratory tract), coryza, sinusitis, reproductive problems, difficulty in peeling and swallowing food, feather picking, plantar pododermatitis, uric gout caused by lesions in the renal tubuli, and difficulty in excretion of uric acid (Figure 17.2).[13]

Protein deficiency, especially of the amino acid arginine, causes poor feathering, the appearance of transversal lines forming a 90° angle with the feather axis (called stress lines), incomplete molting; pinlike feathers (without barbs), and easily breakable wing and tail feathers.[15] Lysine deficiency, on the other hand, results in pigmentation problems, blue and green feathers

FIGURE 17.2. Necropsy of blue-fronted Amazon (*Amizona aestiva*) showing generalized kidney gout, one of the symptoms related to hypovitaminosis A. (Courtesy of Karin Werther).

FIGURE 17.3. Blue-fronted Amazon (*Amizona aestiva*) presenting layers on the beak because of the inability of keratinized tissue to grow normally, which is a common symptom of protein or amino acid deficiency.

becoming yellow or black. Poor nutrition is also one of the causes of the feather-picking syndrome (birds remove feathers from their own body).[7]

Other diseases of nutritional origin seen in psittascine clinics are: iodine deficiency, which results in goiter or hypothyroidism; calcium, phosphorus, and vitamin D deficiencies, which cause changes in the angle of the long bones, pathological fractures, crooking of the beak resulting in malocclusion, and tetanic convulsions; and fatty liver syndrome. Birds often become obese as a result of a high-energy diet, little physical activity (with a consequent low-energy expenditure), and lack of occupation, because when the bird gets bored it ingests food in excess.

Some diseases are caused by a combined deficiency of several nutrients. The surface of the skin, beak, feathers, and nails becomes dry and scaly, the beak grows long and crooked, and toenails and beak become layered, caused by abnormal growth of the keratinized tissue (Figure 17.3), and the skin of the legs and feet becomes thickened and crusted. These are signs of deficiency of vitamin A, protein, biotin, niacin, pantothenic acid, zinc, and manganese.

TABLE 17.2. Nutrient profile recommendations for maintenance of adult psittacines

Nutrient	Minimum	Maximum	Nutrient	Minimum	Maximum
Gross Energy, kcal/g	3200	4200	Pyridoxine, ppm	6.0	
Linoleic Acid, %	1.0		Riboflavin, ppm	6.0	
Crude Protein, %	12.0		Thiamine, ppm	4.0	
Arginine, %	0.65		Vitamin B_{12}, ppm	0.01	
Lysine, %	0.65		Minerals		
Methionine, %	0.30		Calcium, %	0.30	1.20
Methionine + Cystine, %	0.50		Phosphorus,[c] total %	0.30	
Threonine, %	0.40		Calcium: Total phosphorus	1:1	2:1
Vitamins			Chlorine, %	0.12	
Vitamin A, IU/kg	8000		Magnesium, %	0.06	
Vitamin D_3, ICU/kg	500.0	2000	Potassium, %	0.40	
Vitamin E, ppm	50.0		Sodium, %	0.12	
Vitamin K, ppm	1.0		Copper, ppm	8.0	
Biotin, ppm	0.25		Iodine, ppm	0.40	
Choline, ppm	1500		Iron, ppm	80.0	
Folic acid, ppm	1.50		Manganese, ppm	65.0	
Niacin, ppm	50.0		Selenium, ppm	0.10	
Pantothenic acid, ppm	20.0		Zinc, ppm	50.0	

Source: See reference 1.

NUTRITIONAL NEEDS

The pet food regulations of the Association of American Feed Control Officials (AAFCO) have been developed by an expert committee for companion and exotic bird nutrition. They develop suggested nutrient profiles and are an important reference for establishment and evaluation of diets for psittacines (Table 17.2).

The recommendations in Table 17.2 are conservative, being based on extrapolations of the National Research Council requirements for poultry. They are not yet the final word on nutritional needs of psittacines and should be adapted for the different species and, most importantly, more research should be conducted.

Requirements for reproduction and feather molting were not recommended by AAFCO. The protein requirements for psittacine growth were studied by Roudybush and Grau,[12] who established 20% crude protein (CP) and 0.8% lysine as minimums for growth for cockatiels. Carciofi et al.,[2] using a purified diet (corn starch, cellulose, soy oil, isolated soy protein, minerals, and vitamins) and 40 birds, concluded that 18% CP is the minimum necessary for optimal growth of the blue-fronted Amazon (*Amazona aestiva*) and 23% CP the minimum for the best development of the beak, measured by the length of the culmen.

Little data are available on metabolizable energy (ME) needs for maintenance of psittacines. For other nonpasserine birds, values between 130 and 160 kcal ME/kg live weight$^{0.75}$ per day have been proposed. In a series of experiments on food consumption of the turquoise-fronted parrot, Carciofi et al.[2] found that in Brazil a daily ingestion (mean of 10 days) of 138 kcal ME/kg$^{0.75}$ in the winter and 107 kcal ME/kg$^{0.75}$ in the summer was needed. Drepper et al.[5] found 200.8 kcal ME/kg$^{0.75}$ per day was needed for budgerigars (*Melopsittacus undulatus*), a value significantly higher that the requirement proposed previously. This is one of the few reference values for small psittacines.

FEEDS AND FEEDING

To know the nutritional composition of food, the nutrients supplied and deficiencies of several groups of ingredients is a fundamental step for the formulation of diets. Table 17.3 shows the approximate chemical composition of some seeds.

Whether to feed psittacines homemade mixed-food diets or commercial pelleted or extruded diets is an important choice. Wide individual variation in selection and consumption of foods is seen among psittacines. A frequent mistake is the calculation of nutrients supplied based on the amount fed rather than on the amount ingested and/or absorbed.[9] In ad libitum diets based on seeds, fruit, and vegetables, seed ingestion normally represents more than 70–85% of ingested dry matter. Total fruit consumption hardly contributes more than 10% of protein or energy, or 4–5% of ingested calcium and phosphorus.[2]

In homemade mixed-food diets there is no effective control of nutrient ingestion by the birds, and seeds become the major part of the food consumed. Data in Table 17.2 show that seeds are deficient in protein

TABLE 17.3. Chemical composition of some seeds used for psittacines

Nutrient[a]	Sunflower	Oat Groats	White Millet [d]	Safflower [c]	Peanut
Dry matter, %	94.70	89.74	—	94.40	92.87
Crude energy, kcal/g	7.65	3.97			6.69
Ether extract, %	59.47	2.59	3.50	40.70	46.69
Linoleic acid. %	34.46 [c]	—	0.24	29.75	16.63 [c]
Crude fiber, %	3.67	1.21	—	2.60	2.58
Crude protein, %	26.34	10.40	11.50	17.10	27.77
Arginine, %	2.54 [c]	1.02 [c]	0.34	1.85	3.70 [c]
Lysine, %	0.99 [c]	0.61 [c]	0.21	0.57	1.06 [c]
Methionine, %	0.52 [c]	0.24 [c]	0.31	0.27	0.28 [c]
Methionine + cystine, %	1.0 [c]	0.47 [c]	0.50	0.56	0.63 [c]
Threonine, %	0.98 [c]	0.51 [c]	0.31	0.55	0.80 [c]
Nitrogen-free extract %	1.44	73.75	71.20	—	13.51
Calcium, %	0.07	0.04	0.015	0.08	0.04
Phosphorus, total %	0.67	0.36	0.26	0.68	0.32
Magnesium, %	0.28	0.13 [c]		0.17	0.19 [c]
Iron, ppm	60.67	84.0 [c]		89.0	35.0 [c]
Copper, ppm	23.67	7.0 [c]		19.0	11.0 [c]
Manganese, ppm	39.33	32.0[c]		34.0	12.0 [c]
Zinc, ppm	70.0	—		65.0	35.0 [c]
Vitamin A [b], IU/kg	83.0 [c]	1,470 [d]	530.0	0	0 [c]
Vitamin E, IU/kg	—	17.0 [c]		—	98.0 [c]

[a] Values refer to peeled seed, dry matter.
[b] Calculated from carotene.
[c] From reference 14.
[d] From reference 6.

(groat and millet), calcium, total phosphorus, iron, manganese, and vitamin A. They are also deficient in some amino acids that are essential for growth and reproduction, such as methionine, arginine, and lysine, and in the water-soluble vitamins riboflavin, pantothenic acid, niacin, vitamin B_{12}, and choline. The selenium and iodine levels of seeds vary greatly according to their presence in the soil.[14]

Commercial pelleted or extruded diets formulated by reliable firms are practical and safe. These rations are mixtures of seeds, protein sources, amino acids, vitamins, and minerals, ground and processed, guaranteeing good digestibility and a balanced consumption of essential nutrients. Several experiments have proved that these diets are effective when they are adequately formulated. The fertility rates of birds fed with seeds were elevated from 40 to 80% or more, and fledging percentage rates of chicks was raised from 66% to more than 90% when the diet was changed to pelleted or extruded complete diets.

For the proper use of homemade mixed-food diets, the nutritionist must create mechanisms to restrict seed ingestion, increase fruit and vegetable consumption, and supplement the diet with amino acids, minerals, and vitamins. Some suggestions for improvement of homemade mixed-food diets follow.

Sunflower seeds and peanuts contain high levels of protein and are also high in fat; therefore, they yield an unfavorable energy/protein ratio. Other sources of protein must be included, such as cheese, meat, or cooked legumes (soybeans, kidney beans, peas), which have less fat.

The germination process results in chemical changes in seeds, with a reduction of the levels of carbohydrates and fat and an increase in levels of water, vitamin C, and B complex vitamins. For sprouting, small seeds, sunflower seeds, and legumes (lentils, soy beans, or peanuts) are washed and soaked for 4, 6, and 24 hours, respectively. They are then washed, drained, and placed in plastic vessels for 24 hours to germinate. They must be rinsed with running water at 6–8 hour intervals. The resulting sprouts should be fed to the birds as soon as the first root becomes visible. Sprouts may be kept in a refrigerator for several days.

Bone meal, calcium carbonate, or ground oyster shell must be constantly available to psittacines. In many situations this may effectively supply needed minerals; however, it is difficult to determine whether or not consumption of these items has been sufficient, even though the bird plays with them, or if they are being consumed in excess, which may also lead to health problems. Mineral supplementation by means of prepared feeds (cooked seed mix, cooked cake, bread paste) is a better guarantee.

Vitamins must be sprayed onto or mixed into the food consumed the most (but not over seeds in shells that are peeled before being ingested). Dilution in water is not recommended, because the vitamins may not be completely ingested. In addition, vitamins oxidize quickly, especially in the presence of chloride, fluoride, or inorganic iron, losing their biological activity.

A possibly adequate diet may be composed of cooked seeds, such as brown rice, kidney beans, peas, corn, and soybeans in equal parts, supplemented with 1 g tricalcium phosphate and 0.10 g of salt per 100 g of the cooked mixture. Trace mineral and vitamin supplements must be added to this substance or some commercial products destined for birds. The amount to be added may be calculated based on its content of vitamin A. For each 100 g of the cooked mixture, 400 IU vitamin A are needed.

REFERENCES

1. Association of American Feed Control Officials. 1988. New rules for feeding pet birds: Nutrition expert panel review. Feed Management 49(2):23–25.
2. Carciofi, A.C.; Prada, F.; and Sanfilippo, L.F. 1999. Unpublished data.
3. Dorrestein, G.M.; Buitelarr, M.N.; Van Der Hage, M.H.; and Zwart, P. 1985. Evaluation of a bacteriological and mycological examination of psittacine birds. Avian Diseases 29(4):951–962.
4. Dorrestein, G.M.; Zwart, P.; Van Der Hage, M.H.; and Schrijver, J. 1987. Metaplastic alterations in the salivary glands of parrots in relation to liver vitamin A levels. In Proceedings of the 1st International Conference on Zoological and Avian Medicine. pp. 69–73.
5. Drepper, K; Menke, H.; Schulze, G.; Wachter-Vormann, U. 1988. Untersuchungen zum protein und energiebedanf adulter welleusittiche (*Melopsittacus undulatus*).in käfighaltung. [Studies on the protein and energy requirement of adult budgerigars housed in cages]. Kleintierpraxis 33(2):57–62.
6. Earle, K.E.; and Clarke, N.R. 1991. The nutrition of the budgerigar (*Melopsittacus undulatus*). Journal of Nutrition 121(11S):186S–192S.
7. Gould, W.J. 1995. Caring for pet birds skin and feathers. Veterinary Medicine 90(1):53–63.
8. Himmeltein, S.; and Bernstein, K. 1978 Clinical aspects of nutritional secondary hyperparathyroidism in cage birds. Veterinary Medicine: Small Animal Clinician 73(6):761–763.
9. Kamphues, J. 1993. Ernährungsbedingte störungen in der ziervogelhaltung—Ursachen, einflüsse und aufgaben [Nutrition-related disturbances in cage birds]. Monatshefte für Veterinär Medizin 48(2):85–90.
10. Kollias, G.V. 1995 Diets, feeding practices, and nutritional problems in psittacine birds. Veterinary Medicine 90(1):29–39.
11. Nott, H.M.R.; and Taylor, E.J. 1994. Advances in our understanding of the nutrition of pet birds. Wiener Tierarztliche Monatsschrift 85(5):135–140.
12. Roudybush, T.E.; and Grau, C.R. 1991. Cockatiel (*Nymphicus hollandicus*) nutrition. Journal of Nutrition 121(11S):S206.
13. Ryan, T.P. 1988 Vitamin A and its deficiency in birds. Companion Animal Practice 2(8):35–37.
14. Ullrey, D.E.; Allen, M.E.; and Baer, D.J. 1991. Formulated diets versus seed mixtures for psittacines. Journal of Nutrition 121(11S):S193–S205.
15. Villm, D.L.; and O'Brien, S.E. 1988 An overview of pet bird nutrition. Iowa State University Veterinarian 50(2):107–113.
16. Zwart, P.; Schreus, W.H.P.; and Dorrestein, G.M. 1979. Vitamin A deficiency in parrots. Proceedings Erkrankugen der Zootiere Verhandlungsbericht des Internationaler Symposiums 13(17):47–52.

VIRAL DISEASES

Karin Werther

The great difficulty in working with diseases caused by viruses is their diagnosis and treatment. A number of diagnostic tests are used, including isolation of the pathogen from the test material, demonstration of viral particles or inclusion bodies by histopathology, demonstration of viral antigen in infected tissues using viral-specific antibodies, demonstration of viral nucleic acid in infected tissues using viral-specific nucleic acid probes, indirect demonstration of a viral infection by detection of humoral antibodies, or the rise in antibody titers in paired serum samples.

Most of the diagnostic techniques require special facilities and special training of personnel.[1]

Psittacine Beak and Feather Disease (Circovirus)

Psittacine beak and feather disease (PBFD) is caused by 14- to 16-nm diameter, nonenveloped virus with an icosahedral capsid containing a 1.7–2.0 kb, single-stranded, circular, DNA genome.[2] The virus has a broad host range and will infect birds of the Cacatuidae, Psittacidae, and Loridae families.[3] PBFD in New World parrots is a recent discovery and was described in scarlet macaw (*Ara macao*)[4] and blue-fronted Amazon (*Amazona aestiva*),[5] red-lored Amazon parrot (*Amazona autumnalis autumnalis*)[6]. The first confirmed occurrence of this disease in Brazil was diagnosed in a 3-year-old white cockatoo (*Cacatua alba*).[7] Other cases, also in Brazil, were described in *Cacatua goffini, Psittacula krameri,* and *Psittacus erithacus* using the histopathology techniques. Intracytoplasmic inclusion bodies were seen in macrophages of follicular epithelial cells and intranuclear inclusions bodies in lymphocytes at the medullar region of the bursa of Fabricius.[8]

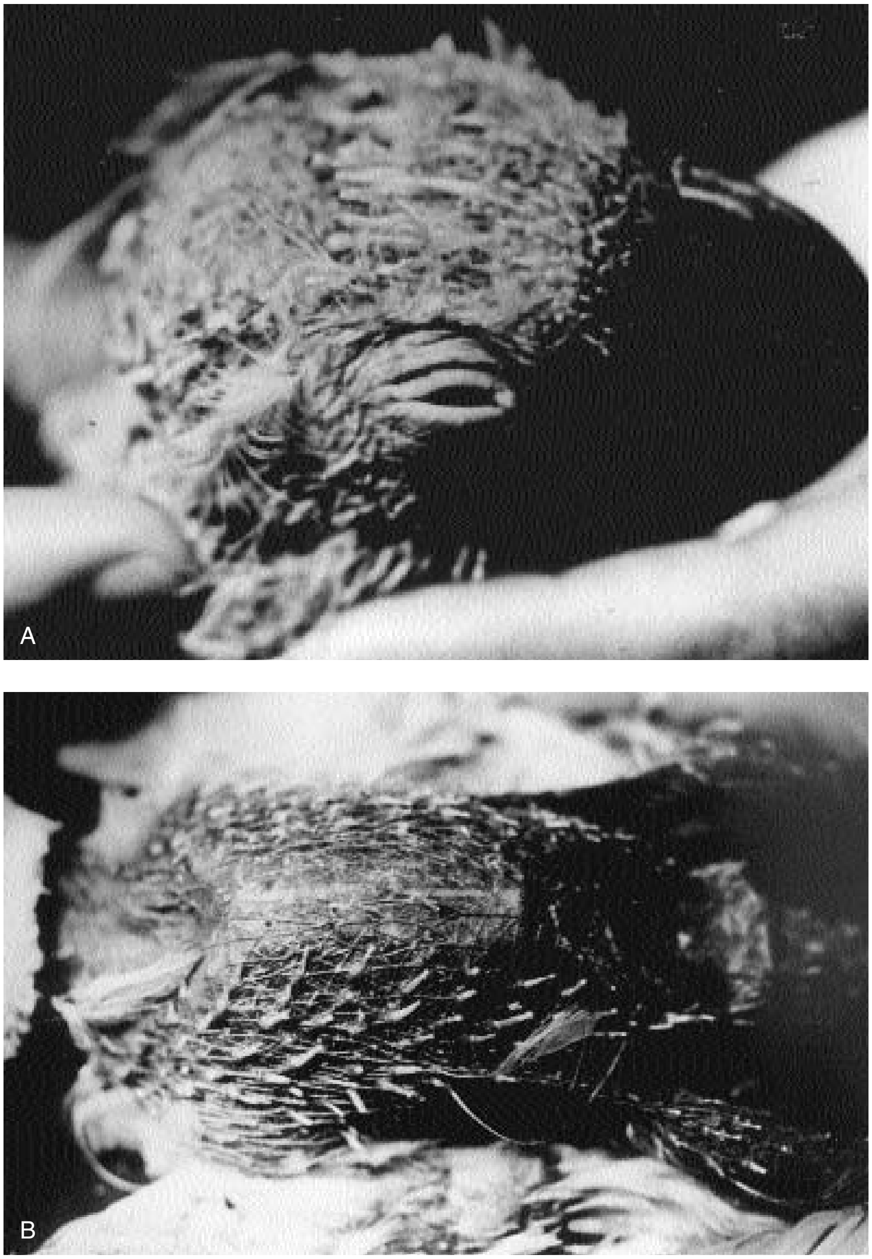

FIGURE 17.4. **A.** *Cacatua alba* with typical feather abnormalities caused by PBFD virus. **B.** *C. alba* with typical feather abnormalities caused by PBFD virus.

The transmission of the virus occurs principally by feather dust, feces, and crop washing.[1] Contact with people represent a potential transmitter, carrying the virus particles on clothing and objects that have had contact with the birds or the place where they live.

The white cockatoo (*C. alba*) was the first species reported to have this disease in Brazil. It had symmetrical feather loss, and dystrophy involved most of the skin. The beak was deformed and necrotic. Feather abnormalities included retained feather sheaths, clubbed and deformed feathers, circumferential constrictions, and blood within feather shafts (Figure 17.4). Partial loss of crest and tail feathers was also observed. The upper portion of the beak was elongated with necrosis of the hard palate. The lower beak also appeared abnormal. Nine months after the beginning of the feather loss, the bird died. The histological examination of the skin and feather follicle with hematoxylin and eosin-stained (H&E-stained) sections revealed a necrotic pulp with an intense infiltrate of heterophils. Portions of viable feather epithelium exhibited necrosis of basal epithelial cells. Some of these cells also contained glassy, basophilic nuclear inclusions. In addition, macrophages within the basal layers of the feather epithelium contained multiple, globular, basophilic, cytoplasmic inclusions (Figure 17.5). The definitive diagnosis was done by DNA in situ hybridization from different tissues such as skin, feathers, spleen, liver,

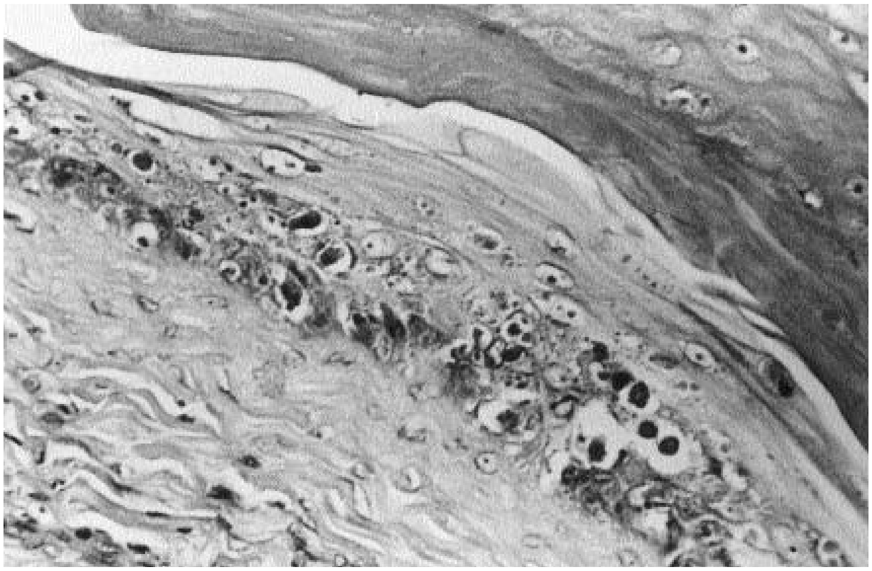

FIGURE 17.5. Photomicrograph of the epithelium from the feather follicle showing inclusion bodies caused by PBFD. (Hematoxylin and eosin stained; ×40.)

intestine, thyroid gland, parathyroid gland, adrenal gland, heart, great vessels, lungs, and kidney. These confirmed the presence of PBFD viral nucleic acid (hybridization in situ tests courtesy of Dr. Latimer, University of Georgia/USA).

This case of PBFD was diagnosed in a cockatoo, but it is possible that this disease may be disseminated in other Psittaciformes in captivity, and even more disastrous, in free-ranging psittacines.

The incubation period varies from 21–25 days to a maximum of 18 months. PBFD virus has hemagglutination activity for erythrocytes from different cockatoo species and guinea pig, but not chicken or sheep erythrocytes[1]. Erythrocytes of blue and gold macaws (*Ara ararauna*), red-shouldered macaws *(Diopsittica nobilis)*, blue-fronted Amazon parrots (*Amazona aestiva*), and orange-winged Amazon parrots (*Amazona amazonica*) were tested for agglutination in the presence of PBFD virus, and none was detected in any of these South American species.[9]

The diagnosis of PBFD can be done by histopathological examination of a plucked growing feather or biopsies of feathers and feather follicles. Characteristic changes in the growing feather and its follicle and the presence of characteristic virus-induced intranuclear and intracytoplasmic inclusion bodies are considered diagnostic.[10]

It is important to make a differential diagnosis with other diseases, such as: adenovirus, trauma, bacterial folliculitis, fungal folliculitis, septicemias, malnutrition, endocrine abnormalities, reaction to antibiotics, and principally with lesions caused by polyomavirus; but these lesions typically resolve after one or two molts, whereas PBFD lesions, as a rule, progress from molt to molt.[1] PBFD is a fatal disease, and no treatment exists.

To control this disease in a collection or breeding center it is most important to test birds and immediately remove the animals with positive test results.[10] Another possibility is a vaccination program from the companion birds.[1]

MANAGEMENT OF POSITIVE BIRDS If a bird from a breeding aviary with feather abnormalities is found to be positive, remove the bird from the area as quickly as possible. Virus-infected birds with feather abnormalities shed large concentrations of the virus in their feather dust, which may easily be carried to other birds by the wind, clothing, skin, or hair. All supplies and equipment that could be contaminated with feather dust from the infected bird should be repeatedly cleaned and disinfected. If a companion bird with feather abnormalities is found to be positive, the bird must never be exposed directly or indirectly to other birds outside the household. Infected companion birds may live a long life when provided a stress-free environment. However, anyone maintaining a PBFD-positive bird must be aware that the virus can be transported to other locations on clothing or in hair. Caregivers should not expose other birds to this virus by entering aviaries, pet shops, or bird shows.[3]

Poxvirus

Poxviruses are large (250-350 nm), enveloped DNA viruses. There are numerous poxviruses, and individual poxviruses have unique characteristics. Some infect only a single species, whereas others infect several.[10] The range of the parrot poxvirus appears to be confined to South American parrots.[11] The mucosal or wet form of pox is common in imported nestling blue-fronted Amazon parrots (*Amazona aestiva aestiva*) and pionus parrots.[3] Psittacine poxvirus infections have been documented in numerous South American parrots and parakeets. *Amazona* sp. and *Ara* sp. are most severely affected.[1] Avipoxvirus can not penetrate intact epithelium and needs traumatic lesions. Mosquitoes and mites serve as the primary mechanical vectors. Epornitics are common in the spring and fall when mosquitoes are most prevalent. Affected birds are most infectious when lesions or scabs are present. Infected free-ranging birds may serve as source of virus for some species. The incubation period varies from 10–14 days in a natural infection in Amazon parrots, but experimentally, the incubation period is shorter, only 7 days.[3]

The unique clinical signs that occur in Amazon parrots are thought to be caused by virulence factors and not differences in virus strains.[1] Coryza and ocular lesions are frequently the dominate signs in the genus *Amazona*.[1] However, necropsy findings usually include diphtheroid enteritis or myocardial necrosis. Ocular lesions begin as dry areas on the eyelid that become crusty with exudate, sealing the lids closed. Secondary infections are frequently keratitis, followed by ulceration, perforation of the globe, panophthalmia, and finally ophthalmophthisis. When secondary infections are present, there is distortion in the margin of the eyelids and loss of filoplumes around the eyes.[3]

In Amazon parrots severe upper respiratory tract disease occurs, along with diphtheritic lesions on the oral pharyngeal esophageal or crop mucosa, respiratory distress, depression, anorexia, diarrhea, and bloody stools. Mortality rates are highest when diphtheritic lesions are secondarily infected.[3]

Gross lesion are visible on the skin or oral mucosa. There are fibrinonecrotic lesions on the gastrointestinal tract and ulceration of the nasal, tracheal, and bronchial mucosa.

HISTOPATHOLOGY Necrosis of cells lining the mouth, esophagus, crop, or trachea occurs. Bollinger bodies are seen in skin or mucosa of the sinuses, trachea, crop, esophagus, or pharynx.[3]

Diagnosis in the living bird is accomplished by virus isolation from vesicles, necrotic mucosa, pharyngeal swabs, or feces, or by demonstration of poxvirus in lesions using cytology or electron microscopy, agar gel immunodiffusion (AGID) assay, virus neutralization (VN) assay, enzyme-linked immunosorbent assay (ELISA), or hemagglutination inhibition (HI) assay to detect antibodies.[3] Diagnosis at necropsy is by microscopic examination of tissues for intracytoplasmic inclusions, virus isolation from skin lesions, mucosal lesions, or liver [3].

Differential diagnosis should include trauma, *Trichophyton* sp., knemidikoptes mites, papillomavirus, and bacterial infection. In the diphtheritic form consider candidiasis, hypovitaminosis A, aspergillosis, trichomoniasis, or herpesvirus.[3]

THERAPY Gently remove infected tissue and thoroughly cleanse open wounds. Treatment, when necessary, is predominately supportive and is designed to keep the animal alive until its own immune system can eliminate the infection. In parrots vitamin A (10,000–25,000 IU/300 g, intramuscularly (IM), once) has been suggested to be efficacious. Antibiotics and antifungals are indicated when secondary infections complicate the diseases. Fluid therapy and tube feeding may be necessary in the anorectic bird. Intensive management of ocular lesions in nestling parrots is felt to limit complications. Topical chloramphenicol ointment is also applied. Scabs should be allowed to fall off without assistance because manual removal appears to result in more lid damage. [11]

PREVENTION Vaccination at 10–14 days old is recommended in areas with density of mosquitoes.[1] Taxon-specific vaccines are available for only a few of the avian poxviruses. Vaccines are commercially available for psittacine poxvirus and should be considered to prevent infections in high-risk populations (imported birds, petshop birds, those exposed to imported birds, birds in areas with high densities of mosquitoes).[12] Vaccination trials with a killed pox vaccine suggested that early vaccination of parrots in the country of origin immediately after capture would significantly reduced mortality.[13]

CONTROL Eliminate mosquitoes. Control in imported hand-fed parrots requires proper hygiene. Each bird needs to be fed with his own hand-feeding implement, and affected birds need to be isolated and fed last. Handlers must wash their hands frequently.[13]

The virions are stable when outside the bird, inactivated with 1% potassium hydroxide, at 50°C for 30 minutes or 60°C for 8 minutes, steam, 2% sodium hydroxide (NaOH), and 5% phenol. Isolate infected birds and those birds that are directly exposed. Destroy contaminated wooden perches or nest boxes, sterilize contaminated nets, towels, clothing, holding containers, equipment, and supplies.[3]

Adenovirus

ETIOLOGY Adenoviruses are nonenveloped, 70–90 nm in diameter.

EPIZOOTIOLOGY Species have varying degrees of susceptibility, with the incubation period undetermined.

Some strains are transmitted horizontally.[3] Most practitioners first encounter adenovirus infections in a necropsy of a bird that died from other causes.[14] Adenoviruses have been described in lovebirds, budgerigars, cockatoos, South American parrots, eclectus, and Amazon parrots.[14]

SIGNS Signs of infection are rapid death without premonitory signs, depression, anorexia, yellowish-green diarrhea, cloacal hemorrhage followed by enteritis, hepatitis, pancreatitis, encephalitis, splenitis, conjunctivitis, and death.

At necropsy the liver is enlarged, friable, and discolored, with mottling and subcapsular hemorrhages. There is congestion of the intestines, necrosis in the liver, spleen, pancreas and/or intestines, intranuclear inclusion bodies in the liver, spleen, and pancreas or intestines. The inclusion bodies are similar to those caused by polyomavirus, herpesvirus, or PBFD virus. Presence of inclusion bodies in clinically normal birds suggest asymptomatic infections.

DIAGNOSIS Differential diagnosis should consider bacterial hepatitis or enteritis, chlamydiosis, salmonellosis, polyomavirus, reovirus, Pacheco's diseases virus, paramyxovirus, and influenza A virus.

Diagnosis in the living bird is performed by virus isolation or demonstration of virus from feces or pharyngeal secretions using electron microscopy.

Postmortem diagnosis uses suggestive microscopic changes; virus isolation from liver, intestines, kidney, or pancreas; and viral-specific DNA probes.

MANAGEMENT Vaccines for psittacines are not available. The virus is stable outside the host and resistant to many disinfectants, but is inactivated by 1 hour of contact with formaldehyde and iodophors.

Supportive care should be provided: fluids, assisted feeding, and broad spectrum antibiotics to prevent secondary infections.

Amazon Tracheitis Virus (Herpesvirus)

ETIOLOGY This virus is enveloped and pleomorphic, 120–200 nm in diameter.

EPIZOOTIOLOGY A virus believed to be a mutation of the infectious laryngotracheitis virus of chickens has been observed to cause a severe upper respiratory and tracheal disease in Amazon parrots and *Neophema bourkii*.[14] Transmission is probably through respiratory secretions.

SIGNS Signs are rapid death following a brief period of severe dyspnea; chronic upper respiratory disease; diphtheritic membranes in the trachea; necrosis of cells lining the respiratory tract, pharynx, larynx, esophagus, or crop.

DIAGNOSIS Differential diagnosis should consider bacterial sinusitis, rhinitis, or tracheitis, chlamydiosis, Newcastle disease virus, influenza A virus, aspergillosis, trichomoniasis, tracheal mites, hypovitaminosis A, and diphtheritic form of poxvirus.

In the living bird, diagnosis is made by virus isolation from tracheal swabs.

In postmortem diagnosis, intranuclear inclusions bodies may be difficult to detect in the affected tissues, but virus is isolated from trachea or lungs.

MANAGEMENT Efficacy of inactivated infectious laryngotracheitis (ILT) virus vaccines is undetermined and attenuated live ILT virus vaccines should not be used.

Pacheco's Disease Virus, Herpesvirus

ETIOLOGY This is an enveloped and pleomorphic virus, 120–200 nm in diameter.

EPIZOOTIOLOGY Some psittacine birds are reported to be susceptible (African grey parrots, cockatiels, conures, lories, macaws, Amazon parrots, cockatoos, eclectus parrots, lovebirds, rosellas). Virus is shed in feces and pharyngeal secretions several days before death and enters new birds when ingested. Latently infected birds shed virus intermittently. Incubation period varies from 3 to 14 days.

SIGNS Signs are rapid death without premonitory signs, asymptomatic or greenish-yellow diarrhea, regurgitation, and CNS signs. Elevated serum enzyme, lipemia, leukopenia, and anemia may occur.

DIAGNOSIS Enlarged hemorrhagic reddish to yellow-brown liver, enlarged spleen and congestion of blood in abdominal organs, and hemorrhages on the surface of various organs can be observed at necropsy.

A histopathologic review reveals necrosis of the liver, spleen, kidneys, and other tissues. There are intranuclear inclusion bodies in the liver, spleen, kidneys, and other tissues.

Differential diagnosis should consider bacterial hepatitis, chlamydiosis, salmonellosis, heavy metal toxicity, avian polyomavirus, reovirus, and adenovirus.

In the living bird diagnosis is made by virus isolation from feces or pharyngeal swabs, virus DNA probes from feces, and detection of antibodies.

In postmortem diagnosis intranuclear inclusions are evident; use virus-specific DNA probes, virus isolation from liver, spleen, brain, or kidney.

MANAGEMENT Inactivated vaccines are available. Virions are unstable when outside birds; most disinfectants inactivate the virus.

Administer 80 mg/kg acyclovir orally every 8 hours.

Proventricular Dilatation Disease, Neuropathic Gastric Dilatation, Psittacine Wasting Syndrome

ETIOLOGY The etiology is not confirmed, but probable causes are enveloped viral particles, measuring 80 nm in diameter.

EPIZOOTIOLOGY Experimental reproduction of proventricular dilatation disease in psittacine birds employed *Eclectus roratus, Ara macao, Cacatua moluccensis, C. alba, Amazona ventralis*. This virus was recovered consistently from naturally affected and experimentally infected birds.[15] Clinical or microscopic changes characteristic of the disease have been described in over 50 species of psittacine birds. Any age of bird is susceptible, however, adult birds appear to be more commonly affected.[3] Necropsies of macaws (*Ara araruana, Ara macao*) and *Eclectus roratus* in Brazil were suggestive, based on clinical history, radiography, and finding lesions suggestive of PDD, but in situ DNA hybridization didn't confirm the presence of the virus from PDD. The transmission from this disease is suspected to be direct or indirect.

Clinical observations suggest incubation is several weeks to 4 years.

SIGNS There are gastrointestinal signs, neurological signs, or a combination of both: progressive weight loss, despite a good appetite, passage of poorly digested food, regurgitation, CNS signs, including ataxia and seizures. Increased creatinine kinase (CPK) activity may be an indication of PDD, hypoproteinemia, hypoglycemia, heterophilia, and anemia.

DIAGNOSIS Emaciation, dilatation of the proventriculus and/or ventriculus, flaccid ingesta-filled crop, dilatation of the intestines with undigested food and gas can occur at necropsy.

Differential diagnosis should consider fungal proventriculitis, megabacteriosis, parasitic enteritis, foreign bodies, neoplasm, heavy metal toxicosis, bacterial enteritis, papillomatosis of the proventriculus/ventriculus or any intraluminal or extraluminal mass that prevents the passage of food. Proventriculus of neonates is normally dilated.

In the living bird, acute phase is suspected in birds with rapid gastrointestinal transit time. Chronic phase is suspected in birds with slowed gastrointestinal transit time. A crop biopsy will confirm the diagnosis.

In postmortem diagnosis, accumulation of lymphocytes and plasma cells in the nerves of the gastrointestinal tracts, brain, or spinal cord can be seen.

MANAGEMENT Avoid direct or indirect contact with affected or exposed birds.

For treatment, supportive care, assisted alimentation, broad spectrum antibiotics should be supplied, as needed.

Papillomaviruses

ETIOLOGY Papillomas are suspected, but unproven, to be caused by a virus.[3] Papillomaviruses are nonenveloped double-stranded DNA virus, 45 nm in diameter.[14]

EPIZOOTIOLOGY A retrospective study of cloacal papillomas of psittacine birds was performed on tissue specimens submitted from 1993–1998. Cloacal papillomas occurred most frequently in Amazon parrots, macaws, and cockatoos. There was no apparent gender predisposition to papilloma development. The median age of affected birds was 4.5 years.[16] The cloacal form was observed in the blue-fronted Amazon (*A. aestiva* and *Ara maracana*) at the Brazilian Zoological Garden (personal communication).

Incubation period is unknown. If an infectious agent is involved, it appeared to be of low transmissibility.

SIGNS Most birds are asymptomatic, with cloacal masses, tenesmus, putrid-smelling feces, infertility, recurrent enteritis, hematochezia, recurrent prolapses, and accumulation of droppings around the vent. Oral or esophageal masses, halitosis, dysphagia, dyspnea or wheezing, and intestinal tract masses, anorexia, chronic weight loss, or vomiting may mimic proventricular dilatation disease.

DIAGNOSIS At necropsy proliferative masses on mucosa of the oral cavity, gastrointestinal tract, or cloaca are observed. Cancer of the liver and pancreas is common in affected birds.

Histopathology shows that there is proliferation of epithelial cells on a well-vascularized, fibrous stalk. Classic description involves acanthosis and hyperkeratosis. Diagnosis should consider neoplasm, oral masses, hypovitaminosis A, and poxvirus. In the living bird, a 5% acetic acid solution will cause suspected lesions to appear white.

In postmortem diagnosis, microscopic examinations of suspected lesions show characteristic changes. Microscopic examinations of the liver and the pancreas show neoplastic changes.

MANAGEMENT Examine all birds during the quarantine period to prevent introduction of affected birds, and restrict contact between affected and unaffected birds.

Oral or esophageal lesions can be surgically or radiosurgically removed. Cloacal lesions are best removed with silver nitrate cauterization.

Polyomavirus (Non-Budgerigar Psittacines)

ETIOLOGY Polyomavirus is a nonenveloped, double-stranded DNA virus, 40–48 nm in diameter.

EPIZOOTIOLOGY Different species of macaws (scarlet macaws, blue and gold macaws, military macaws, green-winged macaws, and Hahn's macaws), Amazon parrots (double yellow-headed Amazon, orange-winged Amazon parrots, yellow-naped Amazon parrots, red-lored Amazon parrots, blue-fronted Amazon parrots), and other Psittaciformes showed typical lesions.[17] Most infections in psittacines are subclinical, and infested birds developed an antibodies response; latent infections are suspected but not confirmed.

Experimentally infected psittacines shed virus in feces from 2–7 days after inoculation.

It is transmitted orally, intramuscularly, and intravenously (experimental); virus is present in feces. Vertical transmission is not documented.

SIGNS Subclinical infections are most common. It is peracute with no clinical signs in young birds. Signs are rapid death 12-48 hours after depression, delayed crop emptying, regurgitation, diarrhea, and subcutaneous hemorrhage. There is bleeding from injection sites or feather follicles. Hematuria in eclectus parrots. Feather abnormalities are rare.

At necropsy, hepatomegaly, splenomegaly, pale swollen kidneys, pale cardiac and skeletal muscle, and subcutaneous hemorrhage of the intestines, liver, and heart can be observed.

On histopathology massive liver necrosis and hemorrhage throughout the body are characteristic. Intranuclear inclusion bodies may occur in the liver, spleen, and kidneys.

DIAGNOSIS Differential diagnosis should consider feather lesions, PBFD virus, adenovirus, endocrine abnormalities, bacterial infections, fungal infections, traumatic injuries, and drug reactions (cephalosporin and penicillin), systemic lesions, chlamydiosis, liver diseases, clotting disorders, bacterial septicemia, Pacheco's disease virus, adenovirus, and reovirus.

In the living bird, diagnosis requires DNA-probe detection of virus in feces, microscopic evaluation of liver biopsies, and demonstration of a fourfold increase in antibody titer in paired serum samples. Infected birds may have increased activities of lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alkaline phosphatase (AP).

In postmortem diagnosis, there is demonstration of virus particles as seen by electron microscopy, isolation of the virus in cell culture, specialized staining of suspect lesions using viral specific antibodies, or the detection of viral nucleic acid using polyomavirus-specific DNA probes.

MANAGEMENT Vaccination with inactivated vaccines is available. Sound hygienic practices, maintaining closed aviaries, preventing visitors from entering avian nursery, and attempting to identify and isolate subclinical shedders using viral specific DNA probes are needed.

Polyomavirus is environmentally stable and resistant to inactivation by chlorhexidine and partially resistant to some iodine-containing disinfectants and quartenary ammonium; it is inactivated by Clorox, stabilized chlorine dioxide, phenol, and ethanol.

Supportive care is needed.

FUNGAL INFECTIONS

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Aspergillosis

ETIOLOGY Aspergillosis is the most frequently observed fungal disease in birds. It is opportunistic[11] and is generally characterized by respiratory distress.[3] In the majority of cases, *Aspergillus fumigatus* is responsible for the pathologies diagnosed,[4,11] followed by *Aspergillus flavus* and *Aspergillus niger.*[3] The blue-fronted Amazon and the African gray parrot have been reported as the species of psittacidae most susceptible to the fungus.[3,11] *Aspergillus* spp. occur naturally in the environment and proliferate in dark, damp, poorly ventilated sites covered with fecal material, in damp nests, contaminated corn and peanuts, and wet skin and feathers.[11]

TRANSMISSION Birds are frequently exposed to the fungus; spores may be inhaled or ingested, especially if the psittacine is fed moldy seeds. Fungi are also capable of penetrating broken skin or eggshells[11] and may contaminate developing embryos. Healthy birds are resistant to infection even when exposed to high spore counts, but birds with weakened immune systems may develop the disease even when exposure is only to low spore counts.[3]

The factors most commonly related to breakdown of the immune system are coexistent diseases, treatment with antibiotics (especially tetracyclines)[3] or corticosteroids, stress, respiratory irritants (cigarette smoke, ammonia),[3] and malnourishment, especially vitamin A deficiency that results in a metaplastic alteration of the respiratory epithelium, which is associated with susceptibility to aspergillosis.[11] Nestlings and aged birds are highly susceptible.[3] Aspergillosis is not usually thought to be contagious; more recent studies do not reject this possibility, because viable fungal elements have been observed in bronchial exudates.[1] Experiments carried out with material collected from the saliva, pharynx, trachea, and nasal sinuses of healthy birds[4] demonstrated fungal growth, as did samples from feces of healthy birds.[11]

CLINICAL SIGNS In the periorbital sinuses, it generally begins unilaterally, with chronic purulent mucous discharge, and it may extend to the beak, causing it to become malformed.[11] The syrinx or the bifurcation of the trachea appear to be common locations for *Aspergillus* spp. colonization, resulting in blockage of the air passages with necrotic debris.[3] The first overt clinical signs are changes in or loss of the voice, which progresses to severe dyspnea and death.[11] Other signs include open-beak breathing, tail bobbing, head movements, pronounced sternal movements, and stretching of the neck.[11]

The acute form generally attacks young birds, resulting in multiple small white miliary granulomas in the lungs and air sacs. Clinical signs include dyspnea with open-beak breathing, anorexia, diarrhea, and sudden death in a few days.[11]

The subacute form lodges in the air sacs, presenting a slow, progressive course over a period of more than 3 weeks. It usually develops following prolonged exposure of an immunosuppressed bird to a low concentration of spores.[11] The caudal thoracic and abdominal air sacs are generally the first point of infection. However, numerous nodules palpable through the skin in the neck region, located exclusively in the cervical air sacs, have been reported in a cockatoo.[16] Lesions may be extensive throughout the respiratory system before the first overt signs. Clinical signs include weight loss, depression, vomiting, polyuria, polydipsia, respiratory distress after exercise, and, occasionally, signs that the CNS has been affected, such as torticollis and ataxia.[2] Posterior paralysis was reported in a black palm cockatoo, caused by dissemination of the fungus from the abdominal air sac to the kidneys and nerves.[7] Biliverdinuria is also common when there is hepatic involvement.[9] Other colonization sites include the skin, muscles, gastrointestinal tract,[3] liver, kidneys, eyes, brain, and bones.[9] Aspergillosis may be associated with ascites, caused by peritonitis or cardiopulmonary problems, induced through thrombus in the pulmonary veins.[3]

DIAGNOSIS Diagnosis is based on history, clinical signs, hematological findings, culture, x-ray, histology, cytology, serology, endoscopy and exploratory surgery.[3] Clinical pathology may reveal leukocytosis, heterophilia, monocytosis, and lymphopenia,[11] and anemia is also often present.[3] When a culture is positive without the presence of overt signs, it is not considered diagnostic for the disease, because the fungus is ubiquitous. Radiography may reveal dark spots of various sizes, diffused or focused in the region of the air sacs; unilateral or bilateral distension of the thoracic caudal and abdominal air sacs; increased focal or diffused radio density in the pulmonary region, and in some cases, rounded radio-dense dark spots at the termination of the trachea and the bifurcation of the bronchial tubes; hepatomegaly and nephromegaly, probably caused by mycotoxins released by the fungus; and splenomegaly, probably resulting from bacterial and viral infections that predispose the bird to development of the fungal disease.

Histologic examination of the granulomas generally reveals necrotic focal points surrounded by macrophages, heterophils, and giant cells, sometimes with connecting capsular tissue.[3] ELISA-method serological tests have also been used;[14] however, they do not form part of routine diagnosis in most countries, and for many species they are not yet totally accurate. Generally, laparoscopy reveals a white discharge in the trachea; white or yellow plates in the syrinx, bronchial tubes, air sacs, or other membranous surfaces; thickening of the air sacs, with or without caseous exudate; nodules in the pulmonary parenchyma with consolidated caseation;[11] and caseous nodules inside the parenchymatous organ.[11] The white or yellow plaques in the air sacs may be covered with a green-gray pigmented mold.[9] Cytology of tracheal or air sac flushings demonstrates the presence of ramified septate hyphae.

TREATMENT Treatment represents a challenge. It appears that the disease can only be controlled and not eliminated. Treatment includes obligatory correction of stress factors.[3] General support and application of antibiotics, depending on sensitivity tests, is necessary. The success of treatment depends on the location and extent of the lesions,[3] because surgical removal of the granulomas and direct application of antifungal agents to the lesions are more effective than less aggressive regimens. Treatment should be aggressive and prolonged; it is suggested that therapy continue for as long as 2 months subsequent to the remission of clinical signs.

ANTIFUNGAL AGENTS Amphotericin B is used initially 1.5 mg/kg every 8 hours for 3-5 days, intravenously (IV).[9,1] Intratrachealy (1 mg/kg diluted in saline solution 0.9%) it is administered every 8–12 hours via the glottis on inhalation, for tracheal and pulmonary aspergillosis;[3] for nebulization, 1 mg/kg is diluted in saline solution 0.9% for 15 minutes every 12 hours. It is useful for recent infections of the upper respiratory tract.[11] Surgical irrigation uses 0.05 mg/mL diluted in saline solution 0.9%.[11] Topical ointments are used for external lesions.

Flucytosine (20–60 mg/kg orally) is administered every 12 hours. Ideally it should be given in conjunction with amphotericin B, because it is a fungistatic that requires prolonged treatment. The drug may provoke bone marrow toxicity. It has good intratissue distribution, including the CNS.[9]

Ketoconazole (20-50 mg/kg orally) is given every 12 hours for 2–6 weeks.[8]

Itraconazole (5-10 mg/kg) is administered every 12 hours. For the African gray parrot, 5 mg/kg is used. Itraconazole has been considered as the most effective medication. Treatment should be prolonged.

Fluconazole (10 mg/kg) is also used.[17]

Myconazole (10 mg/kg IM) is given once a day for 6–12 days (dosage recommended for birds of prey).[14]

Enilconazole, miconazole, chlortrimazole are all topical ointments.

Candidiasis

ETIOLOGY *Candida albicans* is an opportunistic yeast infection frequently associated with diseases of the gastrointestinal tract (GIT).[3,9] Species of yeast infection may be found in small numbers in the GIT and the urinary and reproductive systems of healthy birds.[4] *C. albicans* is the most commonly encountered organism, but *Candida parapsilosis* has been reported as the cause of systemic infection in an adult blue-fronted Amazon parrot.[9,11]

TRANSMISSION Candidiasis is more prevalent in young birds,[9] in which it may occur in primary form, probably as a result of the immaturity of the immune system.[3,11] Cockatiel,[3] parakeet, and lovebird[11] hatchlings appear to be especially susceptible to primary yeast infections, however, old birds may also suffer sudden onset of the disease. Other factors of predisposition include immunosuppression, the presence of parasites, malnourishment, vitamin A deficiency, [9,11] coexisting diseases, lack of hygiene, stress, dirty nests, damp floors, and prolonged treatment with antibiotics. Antibiotics destroy the normal bacterial flora of the oral and gastrointestinal mucous membranes, which have an inhibiting effect on the growth of the various species of yeasts, and their suppression through the use of antibiotics, especially tetracycline,[9] may permit yeast proliferation and development of the disease. Untreated water, contaminated food,[4,11] contaminated parent birds that feed their chicks,[11] and saliva, feces, and other secretions from contaminated birds[4] are other sources of transmission. Chick formulas fed when they are too hot may cause burn lesions in the mucous membranes of the crop and a predisposition to yeast growth.[11]

Yeast infection is generally confined to the crop, causing a prolonged emptying time and malnourishment. It is a frequent cause of crop impaction and death in psittacine hatchlings. It may also invade the mouth, infraorbital sinuses, esophagus, proventriculus, gizzard, and intestinal tract, especially the small intestine.[9] Disease occurs when a surface colony penetrates to a deeper location.[9] In the majority of cases, yeast infection is a secondary invasion.[9,11]

CLINICAL SIGNS Clinical signs are most commonly related to GIT and vary according to the point of infection. Mouth and beak infections may cause halitosis, mucous exudate[9] with the presence of yellow-white necrotic plaques in the oral cavity.[11] The presence of several oral lesions may cause obstruction and interfere in normal feeding and respiration, leading to debilitation. The crop is most commonly affected in young birds, and signs are regurgitation caused by loss of tonicity in the crop wall, slow emptying time, mucous-catarrhal exudates, anorexia when the infection occurs in the mucous membranes,[9] weight loss or reduced body growth and development. Deep infections advance to complete stasis and thickening of the crop,[9] which becomes wrinkly like a Turkish towel.[11] Infections of the crop may occur without oral lesions.[11] In infections of the proventriculus and lower GIT, vomiting occurs, as well as indisposition, weight loss, and diarrhea.[9] Malnourishment occurs from chronic enteritis and decreased absorption of nutrients.[9] Infections of the crop, proventriculus, and lower GIT may occur simultaneously.[9]

Systemic yeast infection is rare and generally occurs only in severely debilitated birds. The microorganism is found in the blood, bone marrow, or parenchymatous organs. *Candida parapsilosis* has been reported as causing systemic infection in a parrot. Radiographic examination revealed increased density in the long bones, hepatomegaly, and splenomegaly.[11]

Skin lesions in psittacines involve the commissures of the mouth, the nose, cloaca, and feather follicles on the head, ventrum, and back.[11] Cutaneous infections induce an inflammatory and hyperkeratose reaction.[11] Conjunctivitis and lesions on the cornea have been reported in an African grey parrot with a vitamin A deficiency, in which *C. albicans* was isolated.

DIAGNOSIS Diagnosis cannot be based solely on results of a culture, because the organism occurs naturally in the GIT of psittacines. Diagnosis must be based on the clinical signs, history of predisposing factors,[9,11] demonstration of existing lesions, identification of a large number of yeast organisms in cytological samples of exudates, and positive culture and identification of pseudohyphae in tissue samples.[9]

TREATMENT Treatment should begin with the elimination of predisposing factors. Nystatin is the most frequently used medication[9] in the first stages of treatment for the upper GIT, although it is not absorbed.[3] It should be in direct contact with the lesions. Dosages range from 250,000 IU/kg twice per day to 300,000 IU/kg every 8 hours.[3,9]

Ketoconazole at a dosage of 10-30 mg/kg twice a day for 21 days is recommended when the disease is refractory to nystatin and infection is acute. Possible side effects of ketoconazole are vomiting and a raised level of liver enzymes.[3] The drug may be mixed with orange or pineapple juice to facilitate dilution. Yeast infections prefer alkaline environments, and acidification of the GIT has a therapeutic affect.

Fluconazole tablets(2–5 mg/kg) are administered in a single dose for 7 days, when infection is resistant to ketoconazole.

Amphotericin B (1.5 mg/kg IV) is administered three times a day for 3 days; 1.0 mg/mL is administered intratracheally in saline twice a day;[3] 1 mg/mL saline is nebulized for 15 minutes twice a day.[3]

Flucytosine (20–60 mg/kg orally) is administered every 12 hours. Unfortunately, the organism generally develops rapid resistance to this medication;[9] dosage may be increased up to 250 mg/kg orally twice a day for 21 days.[3]

Itraconazole (beads in capsules,5–10 mg/kg) is given twice a day in food for 7–21 days. This drug presents no advantages over other medications.

Topical treatment of the oral and skin lesions consists of scraping and application of amphotericin B 3% ointment.[9] Ocular lesions have been treated with the same medication, and subconjunctive infections with a solution containing 25 mg of amphotericin per milliliter of sterile solution.[9] Severely affected birds should receive supplements of vitamins A and B complex.

Uncommon Mycoses

Less common fungi have been reported as pathologic agents in psittacines. Immunosuppressed birds, prolonged treatment with antibiotics, dirty and poorly ventilated enclosures, and coexisting diseases are factors that allow some opportunistic saprophytic fungi to become pathogenic.

Cryptococcus neoformans is normally encountered in the feces of psittacines[9] and rarely causes disease, probably because it is inhibited by the antifungal activity of the normal bacterial flora and because *C. neoformans* develops poorly or does not develop at all in the higher body temperature of birds, which averages 40°C.[9,11] It has been reported in the African grey parrot,[9,11] the thick-billed parrot (*Rhynchopsitta pachyrhyncha*),[9,11] the green-winged macaw (*Ara chloroptera*),[3, 9,11] the Moluccan cockatoo,[3] and the triton cockatoo.[11] The upper respiratory tract is more susceptible to initial colonization because of its lower temperature.[9.11] Spreading to the CNS appears to be common.[11] The lesions contain a gelatinous myxomatous material.[11] It may spread to the meninges, brain, intra- and periocular tissues, tongue, and bone marrow.[9] Frequently observed clinical signs include weight loss, apathy, anorexia, diarrhea, nasal discharge, soft tissue swelling around the sinuses and periorbital tissue,[9] moderate to severe dyspnea[3,9] if there is colonization in the lower respiratory tract, increased periorbital volume,[9] blindness, paralysis, and anemia.[3]

Diagnosis is based on clinical signs, cytological and histopathological examinations and culture.[9] Hematology generally reveals anemia and heterophilia. At necropsy, a gelatinous substance may be encountered in the long bones, respiratory tract, abdominal cavity,[3] sinuses, and brain.

Treatment for acute infections depends on early diagnosis and aggressive therapy. Fluconazole is the medication of choice for mycotic infections of the eye, cerebral spinal fluid, and other important sites.[9] Ketoconazole, or a combination of itraconazole and amphotericin B[9] have also been prescribed.

Trichosporum beigelii was diagnosed in a green-winged macaw suffering from emaciation, polyuria, and granulomas in the myocardium, liver, and lungs.[3, 9,11] It was also cultured from the necrotic liver and pneumonic lung and air sacs of an Amazon parma.

Nocardia asteroides has been reported involving the lungs and air sacs of two pesquet parrots.[3]

Malassezia pachydermatis has been reported in an adult female scarlet macaw suffering from weight loss, weakness, and voice alteration, along with capillary infestation. Endoscopy revealed mucous membrane ulceration in the crop. Cultures identified *Escherichia coli* and *klebsiella* spp., as well as *M. pachydermatis*. It was suspected that *M. pachydermatis* may be resident in the bird's normal microflora.[11] *M. pachydermatis* was identified as the agent causing dermatitis in cockatiels and eclectus parrots, diagnosed in histopathological studies of the skin and feathers of the birds.[11]

Curvularia geniculata infection in a grand eclectus parrot[3,11] was reported to cause tortocollis, opisthotonos, convulsions, and paralysis of the right wing. At necropsy, grayish-black nodules in the left upper thoracic cavity, lung, cerebral hemisphere, and optic lobes were found.[11]

Mucor spp. was isolated from a 4-year-old military macaw with acute bronchopneumonia, with yellow exudate and bilateral thoracic and caudal air sacs filled with a gelatinous substance.[11] It was also reported in the air sacs, kidneys, and tongue of African grey parrots[9] and in budgerigars with catarrhal pneumonia.[11]

Absidia corymbifera produced lesions in the kidneys and air sacs of a recently imported adult African grey parrot with swelling and erosions of the tongue and a viscous exudate.[11] It was also identified in the ventricular thrombus and myocardium of a depressed lovebird.[11]

Penicillium chrysogenum and *Penicillium cyclopium* have been associated with feather damage and pruritus in psittacidae.[11] *P. cyclopium* was isolated from a 3-year-old lesion on the beak of an adult macaw (*Ara ararauna*). Necrotic lesions were also located inside the tissue of the cornea.[11]

Dermatomycoses

Skin mycosis is uncommon in psittacines.[9] *Trychophyton* spp., *Microsporum gypseum, Candida* spp.,[9] *Mucor cicinelloides, Rhizopus arrhizus, Penicillium chrysogenumm. P. Cyclopium, Aspergillus candidus,* and *Aspergillus phonenicis*[11] have been reported in several species of birds. *Penicillium* and *P. cyclopium* have been associated with feather damage and pruritus in psittacidae.[11] Clinical signs include self-mutilation,[9] skin thickening, brittle feathers,[9] pruritus, and feather loss.[9,11]

Dermatomycoses are difficult to treat, with removal of the feathers in worst condition and application of a 10% chlorhexidine solution 1–2 times a week, until plumage begins to grow back, is recommended.[6] The use of nystatin-based ointments is suggested for yeast infections.

REFERENCES

1. Alley, M.R.; Castro, I.; and Hunter, J.E.B. 1999. Aspergillus in hihi (*Notiomystics cincta*) on Mokoia Island.
2. Atkinson, R.; and Brojer, C. 1998. Unusual presentations of aspergillosis in wild birds. In Proceedings of the American Association of Avian Veterinarians. Omaha, Nebraska, pp. 177–181.
3. Bauck, L. 1994. Mycoses. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Applications. Lakeworth, Florida, Wingers Publishing, pp. 997–1006.
4. Benez, S.M. 1998. Fungos, leveduras e micotoxinas [Fungi, yeast and mycotoxins]. In S.M. Benea, ed., Aves Silvestres, Ornamentais, avinhados: Criação, clínica, teoria, prática. São Paulo, Robe Editorial, pp. 336–343.
5. Campbell, T.W. 1986. Mycotic diseases. In G.J. Harrison, ed., Clinical Avian Medicine and Surgery. Philadelphia, W.B. Saunders Company, pp. 464–471.
6. Clubb, S.L.; and Herron, A. 1998. Feather discoloration due to saprophytic fungal growth. In Proceedings of the American Association of Avian Veterinarians. Omaha, Nebraska, pp. 71–73.
7. Greenacre, C.B.; Latimer, K.S.; and Ritchie, B.W. 1992. Leg paresis in a black palm cockatoo (*Probosciger aterrimus*) caused by aspergillosis. Journal of Zoo and Wildlife Medicine 23(1):122–127.
8. Grimm, V.F.; Koster, J.; Pscherer, G.; and Werther, K. 1995. Diagnose und Therapie der Aspergilose bei verschiedenen Vogelarten [Diagnosis and treatment of aspergillosis in various bird species]. In Verhandlungsbericht des 37. Internationalen Symposiums über die Erkrankungen der Zoo- und Wildtiere, Berlin, Institut für Zoo- und Wildtierforschung im Forschungsverband Berlin e. V. pp. 393–398.
9. Oglesbee, B. Mycotic diseases. In In Verhandlungsbericht des 37. Internationalen Symposiums über die Erkrankungen der Zoo- und Wildtiere, Berlin, Institut für Zoo- und Wildtierforschung im Forschungsverband Berlin e. V., pp. 323–331.
10. Pscherer, G. 1995. Klinische Studien zur Therapie der Aspergillose bei Psittaciformes mit Flukonazol, Ketokonazol bzw, Itraconazol. DVM thesis, Tierärztlichen Fakultät der Ludwig-Maximilians-Universität München. pp. 119.
11. Reavill, D. Fungal Diseases. pp. 586–595.
12. Redig, P.T.; Orosz, S.; and Cray, C. 1997. The Elisa as a management guide for aspergillosis in raptors. In Proceedings of the American Association of Avian Veterinarians. Houston, Texas, pp. 99–104.
13. Redig, P.T.; Concannon, T.; and Post, G. 1986. A review of current methods for the diagnosis and treatment of avian aspergillosis. In Proceedings of the Annual Meeting of the American Association of Zoo Veterinarians. Chicago, Illionois, pp. 90–92.
14. Redig, P.T. 1986. Mycotic infections of birds of prey. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 420–425.
15. Roberts, K.Z.; and Cray, C. 1998. An update on the application of aspergillosis antigen diagnostic testing. In Proceedings of the American Association of Avian Veterinarians. Omaha, Nebraska, pp. 95–97.
16. Shepherd, M. 1998. Case report: Severe mycotic air sacculitis in a palm cockatoo isolated to the cervical air sacs. In Proceedings of the American Association of Avian Veterinarians. Omaha, Nebraska, pp. 233–237.
17. Werther, K. 1993. Bewertung der Radiographie zur Diagnose de Lungen-Luftsack-Mykose bei Psittaciformes. DVM thesis, Tierärztlichen Fakultät der Ludwig-Maximilians-Universität München.

NONINFECTIOUS DISEASES

Karin Werther

Poisoning

For the correct diagnosis of intoxication meticulous clinical history is indispensable. The most frequent toxicity observed in Psittaciformes in captivity, principally the pet birds, is heavy metal poisoning (lead, zinc, and other) that the bird acquired by ingesting diverse metallic objects in its environment, such as galvanized wire, foil from champagne bottles, or by having access to fishing weights. The animals show characteristic signs, such as anorexia, apathy, depression, incoordination, vomiting, polyuria, and polydipsia, and blood in feces or in urine. Radiographs showing the metallic particle are important in making a diagnosis.

Treatment includes chelating agents such as calcium ethylenediaminetetraacetate (Ca-EDTA) 20-40 mg/kg body weight, IM, every 8–12 hours, as needed, fluid therapy to increase the renal excretion of the chelated metal, supportive care, vitamin E (0.05-0.1 mg/kg body weight, IM every 24 hours). To eliminate the metal particle, barium sulfate (1 mL/100 g body weight, orally, every 24 hours) is appropriate.

Another important poisoning is with organochlorates or organophosphates. These may be consumed in food or in the environment. Typical signs are anorexia, apathy, depression, incoordination, vomiting, polyuria and polydipsia, shedding of tears, and salivation. Supportive care and treatment with atropine sulfate (0.01-0.02 mg/kg body weight IM or subcutaneously [SC], as needed) will be helpful.

Poisoning from rodenticides that contain anticoagulant products, such as Warfarin, may occur. Birds show depression, anorexia, anemia, blood in the feces and/or urine, eventually vomiting. The antidote, vitamin K (0.2-2.5 mg/kg body weight IM, as needed) and supportive care are recommended.

Metabolic Problems

GOUT Gout (visceral, renal, and at the articulations) is often observed in psittacines. Important factors in causing gout are dehydration, high ingestion of protein, primary renal disorders or insufficiency, and vitamin A deficiency. The success of the treatment is limited by the low uric acid solubility and the high toxicity. Depending on the intensity of the lesions (on the pericardium, other serous membranes, and principally by the renal insufficiency), it may not be possible to treat the bird. The signs are not specific and include anorexia and apathy. Diagnosis by radiology is possible only if there has been calcification of the uric acid crystals.

NUTRITIONAL DEFICIENCY As a consequence of inadequate nutrition offered in captivity for pet birds (eating human food such as: chocolate, popcorn, coffee, milk, rice and beans, spaghetti, corn meal mixture with milk or water, cake, sunflower seeds only, pineapple or fried potatoes), the birds may show typical syndromes. These kinds of foods are poor in proteins, vitamins, and minerals, and also contain high levels of salt and oil. The birds may show diverse problems such as metabolic bone disease, poor growth syndrome, obesity, hypovitaminosis A, greased and matted feathers, polydipsia, polyuria, scaling, and deforming beaks. Radiographs often observe hepatomegaly and nephromegaly. A change of the color of the feathers (achromatosis) may be also associated with a nutritional disorder (Figure 17.6). In some cases arteriosclerosis was diagnosed at necropsy.

FIGURE 17.6. Amazon parrot with dark brown feather on the coat, indicative of a nutritional deficiency.

CHRONIC EXUDATIVE DERMATITIS An important problem that is probably metabolic in nature is chronic exudative dermatitis (CED). Normally it is described in lovebirds, budgerigars, and cockatoos,[18] but it has also been observed in Amazon parrots. The skin lesions are symmetrically bilateral on the internal aspect of the femur (Figure 17.7) or on the patagium on the wings. These lesions are moist, crusty, and heal poorly. Secondary bacterial and fungal infection may occur. The etiology may be a nonspecific hepatopathy that must be resolved before trying to resolve the skin lesions. The hepatopathy is often associated with nutritional deficiency.

REFERENCES

1. Gerlach, H. 1994. Viruses. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 862–948.
2. Ritchie, B.W.; Niagro, F.D.; Lukert, P.D.; Steffens, W.L.; and Latimer, K.S. 1989. Characterization of a new virus from cockatoos with psittacine beak and feather disease. Virology 171:83–88.
3. Ritchie, B.W. 1995. Avian Viruses: Function and Control. Lakeworth, Florida, Wingers Publishing, p. 515.
4. Greenacre, C.B., Latimer, K.S., Niagro, F.D. Campagnoli, R.P., Pesti,D., and Ritchie, B.W. 1992. Psittacine beak and feather disease in a scarlet macaw (*Ara macaw*). Journal of the Association of Avian Veterinarians 6(2):95–98.
5. Huff, D.G.; Schmidt, R.E.; and Fudge, A.M. 1988. Psittacine beak and feather syndrome in a blue-fronted Amazon (*Amazona aestiva*). Association of Avian Veterinarians Today 2:84–86.
6. Latimer, K.S.; Rakich, P.M.; Steffens, W.L.; Kircher, I.M.; Ritchie, B.W.; Niagro, F.D.; and Lukert, P.D. 1991. A novel DNA virus associated with feather inclusions in psittacine beak and feather disease. Veterinary Pathology 28(4):300–304.
7. Werther, K.; Durigon, E.L.; Raso, T.F.; Latimer, K.S.; and Campagnoli, R.P. 1998. First case of psittacine beak and feather disease in Brazil. In Proceedings of the International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds.
8. Guimarães, M.B.; Madeira A.M.B.N.; Allgayer, M.C.; Tadano, A.C.S.; and Matushima, E.R. 1999. Casuística de doença do bico e da pena em psitasídeos atendidos no Ambulatório de Aves-FMVZ-USP no período de janeiro de 1998 a maio de 1999. In Proceedings of the

FIGURE 17.7. Amazon parrot showing typical CED lesions at the internal face of the femur.

3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens, p. 31.
9. Soares, P.; Guimaraes, M.B.; and Durigon, E.L. 1998 The haemagglutination spectrum of psittacine beak and feather disease virus in Brazilian psittacine birds. In Proceedings of the International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds.
10. Phalen, D.N. 1997. Viruses. In R.B. Altmann, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery,. Philadelphia, W.B. Saunders, pp. 281–322.
11. Karpinsky, L.G.; and Clubb, S.L. 1985. Post pox ocular problems in blue-fronted Amazon and blue-headed Pionus parrots. In Proceedings of the Association of Avian Veterinarians. Boulder, Colorado, pp. 91–100.
12. Winterfield, R.W.;,Clubb, S.L.; and Schrader, D. 1985. Immunization against psittacine pox. Avian Diseases 29(3):886–890.
13. Clubb, S.L.; and Esklund, K.H. 1988. Field trials with a killed psittacine pox vaccine. In Proceedings of the Association of Avian Veterinarians. Houston, Texas, pp. 145–152.
14. Altmann, R.B.; Clubb, S.L.; Dorrestein, G.M.; and Quesenberry, K. 1997. Avian Medicine and Surgery. Philadelphia, W.B. Saunders, p. 1070.
15. Gregory, C.R., Ritchie, B.W.; Latimer, K.S.; Steffens, W.L.; Campagnoli, R.P.; Pesti, D.; and Lukert, P.D. 1998. Experimental transmission of psittacine proventricular dilatation disease (PDD) and preliminary characterization of a virus recovered from birds with naturally occurring and experimentally induced PDD. In Proceedings of the International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds.
16. Stedman, N.L.; Latimer, K.S.; and Rakich, P.M. 1998. Cloacal papillomas in psittacine birds: A retrospective histopathologic review. In Proceedings of the International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds.
17. Ritchie, B.W.; Latimer, K.S.; Pesti, D.; Campagnoli, R.; and Lukert, D. 1998. A review of host response to the inactivated polyomavirus vaccine in experimental and field settings. In Proceedings of the International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds.
18. Grimm, F.; and Gylstorff, I. 1987. Vogelkrankheiten. Stuttgart, Verlag Eugen Ulmer, p. 609.

MISCELLANEOUS DISEASES

Maria de Lourdes Cavalheiro

Chlamydiosis

Wild animals are frequently incriminated as carriers of infections and parasitic diseases of domestic animals. Unfortunately, funds to investigate such allegations are rarely available. Until this situation is corrected, it will be difficult to understand the epizootiology of diseases such as chlamydiosis and mycoplasmosis.

Chlamydia spp. are energy-dependent obligate intracellular parasites. Two species of *Chlamydia* normally infect humans (*Chlamydia trachomatis* and *Chlamydia pneumoniae*). Two other species infect animals; *Chlamydia pecorum* causes enteritis, encephalomyelitis, and polyarthritis in ruminants and *Chlamydia psittaci* may cause systemic diseases in many species of reptiles,[8] birds, and mammals, including humans. *C. psittaci* has many serovars. Subspecies or strain-specific variability may be determined through the major outer membrane protein (MOMP).[4]

Disease caused by *C. psittaci* may be called ornithosis, psittacosis, parrot fever, or, most frequently, chlamydiosis. Chlamydiosis is widespread and has been reported in many countries, although its epizootic role is not clearly understood. The zoonotic importance of some serovars remains uncertain.

DEVELOPMENTAL CYCLE Five major phases characterize the *Chlamydia* cycle:

1. Attachment and penetration of a target cell by the elementary body (EB). The EB cannot propagate but is an infectious toxic organism.
2. Transition of the metabolically inert EB into a metabolically active reticulate body (RB). The RB cannot generate a high-energy phosphate bond; thus, its adaptation as an intracellular parasite is clarified due to dependency on eukaryotic cells for energy.
3. Growth and binary division of the RB resulting in many microcolonies with hundreds of chlamydial organisms per cell, named Levinthal-Cole-Lillie (LCL) inclusions
4. Maturation of the noninfectious RB into an infectious EB
5. Release of the EB newly formed from the host cell by lysis within 48 hours after initial infection

CLINICAL SIGNS Clinical signs of chlamydiosis may vary from no signs at all to mild to severe systemic changes that may be affected by host immune competence, pathogenicity of the particular strain of *C. psittaci,* stress factors, or preexisting infections. When present, signs in birds include anorexia, weight loss, diarrhea with greenish to yellowish droppings, low body temperature, lethargy, dyspnea, rales, sinusitis (budgerigars), air saculitis (Psittaciformes), coryza (pigeons), pneumonia, emaciation, and dehydration. Sometimes signs associated with encephalitis may be observed. In koalas, signs may involve intestinal, respiratory, ocular, and reproductive systems, so sterility may be seen in these cases.[12]

Both inapparent and apparent active infections are usually underdiagnosed because chlamydiosis may easily be mistaken for unspecific bacterial pneumonia, mycoplasmal pneumonia, systemic fungal infections, influenza, tuberculosis, Pacheco's disease, Newcastle disease, or tuleramia. It may be difficult for a practitioner to obtain adequate laboratory confirmation. Furthermore, most countries in Latin America face another serious problem—no current regulations for diagnosis, control or treatment. There are no legal determinations for human beings or for animals in quarantine at zoos. For example, in Brazil, in 1998, a serologic test to diagnose human chlamydiosis by immunofluorescence was established in a commercial laboratory in São Paulo State. Unfortunately, it is still much too expensive, and many clinicians do not ask for that test because their patients will not be able to pay for it. In several countries, chlamydiosis is still almost unknown or, at least, is not considered a disease.

HISTOPATHOLOGY In agreement with citations in the literature, recent histopathological findings in two *Pionites leucogastes* received at Foz Tropicana Parque (Paraná State of Brazil) reported hepatitis with multifocal necrosis and histiocytosis, chlamydia-laden macrophages, and the spleen showing accumulation of iron pigment (Zalmir Cubas, personal communication, Foz do Iguacu, Brasil, 1996).

EPIZOOTIOLOGY Chlamydial organisms are usually transmitted by inhalation of EB in aerosolized infected feces. After an incubation period of 48 hours, infection begins in the respiratory system, spreading to the liver and spleen as soon as it becomes systemic. Ingestion and aerosol inhalation are the most important routes of transmission, although parent-to-young transmission through feeding may also be important. The importance and incidence of routes, morbidity, and mortality in the wild is not well known.

Studying red-tailed Amazon parrot (*A. brasiliensis*) free-ranging nestlings in two locations of its habitat, the author found low positive to negative reactions to a serological test[1] even when ectoparasites, such as *Dermanyssus* spp. were found or physiological parameters were considered to be altered. All chicks were clinically normal and developed normally. Clinical pathology findings were decreased packed cell volumes, monocytosis, endoparasitism (*Eimeria* spp.), gram-negative growth of nest substrate samples, gram-negative growth of cloacal samples, and fungal growth (*Candida* spp. and *Aspergillus* spp.) A single nestling showed *Chlamydia* low positive reaction, but some days later, no reaction (paired sera). The other chick from the same nest always tested negative.

Results of this study suggest that transmissions of *Chlamydia* in the wild may be important to maintaining infection in some populations and that infection has reached equilibrium between parasite and host. Nevertheless, such equilibrium is clearly disrupted in such stressful situations as habitat destruction, capture for trade, starvation, malnutrition and inadequate care in captivity, because carriers may begin to shed the organism after such events.

Preliminary work identified no clinical signs in eight Amazon parrots that came from the illegal pet trade, with high ELISA titers[2], latex agglutination results, and hyperproteinemia.[3] Two years later, with no treatment, these parrots were still showing no clinical signs and exhibiting normal behavior. Results of a test with paired sera demonstrated low positive to medium positive reaction, suggesting that latent infection may persist in parrots without producing illness.

Tests by direct immunofluorescence[3] of 95 captive *Amazona* parrots from three states of Brazil indicated a high incidence of EB shedding, as well serological titers that showed the presence of antibodies. All the parrots showed no clinical signs and exhibited normal behavior in their cages.

These recent researches in wild and captive parrots suggest that *Chlamydia psittaci* is widespread among parrots in Brazil and healthy carriers are common. The consequences in regard to human health is yet unknown.

It has been stated that sick untreated birds die within a few weeks. In testing a captive blue-fronted Amazon parrot (*A. aestiva*) that was showing feather damage, yellowish fecal smears, sinusitis, airsacculitis, dyspnea, and anorexia, the author found a high positive serological reaction to *Chlamydia*[7]. When its clinically normal cage partner was tested, results confirmed a low positive reaction. Treatment of both birds with doxycycline in the food was carried out for 50 days. Clinical signs disappeared in the ill parrot within 2 weeks after treatment began. Unfortunately, blood concentration data was not obtained, because such laboratory analysis is not easily available.

Chlamydia psittaci strains from Psittaciformes, turkeys, and ducks appear to cause the most severe disease in humans; however, it does not seem to be easily transmitted from infected birds (even parrots) to healthy human beings.[6]

After epidemiological investigation in several zoos from Brazil and other countries in Latin America, several problems in regard to chlamydial disease appear:

1. No appropriate laboratory tests are available.
2. Usually no histopathological postmortem examination is performed.
3. No quarantine or treatment protocol is in place.

4. No periodic or sporadic care is provided to staff.
5. Almost always, only psittacine birds are suspected.
6. Available treatment is commonly based only on clinical signs.
7. Confirmation through laboratory findings is rare.

The majority of zoo administrators questioned believe that diagnosis and treatment methods in their countries are inaccessible and not efficient. The primary difficulties cited were:

1. No or inadequate financial support for researching diseases in their zoos
2. No qualified laboratory to carry out tests. When there are laboratories, the problem becomes financial.
3. "Inaccessibility" of imported tests (once more related to financial problems)
4. Appropriate treatment medication is not available.

In addition, administrators of some facilities have complained about unknown reported regional cases and inadequate or no instruction about how to obtain samples to send to the laboratory.

Mycoplasmosis

The genus *Mycoplasma* is composed of a group of bacteria that belongs to the class Mollicutes and the order Mycoplasmatales. These bacteria lack a cell wall, replicating primarily by binary fission. The genus includes both pathogenic species and species that are part of the normal microbial flora. They have a circular genome of double-stranded DNA that is smaller than that of most prokaryotes.

The order Mycoplasmatales includes three genera: *Mycoplasma, Acholeplasma,* and *Ureaplasma*. They have been associated with much illness, mainly in avian and mammal species.

Mycoplasmosis is the name of diseases caused by these genera. *Mycoplasma* spp. seem to be the most frequently identified agents of disease. Although these agents produce mild to severe health problems, most studies of mycoplasmosis have been conducted with farm animals, because agricultural economic losses justify and support research. Often, free wild animals living near farms are suspected to be responsible for transmission of disease agents. As with *Chlamydia* spp. the epizootic role of *Mycoplasma* spp. is still unclear.

CLINICAL SIGNS AND EPIZOOTIOLOGY The clinical signs of mycoplasmosis vary widely with the animal species, population density, confinement conditions of animals, and other situations that permit increased exposure of individual animals to a mixture of microorganisms. Generally, morbidity is high but mortality depends upon secondary factors. A study of infectious coryza (caused by *Haemophilus paragallinarum*) in Argentina, suggested that outbreaks may be complicated by the presence of several bacterial genera, such as *Salmonella, Pasteurella, Mycoplasma,* and *Chlamydia.*[11] In Brazil, several farm herds have been identified as carriers of species of *Mycoplasma*.

In avian species, *Mycoplasma synoviae* was identified as an infectious agent to chickens, ducks, geese, turkeys, Japanese quail, pigeons, guinea fowl, falcons, red-legged partridges, and house sparrows. Furthermore, several other avian mycoplasmas (AM) have been isolated: *Mycoplasma anatis, Mycoplasma columbinasale, Mycoplasma cloacale, Mycoplasma columbinum, Mycoplasma columborale, Mycoplasma iners, Mycoplasma meleagridis, Mycoplasma gallopavonis, Mycoplasma gallisepticum, Mycoplasma gallinarum, Mycoplasma gallinaceum, Mycoplasma pullorum, Mycoplasma glycophilum* ,and *Mycoplasma lipofaciens.*[1,9,11] Commonly related clinical signs are airsacculitis and synovitis. In chickens, the agent is transmitted from bird to bird or to chicks in the egg. Transmission between free-flying birds and commercial ones is not clear, but Fiorentin and Jaenisch[2] considered that free-flying pigeons (*Columbina picui*) may play an important role. However, the epizootiology of the infection remains obscure.

Even though results of *Mycoplasma* spp. epidemiological investigations indicate the same fundamental problems and difficulties as with *Chlamydia* spp. epidemiology, it is obvious that there is less concern for mycoplasmal diseases than for chlamydioses, perhaps because *Mycoplasma* infections result in low mortality and seem to become hazardous only in the presence of other infectious agents. Notwithstanding, little financial support, no quarantine or treatment protocol, inaccessibility to tests or medication, and lack of knowledge are, without doubt, the explanation of why so few cases are reported.

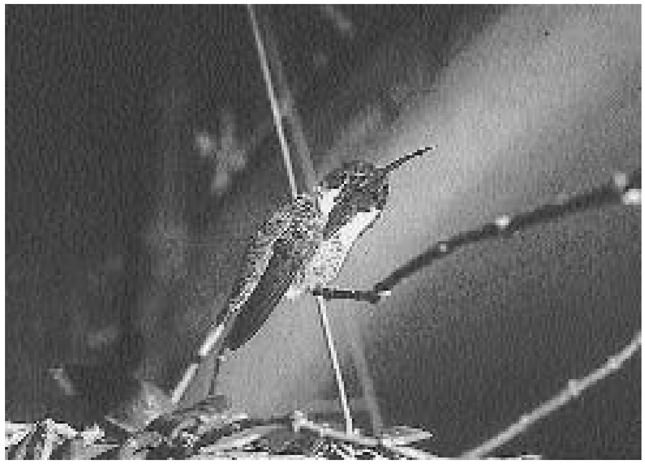
In Latin American countries, knowledge of the frequency and prevalence of mycoplasmal diseases is sparse. It is mandatory that Latin American researchers and practitioners develop both research and treatment protocols and begin more frequent studies of these diseases.

REFERENCES

1. Bencina, D.; Dorrer, D.; and Tadina, T. 1987. *Mycoplasma* species isolated from six avian species. Avian Pathology 16:653–664.
2. Fiorentin, L.; and Jaenisch, F.R. 1994. Tentativa de infecção experimental da pomba-rola (*Columbina picui*)

com *Mycoplasma synoviae* [A trial to infect the free-flying pigeon (*Columbina picui*) with *Mycoplasma synoviae*]. Arquino Brasileiro de Medicina Veterinária e Zootecnia 46(5):573–575.

3. Fontenelle, M.L.C.; and Fontenelle, J.H. 1996. Globulin level correlated with titers of *Chlamydia psittaci* in red-tailed Amazon (*Amazona brasiliensis*), southeastern Brazil. In Proceedings of the American Association of Zoo Veterinarians. Puerto Vallarta, Mexico, pp. 160–168.
4. Gerlack, H. 1994. *Chlamydia*. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 984–996.
5. Graham, D.L. 1989. Histopathologic lesions associated with chlamydiosis in psittacine birds. Journal of the American Veterinary Medical Association 195(11):1571–1573.
6. Harrison, G.J. 1989. A practitioner's view of the problem of avian chlamydiosis. Journal of the American Veterinary Medical Association 195(11):1525–1530.
7. Hung, A.L.; Alvarado, A.; Lopez, T.; Perales, R.; Li, O.; and Garcia, E. 1991. Detection of antibodies to mycoplasmas in South American camelids. Research in Veterinary Science 51(3):250–253.
8. Jacobson, E.R. 1993. Snakes. Veterinary Clinics of North America: Small Animal Practice 23(6):1179–1212.
9. Jain, N.C.; Chandiramani, N.K.; and Singui, I.P. 1971. Studies on avian pleuro-pneumonia-like organisms, 2. Occurrence of *Mycoplasma* in wild birds. Indian Journal of Animal Science 41(4):301–305.
10. Poveda, J.B.; Carranza, J.; Miranda, A.; Garrido, A.; Hermoso, M.; Fernandez, A.; Domenech, J. 1990. An epizootiological study of avian mycoplasmas in southern Spain. Avian Pathology 19:627–633.
11. Sandoval, V.E.; Terzolo, H.R.; and Blackall, P.J. 1994. Complicated infectious coryza outbreaks in Argentina. Avian Diseases 38(3):672–678.
12. Schachter, J. 1989. Chlamydial infections—Past, present, future. Journal of the American Veterinary Medical Association 195(11):1501–1506.



18 Order Trochiliiformes (Hummingbirds)

Kathryn A. Orr
Murray E. Fowler

BIOLOGY

Taxonomy

Hummingbirds are classified in the order Trochiliiformes, family Trochiliidae. There are approximately 650 species, and this is the fourth-largest family in Brazil, which has 150 species in 39 genera. Sixteen species nest within the continental United States.

Distribution

Hummingbirds are native only to the Americas.

Habitat

Habitat is variable according to species. Some are found in temperate climates, but the majority inhabit tropical or subtropical forests. They depend upon a constant source of flowers to provide nectar for nourishment. A given individual may choose selected locations depending on its activity, such as singing, bathing, sunbathing, sleeping, resting, or nesting. During the winter, species adapted to a temperate climate usually migrate to warmer climates, where there are flowering plants.

Anatomy and Physiology

Hummingbirds are some of the tiniest vertebrates in the world, with body weights ranging from 2 to 20 g. The smallest species is the frilled coquette *Lophornis magnifica*. Hummingbirds have the highest metabolic rate of any bird.

They have a shortened humerus and forearm. Half or more of the extent of the wing is composed of carpal and metacarpal bones supporting 10 large primary flight feathers and only 6 or 7 secondaries. The coracoid bones attach to the sternum by a uniquely shallow ball-and-cup socket. Great maneuverability is caused by an almost freely pivotal humeroscapular joint and pectoral muscles comprising 25-30% of the body weight. As well as forward, hummingbirds can fly straight up, down, or backward, and can hover in one place with wing beats of 55–80 per second.[3-9] They have no ankle joint and are not able to walk or hop, rather they only perch or fly.

Hummingbirds feed by extracting nectar from flowers with a long, divided, and grooved tongue mounted in a long, thin, tubular bill. They have a crop for nectar storage, but have no ceca.

Hummingbirds are called the "feathered jewels" of the animal world because of the iridescent feathers on the throat, head, and abdomen of the male. Iridescence is produced by the structure of the barbules and subbarbules, which bring about a prismatic breakdown of light. Feathers with this construction are known as "optical feathers."

Hummingbirds are important as pollinators of a large number of plants. For some plants, hummingbirds are the only pollinator. In other cases they share this function with insects. Hummingbirds also consume large numbers of insects and thus diminish transmission of diseases such as filariasis, yellow fever, malaria, and onchocercosis.

Hibernation

Hummingbirds are extremely active, and in order to prevent exhaustion they have evolved a unique system of torpor to allow "time out" from an otherwise consuming lifestyle. The literature calls this torpid state hibernation, but because it occurs every night, it is not the same as that which may occur in certain rodent species. The normal body temperature of a hummingbird varies from 39–45°C. The heart rate of an active bird may be over 1000 beats per minute. When a hummingbird sleeps, the body temperature may drop 7°C, inducing torpor during which the temperature continues to decrease, reaching as low as 14°C. During torpor the heart rate may slow to 30 beats per minute.[3,5,9]

A hummingbird in a torpid state may be handled for 30–40 minutes without response. When conducting a physical examination it may be difficult to differentiate between a moribund bird and one in torpor.

Migration

Some species do not migrate; some may migrate less than 500 km, and others may migrate as far as 2000 km. Distances of 900 km requiring 20 hours of sustained flight have been recorded. Maximum flight speed is 60 km per hour. During long flights the bird must use its fat reserve. To continue on, the bird must rest and build up a new fat reserve.[3,7-9]

Gender Determination

Hummingbirds are sexually dimorphic, with the male being ornate and the female dull in color.

Longevity

In the free-ranging state, they probably live only 5–8 years. Captive specimens have lived for 16 years.

Predators

In Brazil, the pygmy owl *Glaucidium brasilianum,* a hornet *Campsomeris* spp., and a bird-eating spider *Homoeomma* sp. prey upon hummingbirds. Snakes and lizard may be predators in other areas.[7, 8]

Field Data

Hummingbirds are solitary and highly territorial for both feeding and nesting. Territories are defended with highly aggressive attacks; birds have been known to penetrate the body cavity with their beaks.

In their diet and feeding behavior, each species has preferred flowers for feeding. Feeding is accomplished by hovering, inserting the beak, and extending the tongue into the base of the flower.

Conservation

Population status is unknown for most species. There is no Species Survival Plan (SSP) for hummingbirds, but they are dealt with in the Passeriformes Taxon Advisory Group (TAG), in the United States.*

Reproduction in the Wild

Male and female hummingbirds unite only for courtship and copulation. Courtship flight displays are elaborate and require the full coloration of the male to interest the female. Polygamy is the rule. Females build compact, small nests out of plant material and spider webs. Two white eggs are laid per clutch, but two or three clutches may be produced in a season. Incubation is 13–17 days and is performed only by the female, as is the care of the nestlings. Fledging takes place at 20–35 days.

MANAGEMENT IN CAPTIVITY

Housing

No minimum housing requirements have been established. One zoo provides a walk-through aviary approximately 3.0 × 5.0 m (10 × 15 feet) with extensive plantings and a continuously flowing waterfall and pond. A private aviculturist maintains hummingbirds in flight cages of 5.5 m long × 0.76 m wide and 1.8 m high. (18 × 2.5 × 6 ft), but states that birds may be adequately housed in flight cages only 3.7 m (12 ft) in length. The length is more important than width or height to allow birds to exercise.[5] Sheltered housing must be available to allow the birds to retreat during cold or excessively hot weather. Perches may be supplied either by plantings or strategically located dowels. Bathing facilities must be provided by a flowing stream or by shallow saucers placed on platforms and kept filled with water.

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If the flight cage has a concrete base, fecal material may be hosed off daily. Otherwise, newspaper may be placed on the floor to collect droppings. Newspaper should be replaced daily.

Environment

Optimal ambient temperatures are species dependent. Species adapted to a temperate climate are more cold tolerant than tropical species. Unusually low temperatures for a given species may precipitate torpidity.

Reproduction in Captivity

Many species have been bred in captivity. Sufficient space to cope with territoriality is necessary.[5] Chicks have been hatched in artificial incubators and reared by hand. A Pasteur pipette may be used to simulate the beak of the mother. The chicks are altricial, thus close attention to maintaining optimum ambient temperatures is required. Neonatal problems are similar to those encountered in psittacine hand-rearing. Scrupulous cleanliness must be practiced to avoid candida and other microorganism contamination.

Feeding

Nutrient consumption is primarily carbohydrates. Different species consume from 6-30 times their body weight daily, depending on the degree of activity. Nectars selected by hummingbirds in the free-ranging state must contain 15–25% sugars. Artificial diets generally contain 20–25% sugar. Sugar water alone is not sufficient to sustain these birds. In the wild they also consume insects and spiders. In captivity, insects such as the fruit fly *Drosophila* spp. may be released into an enclosure. A human protein dietary supplement, such as Gevral Protein, may be another source of protein. An envelope of the supplement may be added to 5 L of the sugar solution. A number of commercial hummingbird nectars are available, but the formulation must contain adequate protein.[3,4]

Sufficient feeding stations to provide a source for every two birds should be installed.

RESTRAINT, ANESTHESIA, SURGERY

It is extremely difficult to capture a swift-flying hummingbird that is capable of reversing flight instantaneously. The room should be darkened when capture is attempted. It is desirable to move the bird to a small enclosure, then a small net may be used to catch it. In free-flight aviaries, a mist net may be used. Once captured, the bird may be restrained by cupping it gently in the hand. None are aggressive or capable of injuring the handler.[2]

Isoflurane inhalation is the anesthetic of choice for diagnostic and surgical procedures. An anesthetic chamber or facemask attached to an isoflurane vaporizer is required. The open drop method that is sometimes used with methoxyflurane (Metofane) should not be employed.

Transportation of these birds is always a challenge. They may be loosely wrapped in a cloth jacket or "dressing gown," with the heads protruding so they can feed. They seldom struggle when their wings are secured. A hole just large enough for the head should be cut in the center of the cloth, which is fastened securely around the bird's wings and body by tying the ends below the legs or pinning it above the back. A similar jacket may be employed for immobilization for treating fractures.[4] Nectar must be constantly available. Hummingbirds have also been transported by inducing torpor by lowering the ambient temperature.

Surgery

The most likely reason for surgical intervention is trauma to skeletal structures. Fracture repair of long leg bones may be carried out by using a tape splint like those employed to repair similar fractures in budgerigars or other small birds.

DISEASES

Nutritional Diseases

Starvation from insufficient calorie intake and hypoproteinemia caused by failure to provide an adequate protein source in artificial food are the two most likely nutritional problems.

Parasitic Diseases

Although tapeworms and nematodes have been found in the intestinal tracts of hummingbirds, parasitic disease is rare in these species. It is difficult to establish the presence of diarrhea in hummingbirds because the feces is liquid normally.

Infectious Diseases

Hummingbirds have no unique infectious diseases. Characteristics of selected diseases are found in Table 18.1.[4,9]

Perhaps the most common, and serious, disease of captive hummingbirds is candidiasis of the tongue and oral cavity. The characteristic candida lesion is a catarrhal-to-mucoid exudate consisting of raised,

TABLE 18.1. Selected infectious diseases of hummingbirds

Disease (English)	Disease (Spanish)	Disease (Portuguese)	Etiology	Signs	Diagnosis	Management	Prevention
Tuberculosis	Tuberculosis	Tuberculose	*Mycobacterium avium/ M. intracelluare*	Emaciation,weakness, dyspnea	Acid fast stain of histopathology	Sanitation	Sanitation
Salmonellosis	Salmonelosis	Salmonelose	*Salmonella* spp.		Culture	Antibiotics in food	Sanitation, quarantine
Candidiasis	Candidiasis	Candidiose	*Candida albicans*	Oral plaques, inability to eat, necrosis of tongue	Direct smear	Antibiotics	Cleanliness of food and feeders. Some managers feel that the use of honey in food contributes.
Aspergillosis	Aspergilosis	Aspergilose	*Aspergillus fumigatus*	Dyspnea	Direct smear and culture	Prognosis is poor	Sanitation

white mucosal plaques and whitish-to-clear mucus in the oral cavity, crop, and on the tongue.[5] Clinical signs include dysphagia and regurgitation. The bird may insert the entire beak into the nectar dispenser and then have difficulty swallowing. Subacute and chronic infection may result in partial necrosis of the tongue and beak deformities.

Candida is frequently a secondary invader, particularly in birds that are immunosuppressed. Stress is an important consideration in captive hummingbird care and should be kept in mind when treating the bird. Steps should be taken to minimize stress. Nystatin (Mycostatin, Mystecilin) is the drug of choice for treating candidiasis. Various regimens have been suggested. Aviculturists administer one drop of a suspension (100,000 IU/mL) to the bird orally once a day. Clinical avian veterinarians in the United States recommend 250 IU/g body weight by mouth, twice a day, which is twice the aforementioned daily dose. Tablets may be dissolved in water to a concentration of 50,000 IU/mL then administered at 0.13 mL/15 g body weight orally, twice a day. Other antifungal agents that have been used to treat candidiasis in other caged birds include amphotericin B, ketoconazole, itraconazole, miconazole, fluconazole, and flucytosine.[1]

Opportunistic gram-negative bacterial infections may occur as in other avian species. In hummingbirds a common site for abscess formation is around the base of the beak. Poor sanitation and lack of bathing facilities provide conditions conducive to abscessation.

Conjunctivitis may be observed in recently acquired birds as they adapt to a new diet and feeders. Various ophthalmic ointments have been instilled into the conjunctival sac to correct the condition.

Oral medication may be easily administered to hummingbirds in the nectar mixture, but it is difficult to determine the quantity of nectar consumed, in order to calculate the appropriate amount of medication. A sick bird may also have diminished appetite.

Noninfectious Diseases

TRAUMA Hummingbirds may fly into large glass windows, causing concussion; fractures of the head, neck, and legs or injury to the keel (sternum). Such a keel injury is called “split breast” by aviculturists and may also occur during transit from one location to another. If the laceration is minimal, it will heal in a few days, as long as the environment is such that reinjury does not occur.

HEPATIC LIPIDOSIS The etiology of this malady is unknown, but it is often associated with obesity. The ability to store fat is a requirement for hummingbirds that undergo migration. When the fat reserve is depleted, the bird must rest and restore fat before the journey can continue. These birds have the ability to quickly mobilize fat and transfer it to the liver for lipolysis to convert the fat into fatty acids for metabolic utilization. Lack of exercise may contribute to fatty infiltration of the liver to the extent that normal liver function is inhibited.

Hepatic lipidosis may be reversed by the addition of choline, methionine, vitamin B_{12}, and/or inositol to the nectar. The diet should be adjusted for a lower caloric intake and arrangements provided to increase exercise.

TORPIDITY A special challenge for clinicians is the evaluation of torpor in hummingbirds. Torpidity is employed by hummingbirds in order to conserve energy, usually at night or when the ambient temperature is lowered. If a bird becomes torpid during the daytime, it usually means that the bird is not obtaining enough food or is ill. A bird in torpor sits huddled on a perch and is cold to the touch. Such a bird should be handled with care by enclosing it in the cupped hand. If the bird clings to the perch, let the warmth of the hand bring the bird’s temperature up to normal. Offer nectar by placing the tip of its bill into a feeder. If the bird is in simple torpor, it will revive quickly and may be released into its enclosure. If the bird fails to respond, additional physical examination may be necessary.[5]

STRESS Captive hummingbirds may live in a constant state of low-grade stress. Some birds fail to adapt to the captive situation and instead may cling to the wire of a cage or hide in a planting. If multiple birds are in an enclosure, a bird may be harassed by its cage mates, especially if there are insufficient feeders. An occasional mock battle is normal behavior for hummingbirds, but if one bird is constantly persecuted by another, it is stressful. Management of stress requires correction of the environmental factors that foster stress.[5]

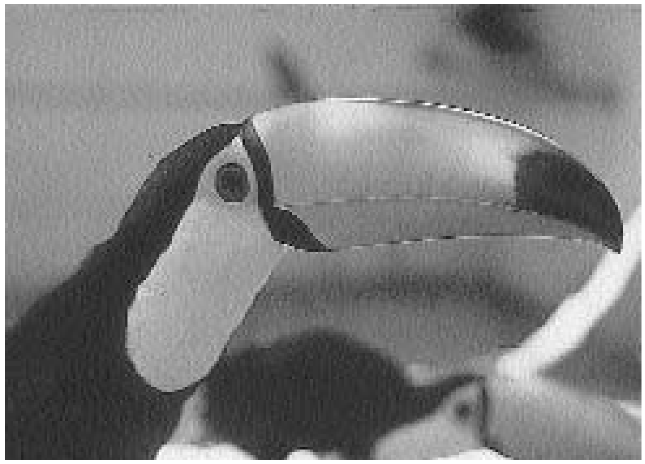
MOLTING Hummingbird molting occurs annually and is usually not a problem. Primary flight feathers are lost a few at a time to enable the bird to fly. If molting is abnormal, the bird may not be able to leave the perch and a feeder must be placed within easy reach. Adequate protein intake is essential during the molt. Offering live fruit flies enhances normal molt.[5]

Preventive Medicine

No vaccines or routine use of parasiticides are recommended for hummingbirds. Cleanliness in food storage and preparation is critical to avoid fungal and yeast contamination. Provision of adequate space to allow territorial behavior without continual aggression is important.

REFERENCES

1. Bauck, L. 1994. Mycoses. In W. R. Branson, G.J. Harrison, and L.R. Harrison, eds.,. Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 997–1006.
2. Fowler, M.E. 1995. Restraint and Handling of Wild and Domestic Animals, 2nd Ed. Ames, Iowa, Iowa State University Press, pp. 307, 327.
3. Greenwalt, C.H. 1960. Hummingbirds. Garden City, New York, Doubleday.
4. Ingram (Orr), K. 1986. Hummingbirds and miscellaneous orders. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 447–456.
5. Mobbs, A.J. 1982. Hummingbirds. Hindhead, England, Saiga Publishing.
6. Ruschi, A. 1982. Beija-flores do Estado do Espiritio Santo [Hummingbirds of the state of Espirito Santo]. Rio de Janiero, Museu de Biologia, EXPED-Expansão Editorial.
7. Ruschi, A. 1982. Aves do Brasil, Vol. 4. Beija-flores [Hummingbirds]. Rio de Janiero, Museu de Biologia, EXPED -Expansão Editorial.
8. Ruschi, A. 1982. Aves do Brasil, Vol. 5. Beija-flores [Hummingbirds]. Rio de Janiero, Museu de Biologia, EXPED -Expansão Editorial.
9. Tyrrell, E.Q.; and Tyrrell, R.A. 1985. Hummingbirds, Their Life and Behavior. New York, Crown Publishers.



19 Order Piciformes (Toucans, Woodpeckers)

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BIOLOGY

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CHARACTERISTICS OF PICIFORM BIRDS

Piciforms are characterized by zygodactyl toes, that is, toes 2 and 3 pointing forward and toes 1 and 4 pointing backward. They also have a special arrangement of the toe tendons and leg muscles, a lack of basipterygoid processes in the skull, a syrinx with one pair of tracheobronchial muscles, the presence of 14 cervical vertebrae, a lack of down feathers in adults, hole-nesting habits, and they lay white eggs.[62]

TAXONOMY AND DISTRIBUTION OF SOUTH AMERICAN PICIFORMES

The order Piciformes is usually divided into five families: Galbulidae, Bucconidae, Capitonidae, Ramphastidae, and Picidae. According to Stotz et al.[78] there are 172 piciform species (17 Galbulidae, 32 Bucconidae, 12 Capitonidae, 34 Ramphastidae, and 77 Picidae) in South American countries.

BIOLOGY OF PICIFORMES

Galbulidae

GENERAL CHARACTERISTICS Jacamars have glittering metallic plumage coloration, long bills, small feet, and long, graduated tails. The sexes are usually separated by the color of the throat. The voice is composed of melodious whistles.[23,64]

HABITAT AND DISTRIBUTION Jacamars are sedentary birds restricted to the Neotropical region, ranging from northern Argentina to southern Mexico. They depend on wooded lands, and several species are found along rivers or streams in forests.[23,64]

DIET AND FORAGING BEHAVIOR The diet of Galbulidae is basically composed of insects captured in flight. They make aerial sallies from a perch to catch passing insects and return to the same perch. The diet is usually composed of large prey, including mainly Lepidoptera (butterflies) and Hymenoptera (wasps), but they also eat Coleoptera, Hemiptera, Diptera, and

Homoptera.[45,58] Before consumption, large prey is pounded on the perch several times to break its wings and other hard chitinous body parts. They regurgitate pellets containing chitinous waste.[11,64]

Where several species coexist, they avoid competition by occupying different ecological niches: larger species hunt in the canopy, whereas smaller species feed on the lower and middle levels; some are restricted to the forest interior whereas others prefer to hunt along the forest edges; and some prey mainly from foliage.[23]

NESTING AND BREEDING BEHAVIOR Using their bills, jacamars excavate nest burrows in earth banks or termite mounds. They remove loose particles of earth with their feet. The nest is usually located in the forest or along river courses. Nest construction is performed by a pair or a group of up to five birds (probably a family). They lay two to four white eggs. The incubation period is 20–23 days, and both sexes incubate. The young are hatched covered with dense down, unlike other Piciformes that are born naked. They leave the nest within 21–26 days and can be recognized by their shorter bills.[11,23,64]

Bucconidae

GENERAL CHARACTERISTICS Bucconidae species are generally called puffbirds because of their fluffy but dull plumage. The head is large, with prominent eyebrows; the iris and the eyelids may be colorful in adult birds; the bill is strong and colorful, and there are whiskers around it; the legs and wings are short; the feet are small; and the tail is narrow. The sexes are alike, but the female may be slightly larger than the male and has duller plumage. Immature birds have shorter bills. The voice is similar to that of Galbulidae, and they can perform duets or choruses.[64]

They may sit motionless for long periods while watching the surroundings. They live in families and sleep among the foliage. Some species may make seasonal migrations. [64]

HABITAT AND DISTRIBUTION Puffbirds are forest birds that inhabit an area from Mexico to southern Brazil. [64]

DIET AND FORAGING BEHAVIOR Bucconidae species are fly-catching insectivorous birds. Like Galbulidae, some species capture flying insects, whereas others capture insects from foliage, tree trunks, and branches. The size of prey varies, and they eat several other arthropods as well as small lizards and vegetable food. Chitinous material is regurgitated in pellets.[11,45,58,64,75]

NESTING AND BREEDING BEHAVIOR Puffbirds nest in burrows excavated in earth, arboreal termite mounds, or rotten wood. The incubation chamber may be bare or lined with dry leaves. They lay two to three shiny white eggs that are incubated by both sexes for approximately 15 days. The young are altricial, with small heel pads. There is no nest sanitation. The pair may receive help in feeding the young, which leave the nest within 20 days.[11,64,75]

Capitonidae

GENERAL CHARACTERISTICS Usually, barbets are brightly patterned, heavy-bodied birds with a deep, broad, pointed bill. The head is large; the bill is surrounded by whiskers; the tongue is relatively long and brush tipped (like toucans) in the most frugivorous species; and the legs are short, but the feet are large. Most Neotropical barbets are sexually dichromatic in contrast with Afrotropical species. The voice is low and harsh, and the pairs perform duets.[62-64]

HABITAT AND DISTRIBUTION Barbets occupy the tropical regions of Asia, Africa, and South America. Neotropical barbets are largely frugivorous forest-dwelling birds, concentrated in the Amazon region. Their distribution reflects a series of forest refuges proposed for this region, because unlike their Afrotropical counterparts, Neotropical barbets are not prone to traverse woodland or open grassland, being true forest species.[62-64] According to Short,[63] "the knowledge of the biology of Neotropical capitonids is poor compared with that of Afrotropical and Asian species."

DIET AND FORAGING BEHAVIOR Barbets forage in the canopy, sometimes in mixed species flocks. They are largely frugivorous, but include arthropods and even small vertebrates in their diet, especially during the breeding season.[58,62-64] There are also records of flower eating,[51,54] which is consistent with a frugivorous diet.

According to Burton,[11] Capitonidae "are the least specialized family of the Piciformes, yet they show some interesting modifications of feeding apparatus structure that suggest possible ways in which the more extreme specializations of other families originated." Remsen et al.[50] analyzed the diet of Neotropical barbets based on museum specimens and concluded that *Eubucco* and *Capito* are less frugivorous than *Semnornis,* which resemble toucans more than New World barbets.

NESTING AND BREEDING BEHAVIOR They may excavate their own nests in rotten wood or occupy woodpecker cavities. Most barbets use their bills for nest excavation, but unlike Picidae, they carry the debris away (like toucans do). The eggs are white and shiny. The young are altricial, hatching after a 13-day incubation.[11,64]

The heel pads are large, and the development of the young is slow. Both characteristics are shared by Ramphastidae and are related to cavity nesting and/or the frugivorous diet, as discussed by Riley.[53] Heel pads occur in most cavity-nesting birds, but are most developed in Capitonidae, Indicatoridae, Ramphastidae, and Picidae.[22] They disappear by the time of the first postjuvenile molt.[3]

Ramphastidae

GENERAL CHARACTERISTICS Toucans are colorful, with a long beak and brush-tipped tongue. The functions of the bill and tongue are unknown, although several hypothesis have been formulated.[3,13,14,16,23,66,77,83,87]

The periophthalmic region is nude and brightly colored; the legs and feet are strong; and the tail is long and graduated in most genera, except *Ramphastos*. Their songs consist of rather simple croaking or yelping calls. The sexes are alike, except in *Pteroglossus viridis* and *Selenidera* spp., but males usually have larger bills.[64]

Toucans live in pairs or in groups. The larger toucans sleep among the foliage, whereas toucanets sleep in holes (they may also be used as nests). They assume a peculiar position to sleep, resting the bill on the back and covering it with the tail.

HABITAT AND DISTRIBUTION Toucans inhabit tropical South and Middle America from southern Mexico to northern Argentina. Haffer[23] studied the speciation of Ramphastidae and verified that the center of abundance of this family is located in the western Amazonian forests, where seven species coexist. These sympatric species, however, differ significantly in body size and bill length. Geographic exclusion of closely related species resulted in a high number of subspecies.

Toucans are typical forest birds, although some species may live in dry woodland or even in nonforest regions, inhabiting gallery forests or savannas. Most toucans occupy the lowlands, but some genera (*Aulacorhynchus* and *Andigena*) are restricted to higher elevations.[23]

DIET AND FORAGING BEHAVIOR Toucans are primarily frugivorous, but also consume arthropods and small vertebrates. They may also consume leaves[58] and flowers.[54] Small fruits are plucked with the mandible tips, then tossed back with an upward jerk of the head. Large insects are rubbed on the perch and held under the feet to remove the wings and legs before consumption. Small seeds are eliminated in the feces and large seeds are regurgitated.

Insects and other animal matter are consumed mainly during the breeding season. Species of toucans are among the most important seed dispersers in the tropical region.[27-29,42,52,55]

NESTING AND BREEDING BEHAVIOR Toucans nest in natural cavities in tree trunks or in woodpeckers' nests, which they may occupy even before the owners abandon it.[34,70] In seriously disturbed forests, where large trees are not available, they nest in fence poles or even in earth banks (P. Scherer-Neto, personal communication; author's personal observation). They may enlarge the nest door or chamber by hammering the wood and carrying the debris away.

The clutch consists of two to four dull white eggs. Both sexes incubate for approximately 16-18 days. The young are altricial with large heel pads and leave the nest in 42-47 days. At fledging they are smaller (especially the bill) than adult birds and the coloration is dull.[59,60]

CAPTIVE BREEDING AND MANAGEMENT Toucans are popular as aviary birds, not only because of their exotic appearance, but also because they are restless and playful. Captive breeding is possible, but has not been highly successful. Regardless of the physical environment, the most important factor to obtain a successful breeding seems to be pair formation and bonding, because captive toucans are aggressive.

A common event in captive breeding of toucans is the killing or mutilation of the young by the parents.[39] Although captive breeding occurs under artificial conditions, some details of pair formation and bonding, egg laying and incubation, and the development of the young were much better understood after captive studies and are important in the conservation of toucans.[37,38,40] Captivity did not change toco toucans' behavioral patterns (55 identified and described) qualitatively, but the frequency and duration of several activities was clearly altered.

Picidae

GENERAL CHARACTERISTICS Woodpeckers are characterized by a rather straight, often chisel-shaped bill and a long, hard-tipped and extensible tongue. In addition to the unique bill, they have adaptations in the skull and neck to permit hammering in wood. The tongue is used to catch insects inside wood or termite mounds, facilitated by a mucous coating. Woodpeckers have diverse combinations of color and sexual dichromatism, mainly on the head. The legs are short and the hard tail helps to support the bird when climbing trees in the upright position. They range in weight from about 7–700 g.[62,64]

Generally, woodpeckers are resident, permanently territorial birds. They sleep in holes and may live in groups (*Melanerpes* spp.). To communicate, a number of vocalizations are used in varying circumstances, as well as a typical instrumental sound (drumming) and diverse visual signals, including displays.

HABITAT AND DISTRIBUTION Woodpeckers are distributed almost worldwide, and the Neotropical region is particularly rich in species. They are forest species, with the exception of *Colaptes* spp., which live in more open areas.[62,64]

DIET AND FORAGING BEHAVIOR Their salivary glands and tongue barbs form an efficient insect-catching device for woodpeckers, but their diet is diverse, including insects (adults, larvae, and eggs), arthropods, fruits, seeds, nuts, honey, and sap.[62,64] Some species of the genus *Melanerpes* may store acorns and other food items, even in tropical regions.[76,93] Where several species coexist, they occupy different strata or employ different techniques to obtain food.[64]

NESTING AND BREEDING BEHAVIOR Woodpeckers nest in tree cavities (one or more cavities), which the pair build each breeding season. They lay two to four shiny white eggs that are incubated by both sexes. The incubation period is short (11–14 days). Hatchlings are altricial. The nest is kept clean except in fruit-eating species (whose feces are usually less firm). The nestling period is 18–35 days or longer, depending on the size of the species. Ant-foraging species feed their young by regurgitation and at long intervals, whereas more frugivorous species feed the young almost constantly.[62] The latter have helpers that, similar to toucans, are the young produced in the previous year. The presence of helpers in one cooperatively breeding species (*Picoides borealis*) was associated with improved nestling survival.[47]

CONSERVATION AND MANAGEMENT OF PICIFORMES

Because all Neotropical piciforms are hole-nesting forest birds, the conservation of mature forest ecosystems is essential for their survival. Eventually some forest-dwelling species are able to find food in secondary habitats, but only large tracts of mature forest have old trees large enough to provide nesting cavities. Even in undisturbed habitats there is keen competition (both intra- and interspecific) for nesting sites, and it is largely increased in disturbed areas. A safe and sound nest site reduces nest predation significantly. Predation may disturb bird populations dramatically (personal observation).

The Neotropical region is particularly rich in frugivorous birds and the order Piciformes includes two families (Capitonidae and Ramphastidae) that are specialized in frugivory and a third (Picidae) that includes fruit in its diet. Frugivory and seed dispersal are essential for the maintenance of the spatial heterogeneity of plant species and ecosystem integrity.[30,31] Large frugivorous forest birds, including toucans, are among the most endangered avian groups in the neotropics.[79] In forest remnants of southeastern Brazil several species of large-canopy frugivorous birds disappeared long ago.[90]

Toucans are endangered in most areas because of habitat destruction, hunting, and illegal trade. They have traditionally been maintained as pets by native peoples,[14,20] who also hunted them for their meat and feathers.[8,57,64,80,85] The beak is used in popular medicine[64] and as a trophy.[84]

Some woodpeckers are also endangered, primarily because of habitat destruction. They are key species in forest ecosystems because they provide nests for other birds, mammals, reptiles, amphibians, and arthropods. They also consume large amounts of insects and their larvae that attack forests and citrus plantations.[64] But such benefits are promptly forgotten when some species (regarded as pests) arrive in orchards to eat fruit.[2,58]

Because of their ecological and economic importance, the conservation and monitoring of piciforms is an essential part of the management of Neotropical protected areas. In the forest remnants of southern Brazil, the author has been monitoring populations of toucans and some species of woodpeckers (along with other large frugivorous bird species) for almost 10 years. Small isolated fragments show dramatic declines in their populations, and some management guidelines are being proposed to protect and eventually restore them.

REFERENCES

1. Alabarce, E.A. 1981. Estudio comparativo de la porción superior del tracto digestivo y alimentación de dos pícidos de la Província de Tucumán [Comparative study of the digestive tract and diet of the woodpeckers of Tucuman]. Acta Zoologica Lilloana 36(2):129–137.
2. Alvarez del Toro, M. 1980. Las Aves de Chiapas [Birds of Chiapas]. Mexico, Universidad Autonoma de Chiapas.
3. Austin, O.L., Jr. 1961. Birds of the World. New York, Golden Press.
4. Beebe, W.; Hartley, G.I.; and Howes, P.G. 1917. Tropical Wildlife in British Guiana. New York, New York Zoological Society.
5. Beecher, W.J. 1953. Feeding adaptations and systematics in the avian order Piciformes. Journal of the Washington Academy of Sciences 43:293–299.

6. Beltrán, W. 1994. Natural history of the Plate-billed Mountain Toucan *Andigena laminirostris* in Colombia. Miscellaneous Publication No. 2. San Antonio, Texas, Center for the Study of Tropical Birds.
7. Beltzer, A.H.; Amsler, G.P.; and Neffen, M.I. 1995. Biología alimentaria del carpintero real *Colaptes melanochloros* (Aves: Picidae) en el valle aluvial de Río Paraná, Argentina [Feeding behavior of *Colaptes melanochloros* in Argentina]. Anales de Biología 20:53–59.
8. Bennet, C.F., Jr. 1962. The Bayano Cuna Indians, Panamá: An ecological study of livelihood and diet. Annals of the Association of American Geographers 52(1):32–42.
9. Berry, R.J.; and Coffey, B. 1976. Breeding the sulphur-breasted toucan, *Ramphastos s. sulfuratus,* at Houston Zoo. International Zoo Yearbook 16:108–110.
10. Brehm, W.W. 1969. Breeding the green-billed toucan, *Ramphastos dicolorus,* at the Walsrode Bird Park. International Zoo Yearbook 9:134–135.
11. Burton, P.J.K. 1984. Anatomy and the evolution of the feeding apparatus in the avian orders Coraciiformes and Piciformes. Bulletin of the British Museum of Natural History (Zoology) 47(6):331–443.
12. Chebez, J.C. 1994. Los que se Van: Especies Argentinas en Peligro [Endangered species of Argentina]. Buenos Aires, Albatros.
13. Chenery, E.M. 1956. The sulphur and white-breasted toucan. Journal of the Trinidad Field Naturalists' Club 1956:4–11.
14. Crandall, L.S. 1940. Everybody knows a toucan. Bulletin of the New York Zoological Society 43(2):35–47.
15. Dye, S.E.; and Morris, A. 1984. Attempted breeding of the toco toucan, *Ramphastos toco,* at Penscynor Wildlife Park, Cilfrew, Neath, Wales. Avicultural Magazine 90(2):73–75.
16. England, M.D. 1975. Birds of the Tropics. London, Hamlyn.
17. Evans, K.; and Coles, D. 1982. Breeding the crimson-rumped toucanet, *Aulacorhynchus haematopygus,* at Padstow Bird Gardens (Cornwall). Avicultural Magazine 88(4):193–198.
18. Frisch, J.; and Frisch, J.D. 1964. Aves Brasileiras [Brazilian Birds]. São Paulo, Irmãos Vitale.
19. Goeldi, E.A. 1894. As Aves do Brasil [Birds of Brazil], Pt. 1. Rio de Janeiro, Livraria Clássica de Alves.
20. Goodfellow, W. 1900. A naturalist's notes in Ecuador. Avicultural Magazine 6:120–128.
21. Gould, J. 1854. A Monograph on the Ramphastidae, 2nd Ed. London, J. Gould.
22. Gyldenstolpe, N. 1917. Notes on the heel-pads in certain families of birds. Arkiv fuer Zoologi 11(12):1–15.
23. Haffer, J. 1974. Avian Speciation in Tropical South America. Cambridge, Nuttall Ornithological Club.
24. Hansen, E.P. 1997. Breeding the red-billed toucan, *Ramphastos tucanus,* at Reid Park Zoo, Tucson. International Zoo Yearbook 35:253–256.
25. Hilty, S.L. 1985. Distributional changes in the Colombian avifauna: A preliminary blue list. Ornithological Monographs no. 36 (Neotropical Ornithology): 1000–1012.
26. Höfling, E.; and Gasc, J.P. 1982. Análise dos movimentos do bico em tucanos do gênero Ramphastos (Ramphastidae, Aves) [Analysis of the movements of toucans' beak]. Anais da Academia Brasileira de Ciências 54(4):755–756.
27. Howe, H.F. 1977. Bird activity and seed dispersal of a tropical wet forest tree. Ecology 58:539–550.
28. Howe, H.F. 1981. Dispersal of neotropical nutmeg (*Virola sebifera*) by birds. Auk 98:88–98.
29. Howe, H.F. 1982. Fruit production and animal activity at two tropical trees. In E. Leight, Jr., A.S. Rand, and D. Windsor, eds., The Ecology of a Tropical Forest: Seasonal Rhythms and Long-term Changes. Washington, D.C., Smithsonian Institution Press, pp. 189–199.
30. Howe, H.F. 1984. Implications of seed dispersal by animals for tropical reserve management. Biological Conservation 30:261–281.
31. Howe, H.F. 1986. Seed dispersal by fruit-eating birds and mammals. In D.R. Murray, ed., Seed Dispersal. New York, Academic Press, pp. 123–190.
32. Hughes, R. 1988. Hand-rearing the crimson-rumped toucanet, *Aulacorhynchus haematopygus,* at Padstow Bird Gardens, Cornwall. Avicultural Magazine 94(4):183–189.
33. Johnson, R. 1977. Three toco toucans hatched. International Zoo News 148:34.
34. Kilham, L. 1972. Habits of the crimson-crested woodpecker in Panama. Wilson Bulletin 84(1):28–47.
35. Lanyon, S.M.; and Zink, R.M. 1987. Genetic variation in Piciform birds: Monophyly and generic and familial relationships. Auk 104(4):724–732.
36. Leck, C.F. 1972. Seasonal changes in feeding pressures of fruit- and nectar-eating birds in Panama. Condor 74:54–60.
37. Mikich, S.B. 1988. Sobre o Comportamento do Tucanuçu, *Ramphastos toco,* em Cativeiro (II): Etograma e Dados Quantificados (Ramphastidae, Aves) [Behavior of toco toucans in captivity]. Bachelors thesis, Universidade Federal do Rio Grande do Sul.
38. Mikich, S.B. 1991. Análise quantitativa do comportamento de *Ramphastos toco* em cativeiro (Piciformes: Ramphastidae) [Quantitative analysis of the behavior of toco toucans in captivity]. Abstracts of the 1° Congresso Brasileiro de Ornitologia. Belém, Museu Paraense Emílio Goeldi, p. 5.
39. Mikich, S.B. 1991. Comportamento reprodutivo de *Selenidera maculirostris* em cativeiro (Piciformes: Ramphastidae) [Breeding behavior of Spot-billed toucanets in captivity]. Abstracts of the 1° Congresso Brasileiro de Ornitologia. Belém, Museu Paraense Emílio Goeldi, p. 5.
40. Mikich, S.B. 1991. Etograma de *Ramphastos toco* em cativeiro (Piciformes: Ramphastidae) [Ethogram of toco toucans in captivity]. Ararajuba 2:3–17.
41. Mikich, S.B. 1992. A importância da estatística nos estudos bioecológicos: Análise do isolamento ecológico em ranfastídeos (Piciformes: Ramphastidae) [The importance of statistical analysis in the study of ecological isolation in toucans]. Abstracts of the 2° Congresso Brasileiro de Ornitologia. Campo Grande, UFMS. R54.

42. Mikich, S.B. 1994. Aspectos de Comportamento, Frugivoria e Utilização de Hábitat por Tucanos de uma Pequena Reserva Isolada do Estado do Paraná, Brasil (Ramphastidae, Aves) [Behavior, frugivory and habitat utilization by toucans in a small isolated reserve in southern Brazil]. Masters thesis, Universidade Federal do Paraná.
43. Mikich, S.B. 2000. Frugivory by Picidae in forest remnants of south Brazil(Piciformes: Aves). Manuscript.
44. Mikich, S.B. 2000. The frugivorous diet of neotropical Picidae: A review. Manuscript.
45. Moojen, J.; Carvalho, J.C.M.; and Lopes, H.S. (1941). Observações sobre o conteúdo gástrico das aves brasileiras [Stomach contents of Brazilian birds]. Memórias do Instituto Oswaldo Cruz 36(3):405–444.
46. Morton, E. 1973. On the evolutionary advantages and disadvantages of fruit eating in tropical birds. American Naturalist 107:8–22.
47. Neal, J.C.; James, D.A.; Montague, W.G.; and Johnson, J.E. 1993. Effects of weather and helpers on survival of nestling red-cockaded Woodpeckers. Wilson Bulletin 105(4):666–673.
48. Pernalete, J.M. 1989. Breeding the black-necked aracari, *Pteroglossus aracari,* at Barquisimeto Zoo. International Zoo Yearbook 28:244–246.
49. Prum, R.O. 1988. Phylogenetic interrelationships of the barbets (Aves: Capitonidae) and toucans (Aves: Ramphastidae) based on morphology with comparisons to DNA-DNA hybridization. Zoological Journal of the Linnean Society 92(4):313–343.
50. Remsen, J.V., Jr.; Hyde, M.A.; and Chapman, A. 1993. The diets of neotropical trogons, motmots, barbets and toucans. Condor 95:178–192.
51. Restrepo, C.; and Mondragón, M.L. 1998. Cooperative breeding in the frugivorous toucan barbet (*Semnornis ramphastinus*). Auk 115(1):4–15.
52. Riley, C.M. 1986. Foraging Behavior and Sexual Dimorphism in Emerald Toucanets (*Aulacorhynchus prasinus*) in Costa Rica. Masters thesis, University of Arkansas.
53. Riley, C.M. 1986. Observations on the breeding biology of emerald toucanets in Costa Rica. Wilson Bulletin 98(4):585–588.
54. Riley, C.M.; and Smith, K.G. 1986. Flower eating by emerald toucanets in Costa Rica. Condor 88:396–397.
55. Riley, C.M.; and Smith, K.G. 1992. Sexual dimorphism and foraging behavior of emerald toucanets, *Aulacorhynchus prasinus,* in Costa Rica. Ornis Scandinavica 23(4):459–466.
56. Rundel, R. 1976. Model breeding environments for toucans, Ramphastidae, at the Los Angeles Zoo. International Zoo Yearbook 16:106–108.
57. Santos, E. 1952. Da Ema ao Beija-flor, 2nd Ed. Zoologia Brasílica 4. Rio de Janeiro, F. Briguiet.
58. Schubart, O.; Aguirre, A.C.; and Sick, H. 1965. Contribuição para o conhecimento da alimentação das aves brasileiras [Contribution to the knowledge of the diet of Brazilian birds]. Arquivos de Zoologia de São Paulo 12:95–249.
59. Schürer, U. 1985. Die Zucht des Fischertukans (*Ramphastos sulfuratus*) im Zoologischen Garten Wuppertal. Zeitschrift des Kölner Zoo 28(2):87–93.
60. Schürer, U. 1987. Die Zucht des Riesentukans (*Ramphastos toco*) im Zoologischen Garten Wuppertal. Zeitschrift des Kölner Zoo 30(3):97–99.
61. Seibels, R.E. 1979. Breeding the toco toucan, *Ramphastos toco,* at Columbia Zoo. International Zoo Yearbook 19:147–150.
62. Short, L.L. 1980. Woodpeckers of the World. Monograph Series no. 4. Greenville, Delaware, Delaware Museum of Natural History.
63. Short, L.L. 1985. Neotropical-Afrotropical barbet and woodpecker radiations: A comparison. Ornithological Monographs 36:559–574.
64. Sick, H. 1985. Ornitologia Brasileira: Uma Introdução [Brazilian ornithology: An introduction], Vol. 1. Brasília, Editora da Universidade de Brasília.
65. Skutch, A.F. 1944. Life history of the Blue-throated toucanet. Wilson Bulletin 56(3):133–155.
66. Skutch, A.F. 1950. An adventure with toucans. Nature Magazine 43:411–413.
67. Skutch, A.F. 1958. Roosting and nesting of araçari toucans. Condor 60(4):201–219.
68. Skutch, A.F. 1961. The nest as a dormitory. Ibis 103:50–70.
69. Skutch, A.F. 1967. Life Histories of Central American Highland Birds. Cambridge, Nuttall Ornithological Club.
70. Skutch, A.F. 1969. Life Histories of Central American Birds. Pacific Coast Avifauna no. 35.
71. Skutch, A.F. 1971. Life history of the keel-billed toucan. Auk 88:381–396.
72. Skutch, A.F. 1972. Studies of Tropical American Birds. Cambridge, Nuttall Ornithological Club.
73. Skutch, A.F. 1983. Birds of Tropical America. Austin, Texas, University of Texas Press.
74. Skutch, A.F. 1987. Helpers at Birds' Nests; A Worldwide Survey of Cooperative Breeding and Related Behavior. Ames, Iowa, Iowa State University.
75. Smithe, F.B. 1966. The Birds of Tikal. New York, Natural History Press.
76. Stacey, P.B. 1981. Foraging behavior of the acorn woodpecker in Belize, Central America. Condor 83:336–339.
77. Stevens, R.P. 1870. The toucan's beak. American Naturalist 4:622–623.
78. Stotz, D.F.; Fitzpatrick, J.W.; Parker, T.A., III; and Moskovits, D.K. 1996. Neotropical Birds: Ecology and Conservation. Chicago, University of Chicago Press.
79. Strahl, S.D.; and Grajal, A. 1991. Conservation of large avian frugivores and the management of neotropical protected areas. Oryx 25(1):50–55.
80. Straube, F.C.; Bornschein, M.R.; Reinert, B.L.; and Pichorim, M. 1993. Estudo ornitológico dos adornos plumários dos índios Hêta do noroeste do Paraná [Use of feathers by Indians of Parana state, Brazil]. Abstracts of the 3° Congresso Brasileiro de Ornitologia. Pelotas, Universidade Católica de Pelotois. SBO. p. 28.

81. Swierczewski, E.V.; and Raikow, R.J. 1981. Hind limb morphology, phylogeny, and classification of the Piciformes. Auk 98:466–480.
82. Todd, F.S.; Gale, N.B.; and Thompson, D. 1973. Breeding crimson-rumped toucanets, Aulacorhynchus haematopygius sexnotatus, at Los Angeles Zoo. International Zoo Yearbook 13:117–120.
83. van Tyne, J. 1929. The Life History of the Toucan, *Ramphastos brevicarinatus*. Ann Arbor, Michigan, University of Michigan Press.
84. von Ihering, R. 1940. Dicionário dos Animais do Brasil [Dictionary of Brazilian animals]. São Paulo, Secretaria de Agricultura Indústria e Commércio.
85. von Ihering, R. 1968. Dicionário dos Animais do Brasil [Dictionary of Brazilian animals]. Brasília, Universidade de Brasília.
86. Wagner, H.O. 1944. Notes on the life history of the emerald toucanet. Wilson Bulletin 56(2):65–76.
87. Wetmore, A. 1968. The Birds of the Republic of Panama, Pt. 2. Columbidae (pigeons) to Picidae (woodpeckers). Washington, D.C., Smithsonian Institution Press.
88. Wilkinson, R.; and McLeod, W. 1991. Breeding channel-billed toucans at Chester Zoo. Avicultural Magazine 97:179–184.
89. Williams, M. 1984. Toco toucans at the Cotswold Wildlife Park. Ratel 11(1):10.
90. Willis, E.O. 1979. The composition of avian communities in remanescent woodlots in southern Brazil. Papéis Avulsos do Departamento de Zoologia 33(1):1–25.
91. Willis, E.O. 1983. Toucans (Ramphastidae) and hornbills (Bucerotidae) as ant followers. Le Gerfaut 73:239–242.
92. Willis, E.O.; and Oniki, Y. 1978. Birds and army ants. Annual Review of Ecology and Systematics 9:243–263.
93. Yamashita, C.; and Lo, V.K. 1995. Ninhos cooperativos em *Melanerpes flavifrons* e *M. cactorum* (Piciformes: Picidae) [Cooperative nests in *Melanerpes flavifrons* and *M. cactorum*]. Ararajuba 3:56–57.

CAPTIVE MANAGEMENT: FAMILY RAMPHASTIDAE (TOUCANS)

Jerry Jennings

Toucans are among the more spectacular of the Neotropical avifauna, noted for their disproportionately large beaks, flamboyant colors, and extroverted behavior. First noticed by Europeans at the time of the conquest, toucans have been perennial favorites of zoo visitors, ecotourists, biologists, and people of all ages and backgrounds all over the world. They are represented in art, both ancient and modern, and have even found their way onto breakfast cereal boxes. Although toucans have been known to Western civilization for nearly 500 years, they have received scant attention from biologists, and subsequently there is a scarcity of published information on their natural history. Furthermore, they have been successfully managed in captivity only during the past 50 years and have been successfully propagated only since the mid-1960s. Of the existing 41 species (and numerous subspecies), fewer than half have reproduced in captivity, and many of the those only to the F_1 generation.

The first successful captive breeding of a toucan species occurred at the Los Angeles Zoo in 1966, when the crimson-rumped toucanet (*Aulachorynchus haematopygus*) fledged two young. Several other first breedings occurred at the Los Angeles Zoo, including the pale-mandible aracari (*Pteroglossus erythropygius*) and the plate-billed mountain toucan (*Andigena laminostris*). In the mid-1970s and early 1980s, a number of other first breedings occurred in various locations around the United States, and today approximately 17 species have bred on numerous occasions around the United States and in Europe. Four criteria play important roles in successful reproduction: mate compatibility, housing, diet, and general health.

SELECTING A MATE Toucans in their natural state have a wide choice for mate selection, which is denied them in captivity. Consequently, it is unlikely that a particular bird will find a suitable mate unless the aviculturist has a variety of birds with which to work. Random pairing of toucans occasionally results in aggression between birds, but more often results in indifference, with the male and female birds ignoring each other. Time will often overcome indifference, and eventually the birds may decide to breed. When this occurs, and it may require several months to several years, the pair will engage in certain behaviors that demonstrate their mutual interest. Sitting close together, touching beaks, and offering food items to each other are the most obvious signs. These behaviors are usually followed by joint nest inspections, excavation of the nest cavity, copulation, and egg laying. Occasionally, pairs will not properly incubate their eggs or rear newly hatched young. Rather than a sign of incompatibility, such failures are probably a result of outside disturbance, insufficient choice of dietary items, or one or both birds may be imprinted on humans. If a particular pair of toucans has failed to reproduce after several years, they should be separated and re-paired with different mates. These two “new” pairs should not be housed near each other.

HOUSING Proper housing of toucans is technically easy. Cage enclosures need not be fancy, but the more the following conditions are met, the more likely reproduction will occur. First, the cage should be large.

Although cages as small as 3 × 4 meters have produced results, larger cages (4 × 8 × 3 m high) produce higher numbers of young.

Second, flight cages should be screened visually from any other toucan species. Toucans establish territories around their nests, which they aggressively defend. If they can see neighboring toucans, they are less likely to reproduce, and if they share a common wall with other toucans through which they can see, they will spend too much time interacting with the neighbors to hatch and raise young.

Third, toucan pairs should be housed alone for best results. Although there are a number of reports of toucans reproducing in mixed-species enclosures, these successes have usually been in exceptionally large, planted, walk-through flight cages in zoological parks and have come at the expense of other birds that became toucan prey.

Fourth, flight cages must be secure from outside disturbances, including rodents and nocturnal predators. Predators such as raccoons or coatis may frighten toucans at night or catch them on the wire and pull their toes and legs through the wire. Rats and mice scurrying around in the dark disturb toucans and may contaminate their feed and water receptacles with feces and urine. Rats also may scare toucans off the nest at night and eat the eggs or small nestlings.

Fifth, toucan flight cages should be lightly planted to provide security and perching opportunities. Plants should not be so close to the nest that they provide an avenue of approach for predators. Wild toucan nests are usually located in free-standing trees, some distance from any other tree, and free of climbing vines. Toucans prefer a clear view of the surrounding area so they may see danger approaching in time to escape.

DIET Toucans enjoy a wide variety of fruits in the wild and are known for their contributions to forest ecology as seed dispersers. To reproduce this wide selection of items in captivity is difficult, if not impossible, because most of the natural dietary items are not available as cultivated crops. However, there is sufficient variety in cultivated fruits to satisfy toucans, and variety is a stimulus to breeding activity. Toucans, because of the relatively weak muscles of the beak, have specialized in taking advantage of plant species that supply fruits that can be easily picked and swallowed. They are able to "tear apart" soft fruit, but they prefer species that are already bite size, such as the many varieties of ficus. It is important, therefore, to chop fruits that are offered to captive toucans. The ideal size appears to be 1–2 cm in diameter.

Most fruits are recommended for toucans. They especially enjoy colorful fruits, such as papaya, grapes, cherries, and blueberries. They will also readily consume a variety of melons and bananas and any other berry variety. High-acid fruits, such as oranges, tangerines, grapefruit, pineapple, and tomatoes, should be avoided, because these encourage the uptake of excessive iron.

Toucans also require a source of high protein not available in fruit. Proprietary pellets low in iron are recommended, because they offer all that is needed in proteins, vitamins, and minerals. Supplemental feeding of vitamins and minerals when a quality pellet is provided may lead to gout and other metabolic disorders.

The key consideration in selection of dietary items is concern for the iron content. Toucans are vulnerable to a metabolic disease known as hemochromatosis, or the super absorption of dietary iron that is stored in the liver and pancreas. As iron accumulates over the life of the bird, these organs break down, leading to premature death. High-acid fruits contribute to the superabsorption of iron as they weaken the barrier between the intestinal wall and the blood supply, which encourages the absorption of iron. This has been debated in animal medicine, but it is well recognized in human medicine. Prevention of hemochromatosis requires a low-iron diet.

It should be noted that toucans require *both* fruit and pellets. Although it may seem simpler to offer only pellets, toucans require fruit to properly digest their food. Fruit is also the main source of hydration, because toucans drink little water. Water receptacles in the flight cage are used primarily for bathing. Fruit items must be prepared and served fresh on a daily basis. If the birds are housed in a hot climate, it is wise to change the fruit twice daily to prevent spoilage and the buildup of fungi.

Finally, when toucans do reproduce, the adults will be stimulated to care for their young if live food is offered, and frequently will not feed their young if it is unavailable. In the wild, flying insects such as crickets and grasshoppers, are the primary sources of live food. This need is easily satisfied in a captive situation by supplying crickets, which are cultivated and commercially available in the United States. Other insects, such as mealworms, are less interesting to toucans. If toucans are not offered insects during the breeding cycle, they will hunt their own and may take undesirable species, such as pill bugs, earwigs, and other ground-dwelling arthropods that serve as intermediate hosts for several intestinal parasites that affect toucans.

GENERAL HEALTH A reproductive toucan must be in good health. A particular bird may superficially appear to be healthy, but it is not always the case. Birds selected for a breeding program not only must appear to be healthy and have good body weight, but must be free from infectious disease and have a history of proper nutrition. To ensure that this condition prevails, birds should be carefully examined before placement in an enclosure. Such examinations should include a fecal exam for intestinal parasites and cultures for bacterial problems. Blood panels should be performed if an apparent problem exists.

MEDICINE: FAMILY RAMPHASTIDAE (TOUCANS)

Zalmir S. Cubas

RESTRAINT, ANESTHESIA, AND SURGERY

Restraint and Anesthesia

Ramphastids may inflict painful injuries to handlers. Birds may be restrained by holding the bill, taking care not to obstruct the nostrils located at the base of the beak. Isoflurane is the most reliable volatile anesthetic agent for restraint and anesthesia of birds. A modified face mask that fits the long beak may be constructed from a cylindrical plastic bottle (saline solution plastic bottle), which is taped to a dog or cat mask. A rubber glove is stretched over the other side of the mask and taped into place. The beak and nostril are inserted into the mask through a slit in the glove. To reduce mechanical dead space the mask should be the exact size of the beak.

Induction and maintenance gas flow rates for isoflurane are similar to those recommended for psittacines; induction should not exceed 3% and maintenance should be 1.5–2%. Despite the advantages of isoflurane, halothane has been used with a good margin of safety in birds when administered through precision vaporizers. Halothane is less expensive, but has the disadvantage of potentially causing liver disease in exposed hospital personnel (rooms without adequate ventilation) and respiratory and cardiac depression in avian patients.

Endotracheal Cole tubes or tubes constructed from urinary catheters are recommended for surgeries on the head or beak repair procedures. Intubation in ramphastids is a simple procedure; the tongue is small and filamentous and the glottis may easily be seen by pulling out the tongue. The endotracheal tube should adapt perfectly to the trachea without offering resistance, and precaution should be taken not to traumatize the glottis and trachea. The usual anesthesia monitoring procedures, such as respiratory, cardiovascular, body temperature, and glucose monitoring should be followed during surgeries.

The author has used injectable anesthetics in combination with tranquilizers in painful short procedures (duration 15–20 min.). An intravenous (IV) combination of ketamine (20 mg/kg) and xylazine (1–2 mg/kg) administered slowly makes possible the surgical sexing of birds. Xylazine increases muscle relaxation, analgesia, and recovery times. Intravenous injections should be given at a slow rate to prevent arrhythmia and cardiac arrest. Another possible combination of drugs is ketamine (20 mg/kg) and diazepam (1 mg/kg) given intravenously.

Surgery

Beak Repair

Toucans are territorial birds, preferably maintained in pairs. Mate aggression, fights between males, and interspecies aggression are common. During the breeding season, males caged next to each other without the presence of a visual barrier may engage in beak jousting. In an attempt to reach an opponent through the wire, self-inflicted beak fractures may occur, usually in the upper bill. Wild-caught birds and young ramphastids that have recently been introduced into aviaries may fracture the bill by repeated strokes against wire netting. To prevent injuries, a period of adaptation in an aviary sheltered with plastic or nylon cloth on the inner wall is recommended. Mate-related injuries may occur.

Toucans have long but light bills composed of spongy bone protected by a thin wall of keratin. The emergency procedure for fracture is control of hemorrhage. A gauze pad, moistened with povidone iodine, should be held over the wound for a few minutes. Debris should be removed and the wound cleaned and dried thoroughly. Care should be taken not to insert liquid or debris into the spongy bone, which may carry contaminants to the deeper parts of the beak and sinuses. Water-soluble antibiotic ointments may be applied to the wound and gauze dressings taped over the defect. Dressings should be replaced every 24 hours until hemorrhage and infection are controlled. Parenteral antibiotics are also recommended.

Healing of keratin is slow and requires acrylic repair in order to prevent contamination and additional trauma to the site. Abraded surfaces should be cleaned, loose particles of keratin, debris, and necrotic tissues removed, and the edges of the defect leveled. The keratin at the margin of the wound may be sanded for better adhesion of the bonding material. The beak defect (exposed spongy bone) should be covered with calcium hydroxide and sealed with a dental restorative resin composed of bis-glycidil-methacrylate and tritilene-glycol-dimethacrylate (Composto Dental Concise; 3M do Brasil Ltda., Sumaré-SP, Brazil) or similar material that is molded and applied at a level slightly above the surface of the ramphoteca. The acrylic should fill the defect and adhere to the marginal surface of the wound. If necessary, the resin may be ground smooth with a dental or Dremel drill. The repair should be left on for a minimum period of 6 weeks, the final length of time depending on the extent of the lesion. The acrylic repair may be permanently removed only after a substantial layer of keratin has formed on the wound. For aesthetic reasons, prosthetic materials may be used to cover defects of the beak, but it should be understood that these corrective appliances are temporary and must be replaced periodically. Detailed descriptions of beak repair techniques using acrylics may be found in the literature.[2,6]

Injuries

Excessive clipping of the wing alters the bird's balance and may cause repeated falls from a perch, resulting in facial abrasion and keel laceration. Confining injured birds in a small aviary or cage with low perches during the treatment period is necessary to avoid additional trauma and allow healing. Semiocclusive bandages (Tegaderm;, 3M Company, St. Paul, Minnesota, USA) have proven to be useful in facial abrasions with loss of extensive areas of skin. Weak birds sitting on the bottom of the aviary in contact with abrasive cement surface may develop wounds and dermatitis in the metatarsus and hock joint, which may evolve to arthritis and osteomyelitis that resembles bumblefoot (Figure 19.1). The treatment protocols for pododermatitis in raptors are applicable to the metatarsal lesions of toucans. Long-term application of bandages and ointments and parenteral antibiotic therapy are required. The floor of the cage or aviary should be covered with nonabrasive material, such as a rubber mat or carpet.

Bone Fractures

Bone fractures in ramphastids are managed with same surgical techniques applicable to psittacines and other bird groups. Toucans are inquisitive, and their large beaks may trap them in holes and forklike branches, resulting in suffocation. Deep ponds inside aviaries represent a risk of drowning if toucans are unfit to fly.

DIAGNOSIS

A minimum database should be established to facilitate diagnosis, based on physical examinations, weight monitoring, fecal gram stains, fecal examinations for parasite detection, crop washes, plasma protein, hematocrit (packed cell volume, PCV), blood glucose, white blood cell count (WBC), and clinical biochemistry. Additional procedures may be necessary to make a diagnosis, including radiography, endoscopic examinations, microbiologic cultures, and biopsies.

Blood Collection

Jugular venipuncture is a common procedure for obtaining blood samples because the vessel is large and easily seen through the thin featherless skin over the neck. The right jugular vein is usually larger than the left, making it the preferred site for drawing larger volumes of blood.

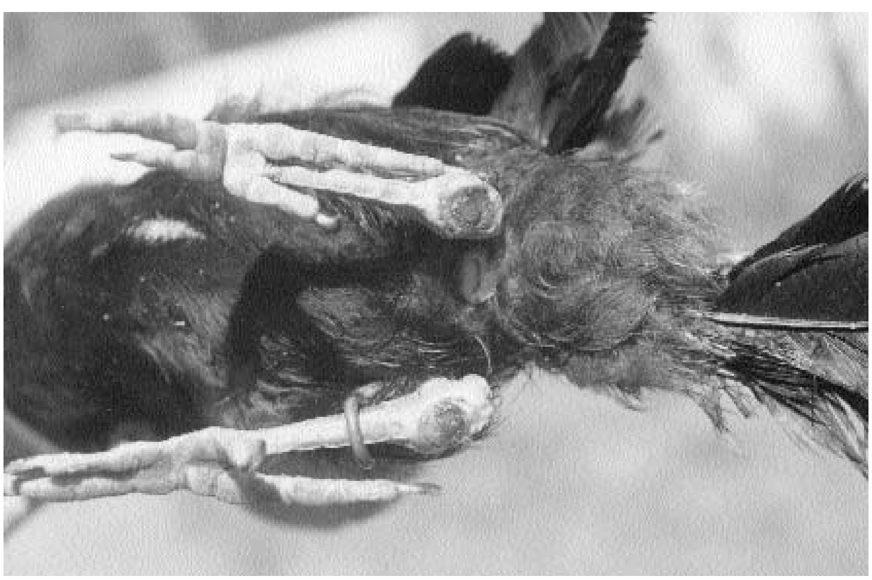


FIGURE 19.1. Dermatitis in the hock joint of a toco toucan.

To collect blood, the bird should be restrained in an upright position, with the neck held in extension to facilitate location of the jugular vein, which sits in the jugular furrow next to the trachea. Wetting feathers with alcohol will aid in localization of the vessel, which may be occluded by finger pressure at the thoracic inlet to favor blood aspiration. Small-gauge needles (29 G ½) should be used to minimize hematoma formation.

The ulnar vein runs superficially along the elbow and is easily identified, because the skin over the vessel is delicate and translucent. The ulnar vein is the preferable site for fluid infusion. Drawbacks are that birds must be restrained in an unnatural position over a hard surface, and wing flapping may make the procedure impracticable, resulting in the formation of large hematomas. Pressure applied with the finger on the puncture site for a few minutes is usually sufficient to stop bleeding. A butterfly catheter aids in stabilization of the vessel and is also useful for fluid administration. The medial metatarsal vein courses superficially along the medial side of the tarsometatarsus. It is not as visible as the ulnar vein, but hematomas are less likely to occur because it is covered by thicker skin. Small volumes of blood required for hematocrit and serum protein determination mays be obtained by toenail clipping.

The reader may find in-depth reviews of avian hematology and clinical chemistry in other publications. Reference values of hematology and biochemistry for ramphastids are found in Tables 19.1 and 19.2.

TABLE 19.1. Means and ranges for hematological values in toucans

Number of birds	86	—
WBC (cells/µL)	13,500	8000–18,000
PCV (%)	49.8	42–60
Heterophil (%)	51.9	41–62
Lymphocyte (%)	50.5	35–70
Monocyte (%)	0	0–2
Eosinophil (%)	0.67	0–3
Basophil (%)	0	0–1

Source: See reference 12.
PCV, packed cell volume; WBC, white blood cell count.

TABLE 19.2. Means and ranges for biochemical values in toucans

Number of birds	86	—
AST (SGOT) (U/L)	243.3	141–340
Uric Acid (U/L)	7.93	2.4–14
LDH (U/L)	257.6	180–319
Glucose (mg/dL)	297.9	222–363
Calcium (mg/dL)	10.2	8.8–11.8
Cholesterol (mg/dL)	175.1	104–254
Albumin (g/dL)	2.1	1.4–2.4
Alkaline phosphatase (U/L)	43.3	14–88
Protein (g/dL)	3.5	2.8–4.4
Globulin (g/dL)	1.79	1.4–2.2
Albumin/globulin ratio	1.42	0.92–2.67
Bile acids (µmol/L)	54.4	16–86

Source: See reference 12.
AST, aspartate aminotransferase; LDH, lactate dehydrogenase.

Fecal Examination (Gram Stain)

Fecal examination is a useful method to assess the clinical condition of the avian patient and may indicate the presence of nematode eggs, coccidia, other pathogenic protozoans, potentially pathogenic enteric bacteria, and yeast. Healthy toucans may show various patterns of intestinal microflora, and rather than establish normal references for fecal Gram stain, the author prefers to assess a bird's overall condition, diet, and management in combination with diagnostic test results. Compared to psittacines, clinically normal toucans have lower bacterial counts and a higher percentage of gram-negative rod-shaped bacteria. However, excessive numbers of gram-negative bacteria and yeast are suggestive of poor nutrition and intestinal flora imbalance. Similar to psittacine birds, high percentages of gram-positive cocci are expected in healthy toucans. To establish normal digestive tract flora, recommendations are to feed the birds a balanced diet and improve husbandry techniques. Antibiotic therapy and acidifying agents of the intestinal tract (e.g., *Lactobacillus,* lactulose apple cider vinegar) are recommended by some practitioners for birds with a high percentage of gram-negative organisms showing clinical signs of enteritis.

DISEASES

INFECTIOUS DISEASES

Bacterial Diseases

In a study of 53 asymptomatic toucans of five different species, the cloacal microflora detected was *Escherichia coli, Staphylococcus* spp., and *Streptococcus* serotype D.[8] *Klebsiella pneumoniae* has been recovered occasionally from the cloaca of healthy toucans. Low immunity may favor bacteria's penetration through the mucosal barrier, causing bacteremia.

Avian pseudotuberculosis (*Yersinia pseudotuberculosis*), a gram-negative bacteria, has been documented as the cause of peracute death in toucans kept in North America and Europe. Rats and mice are probably the reservoir, carrying *Y. pseudotuberculosis* to the aviaries. Necropsy findings included hemorrhagic to fibrinous pneumonia, hepatomegaly, splenomegaly, and cheesy masses or granulomas in many organs. Preventive measures include the implementation of a permanent rodent control program, isolation of suspected birds, disinfecting of cages and food dishes, and hygiene. Placing food dishes in a tray fixed on a solid wall at 1.5 m from the floor of the enclosure makes food inaccessible to rodents in the aviary and reduces spillage.

*Bacteroide*s spp. (nonfragilis) infection was diagnosed in a toco toucan. Lesions seen at necropsy were severe necrotising enteritis, nodules in the intestinal serosa that were replete with foul-smelling caseous material draining into the intestinal lumen (Figure 19.2). Gram-positive bacilli were the predominant bacteria present in this material. Hepatomegaly and multiple granulomatous-like lesions were seen in the liver (Z.S. Cubas,1999, unpublished data). The disease followed a chronic wasting course. Anaerobic isolation of the microorganism was achieved using an enriched media for Clostridia. *Clostridium colinum,* an anaerobic microorganism, has been reported as the causative agent of deaths in six toucans that died without premonitory signs over a period of 4 months. Necropsy findings consisted of necrotizing hepatitis and ulcerative enteritis. *C. colinum* was isolated from the liver of four of the birds. A concomitant finding was elevated iron levels, which may have contributed to the infection.[20] It is suggested that a predisposing condition allowed these bacteria to proliferate in the gastrointestinal tract. High-carbohydrate levels in the food is regarded as being a major factor. Contaminated soil, food, and water are possible sources of infection.

Avian tuberculosis was diagnosed in a black-necked aracari (*Pteroglossus aracari*).[23]

CHLAMYDIOSIS *Chlamydia* sp. was demonstrated by enzyme-linked immunosorbent assay (ELISA) in a pair of toucans housed in a zoo.[14] More research is needed to determine the clinical importance of chlamydiosis in the Ramphastidae family.

Mycotic Diseases

Candida albicans is commonly identified in the feces of healthy and ill ramphastids. White diphtheritic plaque may be seen in the oral cavity of chicks with retarded growth. Yeast organisms are easily identified by cytologic examination of oral smears and fecal samples stained with Gram stain or quick stain. The author successfully treated a saffron toucanet (*Baillonius bailloni*) nestling with nystatin at a dosage of 500,000 IU/kg 3 times a day for 5 days. Ketoconazole at a dosage of a 200 mg tablet crushed in 1 L of water and given as the sole source of drinking water for 10 days was an effective treatment of a red-billed toucan (*R. tucanus*).[13] *Penicillium griseofulvum* was reported in a group of toucanets.[1] Aspergillosis has been occasionally seen by the author in stunted chicks taken from the nest for hand-rearing. It is suspected that feces accumulating inside the nest creates an ideal environment for fungus proliferation and inhalation of spores by immunosuppressed chicks. Reproductive management includes periodic weighing of chicks and removal of feces from the nest.

Viral Diseases

A herpesvirus serologically unrelated to Pacheco's disease virus was recovered from a toucan. The toucan had been in contact with macaws that died of herpesvirus-induced

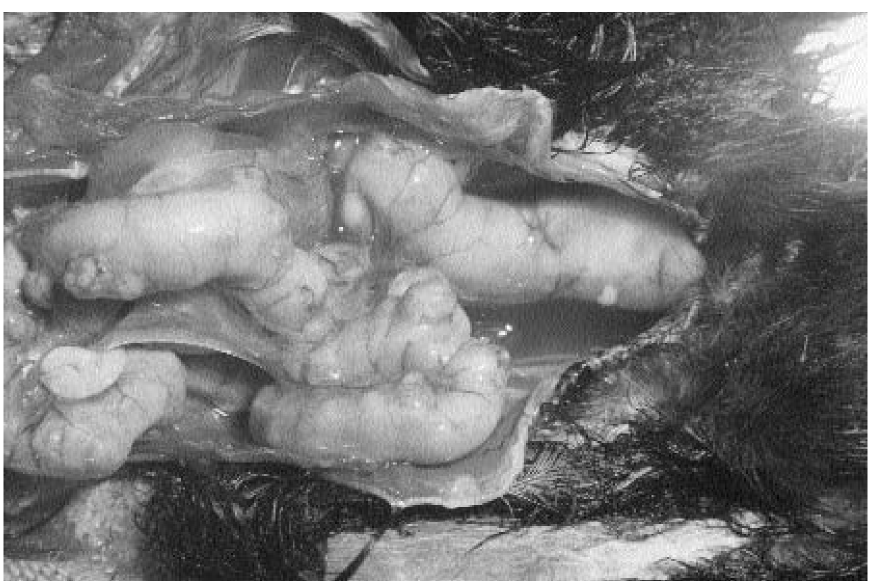


FIGURE 19.2. Nodules in the intestinal serosa with caseous material. *Bacteroides* sp. infection.

hepatitis, a diagnosis based on microscopic changes.[5] Typical lesions of herpesvirus are hepatomegaly, splenomegaly, and intranuclear inclusion bodies in the liver and spleen. Toucans may harbor Newcastle disease virus.

Parasites

CAPILLARIA *Capillaria* spp. are tiny elongated nematodes that may infect the esophagus, crop, and intestine. Ramphastids are particularly susceptible to capillaria parasitism, which may be the most common cause of mortality in captive toucans. The disease accounts in part for the low rate of maintenance and reproduction of ramphastids in captivity. Mortality occurs throughout the year; however, incidence seems to be higher in the hottest months. Parasite eggs remain infective in the environment for several months. The tenuous defense mechanism that balances the parasite-host relationship may be disrupted if the environment or management conditions deteriorate or if the host's immune system becomes impaired. Under these circumstances, mortality rates increase.

All ramphastids seem to be equally affected, nevertheless the author has observed higher mortality in some species than others, the red-breasted toucan (*Ramphastos dicolorus*) and the channel-billed toucan (*Ramphastos vitellinus*), in particular. Host susceptibility and specificity deserve more investigation.

An outbreak of *Capillaria obsignata* in toucans, with acute mortality estimated at 85%, occurred in a Brazilian zoo during the summer of 1997. Necropsy revealed hemorrhagic enteritis and a large number of nematodes in the intestinal lumen. Treatment with ivermectin, levamisole, and mebendazole at standard dosages for birds was not effective (M.S. Gomes, Personal communication, 1998). The author reported *Capillaria columbae* infection in the toco toucan (*Ramphastos toco*).[9] Pathogenicity may vary and may be associated with immune suppression. Concurrent pathologies, such as hepatitis and hemochromatosis, may undermine immune resistance, exacerbating a chronic subclinical parasitic infection. The life cycle of *Capillaria* may be either direct or indirect, depending on the species involved.

Clinical signs are nonspecific and include emaciation, apathy, lethargy, anorexia, brown to bloody diarrhea, anemia, and dehydration. Vomiting has not been observed. Adult worms burrow into the intestinal wall, causing loss of fluids and blood. Thus low PCV and plasma protein may occur in weak birds. Septicemia is a possible complication, resulting from destruction of the integrity of the intestinal mucosa. In advanced cases, ulceration and hemorrhage are seen in the intestine (Figure 19.3).

Eradication of *Capillaria* from a collection is difficult, even employing preventive medication and control of the parasite in the environment. It is suspected that free-living birds may introduce or disseminate nematodes to captive collections of toucans. Preventive and control measures that may significantly reduce infection include elevating feeders and water containers above the floor (not attached under perches); removing perches, plants, and the superficial stratum of soil from highly contaminated aviaries; destroying *Capillaria* eggs on the floor of the enclosure (flame torches are useful); removing *Capillaria*-positive birds from their original aviaries, deworming them, and keeping them in isolated cages for as long as necessary; administering anthelmintics on

FIGURE 19.3. *Capillaria* infection. Ulcers in the intestinal mucosa and tiny worms in the intestinal lumen or mucosa can be found.

a regular basis (every 2–6 months, according to infestation severity in the collection); and performing periodic fecal examinations to assess treatment effectiveness (monthly in critical premises and periods). At least three sequential negative fecal examinations are recommended before considering a bird to be free of parasites. Some of these measures may prove to be difficult to implement in large aviaries and mixed-species exhibits.

Treatment efficacy with anthelmintic depends on *Capillaria* species sensitivity and the anthelmintic regimen instituted. Some *Capillaria* spp. have proven to be resistant to most of the available drugs, including albendazole, ivermectin, pyrantel pamoate combined with oxantel, levamisole IM, and moxidectin IM at the recommended dosages, either when given as the only drug or in combination with another. A listing of common anthelmintic dosages used by the author is found Table 19.3. Repeated administration may be necessary until fecal exams are negative. Adverse effects of the drugs should be considered when calculating dosages. For instance, benzimidazoles are known to cause bone marrow suppression and anemia. The author uses various anthelmintics in combination to obtain a synergistic effect when dealing with resistant strains of *Capillaria*. High dosages of pyrantel pamoate have been used without adverse effects. Rotation of anthelmintics to prevent development of resistance is recommended.

PROTOZOA *Eimeria* oocysts are occasionally seen in asymptomatic birds, but clinical disease may occur, especially under unsanitary conditions and improper management. Species reported are *Eimeria vitellini* in a red-billed toucan (*Ramphastos tucanus*) in a zoo in the Brazilian Amazon region[16] and *Eimeria forresteri*, reported as causing severe diarrhea in a group of toco toucans in South America.[19] Therapeutic protocols similar to those used for prevention and treatment of poultry coccidiosis are recommended: toltrazuril (7 mg/kg orally for 2 days) or sulfaquinoxaline (125–250 mg/L of drinking water 3 days on, 3 days off, and 3 on) or amprolium (50–100 mg/L of drinking water for 5 to 7 days). *Giardia* spp. is commonly seen in clinically healthy birds. Treatment with usual giardiastats, such as metronidazole, tinidazole, and secnidazole is recommended. *Sarcocystis* sp. is a protozoan that requires both an intermediate and a definitive host. In an outbreak in a zoo collection in Brazil, two toco toucans died from sarcosporidiosis. Death was rapid, although the birds were externally in good condition. A remarkable histopathological finding was lung hemorrhages and schizonts within the endothelium of pulmonary capillaries and in macrophages.[14] Opossums (*Didelphis marsupialis*) proved to be the definitive host that passes oocysts in the feces. Large numbers of oocysts were found in the intestinal submucosa of trapped opossums.

OTHER PARASITES *Plasmodium* species have been reported in toucans, but only high levels of *Plasmodium huffi* are known to cause severe anemia and death. Filarid nematodes are occasionally found in wild toucans. The species *Dessetfilaria guianensis* has been described in a free-flying channel-billed toucan (*R. vitellinus*) in French Guiana,[3] and *Dessetfilaria braziliensis* was found in the outer wall of the aorta and pulmonary trunk in two red-breasted toucans (*R. dicolorus*) and one channel-billed toucan in São Paulo State, Brazil (J. H. Fontenelle, personal communication,1999). The author encountered adult filarids in two red-breasted toucans originating in Iguassu National Park, Paraná State, Brazil. The filarids were found in a capsule in the pulmonary trunk and in the left auricle. Filarid nematodes are considered to be incidental findings.

Noninfectious Diseases

Hemochromatosis

Iron is an essential element in normal physiologic processes, and storage in the liver and other organs is a

TABLE 19.3. Anthelmintics used in ramphastids

Anthelmintic	Dosage/Route	Frequency
Albendazole + ivermectin	15–20 mg/kg PO and 0.4 mg/kg PO	Once. Repeat if necessary
Oxfendazole + ivermectin	15–25 mg/kg PO and 0.4 mg/kg PO	Once. Repeat after 15 days if necessary
Fembendazole	50 mg/kg/day PO	Over 5 days
Mebendazole	25 mg/kg bid PO	Over 5 days
Pyrantel pamoate	70 mg/kg PO	Once. Repeat if necessary
Moxidectin	0.2 mg/kg IM	Once. Repeat if necessary
Ivermectin	0.2–0.4 mg/kg SC, PO	Once. Repeat if necessary

IM, intramuscularly; PO, orally; SC, subcutaneously.

natural mechanism. However, it may become pathologic under certain circumstances. Iron accumulation in organs without deleterious effect to the cells is called *hemosiderosis,* whereas the term *hemochromatosis* describes toxic iron deposition in the tissues and compromising of the organ function.

Iron absorption requires its release from the organic compounds ingested and reduction to the ferrous state. After absorption in the gastrointestinal tract, the element is transported by proteins called transferrins and stored in combination with proteins in the forms of ferritin and hemosiderin. Whereas ferritin is not readily seen on histologic sections, hemosiderin appears as brown granules on hematoxylin and eosin stains or as a blue-green color with Prussian blue stain (Figure 19.4). Hemosiderin overload in the cells may lead to hematoma formation, focal hemorrhages, and severe vascular congestion. In advanced cases, fibrosis and cirrhosis develop, and ultimately the liver fails to function. For more detailed information on the mechanisms of iron overload in various animal classes the reader is referred to current texts.[47]

One of the most important diseases in the Rhamphastidae family, hemochromatosis has been reported in 13 species of toucans and aracaris.[22] No species seems to be more susceptible than any other. A higher prevalence has been reported in toco toucans, but because the toco toucan is the most common species in captivity, the seemingly higher incidence may be a result of its more numerous captive population. It is suspected that hemochromatosis has been accompanied by capillariosis and infectious hepatitis in South American zoo collections. Because a high incidence of *Capillaria* infection is evident at necropsy, concurrent iron storage disease may be being overlooked. Studies in poultry supplemented with excess iron have demonstrated an increase in iron absorption in chicks infected with *Eimeria* spp., probably as a result of the disruption of the integrity of the intestinal mucosa.[18] Iron overload may harm liver function and reduce the action of macrophages and

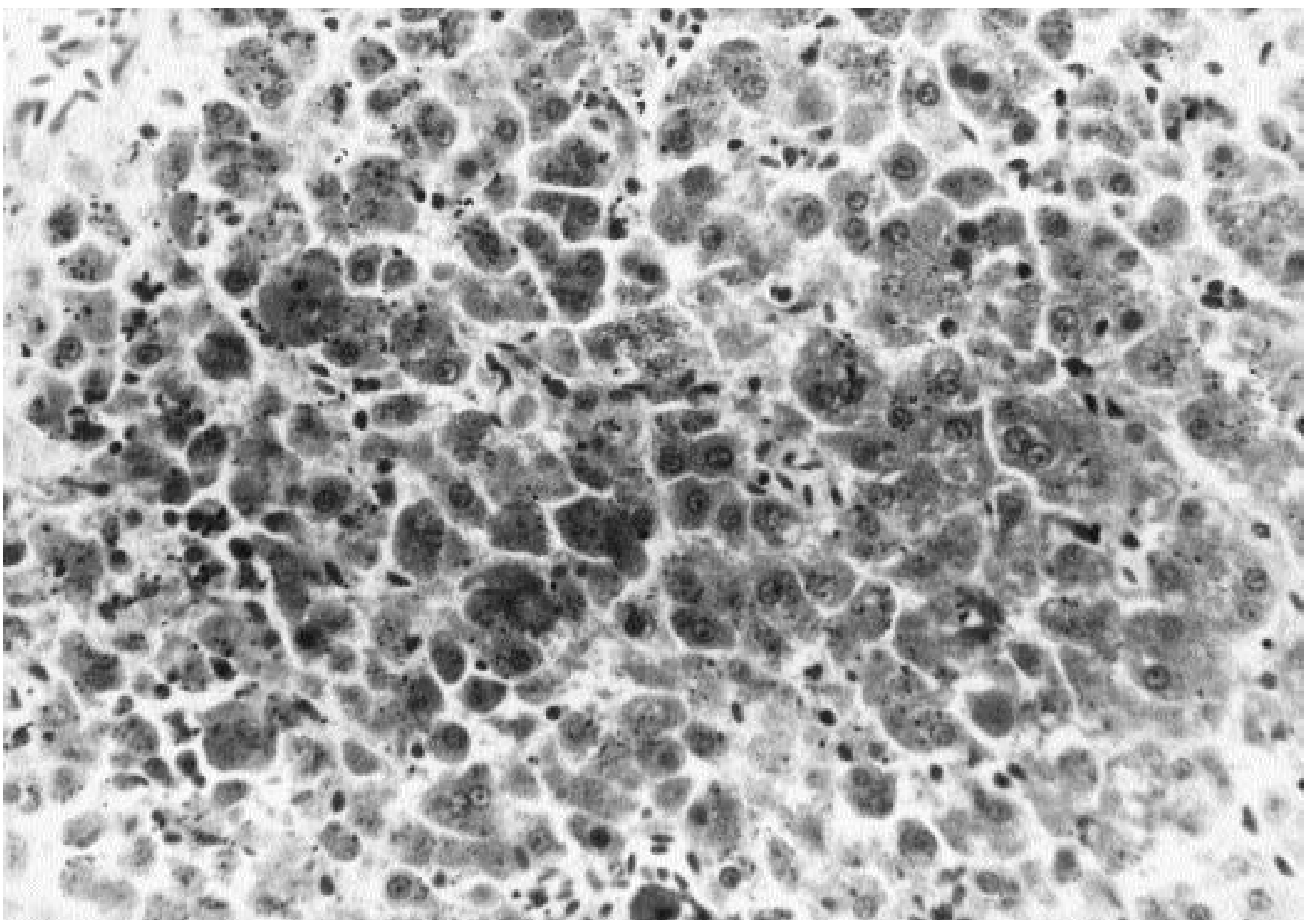


FIGURE 19.4. Liver of a toco toucan with hemochromatosis. Iron deposits appear as dark blue-green granules with Prussian blue stain.

lymphocytes, fostering general bacterial infections and possibly parasitic diseases as well. Determination of the significance of iron deposits in histological preparations must take into consideration the existence and degree of lesions.

Hemochromatosis causes an increase in free radicals. Liver glutathione, an important antioxidant in humans, is depleted in iron-overload patients. Although vitamin C has antioxidant properties, it is not recommended for toucans because it increases the bioavailability and absorption of iron from the digestive tract. For this reason, the current opinion is that fruits high in vitamin C should not be fed to toucans. Alternatively, vitamin E may be used for its antioxidative properties.

The etiology of hemochromatosis in birds is variable, and factors other than nutrition have been incriminated in the disease pathogenesis. Notwithstanding, the most convincing cause in ramphastids is excessive dietary iron intake. It is well documented that toucans receiving diets rich in iron tend to develop deposits in the hepatocytes and Kupffer's cells. Iron deposits may also be seen in the spleen, kidneys, lungs, pancreas, and intestines.[24] Studies in birds-of-paradise indicate that the intestinal absorption rate may reach 90% of the iron intake.[10] A similar efficiency of absorption could explain the high susceptibility of ramphastids to iron toxicity.

Macroscopic lesions in the liver include hepatomegaly, yellowish hepatic discoloration and multiple round foci of different sizes in the parenchyma, fibrin deposition on the surface of the organ and ascites (Figure 19.5). Concurrent hepatitis may occur, with secondary airsacculitis, pneumonia, and septicemia. Acute bacteremia caused by *Klebsiella pneumoniae* with concurrent hepatic iron overload was reported in a green aracari (*Pteroglossus viridis*).[24] Microscopic changes included granular pigments in the hepatocytes and Kupffer's cells and hepatic fibrosis or cirrhosis.

Signs of hemochromatosis may be subclinical, and the patient's general condition may be interpreted as good, because pectoral muscles are well formed and appetite is preserved until death. When clinical signs are present, they include apathy, dyspnea (ascites), and sitting on the ground. Death is usually sudden. Physical examination may reveal a distended abdomen and coelomic effusion. If hepatomegaly is present, radiographic studies may prove to be helpful. Elevated biochemical values of lactic dehydrogenase (LDH), aspartate transaminase (AST or SGOT), and bile acids are parameters indicative of hepatocellular damage or reduced hepatic function. However, serum chemistry, bile acids, and hematology values in birds with iron storage disease may fall between the normal range, which makes measurement of metabolites not fully reliable if interpreted alone.

Hypoproteinemia may also occur as a result of hepatic insufficiency. Specific tests are suggested to assess iron status, including serum iron, total iron-binding capacity (TIBC), serum transferrin levels, and saturation of transferrin. Increased levels and saturation may be indicative of iron overload.[17] Currently, liver biopsy is the most reliable method of diagnosis and monitoring of treatment for iron overload in birds. Unfortunately, this invasive method offers risk to the patient, especially debilitated birds suffering from hepatic damage or coagulopathies. More investigations are needed to develop diagnostic tests that will enable the early detection of this pathology.

FIGURE 19.5. Macroscopic aspect of the liver with hemochromatosis in a toco toucan.

Weekly phlebotomy has been advocated as the best treatment to mobilize iron stored in the liver. Blood removal at the rate of 1–2 mL/week for 4 to 8 weeks was reported by Worell.[25] A proposed treatment protocol is phlebotomy of 1–2 mL/day until either clinical improvement is seen or borderline anemia develops. This initial treatment is followed by weekly phlebotomy until the serum iron level falls to less than 35.82 mcmol/L (200 mg/dL).[17] Hematologic monitoring is essential during treatment to prevent severe anemia. Blood removal on a weekly basis for periods longer than 1 year has been attempted.

Deferoxamine mesilate (DFO) is a specific iron-chelating agent, which has been shown to reverse both the biochemical indicators and clinical signs over a 2-year period of treatment in humans. Deferoxamine chelates iron stored in hepatocytes and other cells as ferritin, hemosiderin, and labile iron. Excretion may be through the feces, bile, and urine. Adverse effects and efficiency of DFO in animals needs to be investigated. Considering the secondary effects of DFO in humans, some disadvantages may be anticipated in animals, such as long-term therapy, potential toxic effects (in humans visual and auditory disturbances, neurotoxicity, allergic reactions, gastrointestinal disorders, renal impairment, and interstitial lung disease), increased susceptibility to bacterial and fungal infection, and poor iron mobilization in patients showing anemia secondary to chronic diseases, with siderosis of reticuloendothelial (RE) cells of the liver, spleen, and bone marrow.[17]

There are few reports of DFO treatment in birds. One successful treatment protocol in a channel-billed toucan (*Ramphastos vittelinus*) was subcutaneous administration of 100 mg/kg DFO daily for 110 days, followed by monthly monitoring by hemogram and liver biopsy, examined histologically by image analyses. After treatment, liver iron levels declined from 450 to 28 μmol/g dry wt. No adverse effect was reported.[7] Adjunct therapy proposed in human medicine includes vitamin E, selenium, vitamin B complex, and folic acid. Herbal remedies have been suggested, as well. Green tea has tannins and is considered to be a natural iron-chelating agent that may help to remove excess iron from the liver. If tea is added to the food, extra calcium is recommended to replace calcium lost from the phytates. Grape seed-skin extract, ginkgo biloba extract (antioxidant properties), and others are empirically used in humans with iron-overload disease.

Currently, all treatment regimens seem to be impractical, especially for large flocks, and are of uncertain effectiveness. Therefore, emphasis should be placed on prevention. Toucans should always be fed low-iron diets. The author has seen mortality in toco toucans fed on dog pellets containing iron level of 80 parts per million (ppm; 80 mg/kg of food). Studies of toucan nutrition are practically nonexistent, but it is assumed that iron levels below 65 ppm are adequate to prevent iron storage disease. Considering that recommended levels for poultry are 30–40 ppm, the author believes that a total dietary iron level within this range is safer for ramphastids. The iron content of the total diet (parts per million in dry matter) should be calculated, not only the iron content in a single food item. Kibbled or pelleted components are rich in iron and may counterbalance the lower levels in fruits and vegetables. Toucans are usually fed boiled eggs, which are, in fact, a good source of animal protein. However, the average iron content of a chicken egg is 2.3 mg, most of it (around 2.0 mg) in the yolk. For this reason, egg white alone is preferred in the diet. Interaction among nutrients is also important to iron absorption and metabolization. Certain organic acids, such as oxalic acid, phytic acid, and tannic acids form insoluble combinations with iron, thus reducing its absorption. These organic acids may be found in certain teas and foods.

Malnutrition

Nutritional disorders result from either deficiency or excess of nutrients in the diet. Young birds reared on a calcium-deficient diet based on fruit are prone to develop rickets. The bills of birds suffering from metabolic bone disease may have a soft and wrinkled appearance. In advanced cases, lameness, painful joints, reluctance to move, and spontaneous fracture of bones may occur. The author has seen bilateral cataracts in a young toco toucan suffering from metabolic bone disease.

Diabetes

Few cases of diabetes mellitus have been reported in toco toucans or keel-billed toucans.[15] Clinical signs include weight loss, decreased appetite, lethargy, ruffled feathers, polyuria, polydipsia, hyperglycemia (normal glucose range is 200–350 mg/dL) and glucosuria. It is suspected that the etiology is related to management and diet, hypothyroidism, and pancreatic islet cell tumors. Changing food to a formulated diet and instituting insulin therapy are the treatments suggested by some authors.

PEDIATRICS

Ramphastid chicks do better if parent raised. However, hand-rearing chicks is becoming a popular practice as new information about toucan pediatrics is obtained and aviculturists become more confident in hand-rearing techniques. For breeding purposes, toucans should be housed in pairs in individual aviaries, preferably out of visual contact with adjacent cages. Nests made of palm tree logs are suitable.

Two to four chicks usually hatch, but some pairs may not be able to raise more than two nestlings. In the author's experience, ramphastid chicks should be weighed and inspected daily or every other day in the first 2 weeks of age. As they grow, the nest log should be checked every 2 to 3 days. Both male and female take care of the chicks, and handling them does not make parents abandon the offspring. Most breeding pairs will accept human presence from the first day of hatching if they are used to people in the aviary. However, care should be taken with nervous birds that are not used to people, for they may become upset and attack the chicks after manipulation. The first weeks of life are the most critical for a chick's survival. Hatchlings should be marked, and those that are not gaining weight or that show signs of disease, such as a lack of the feeding response, dehydration, pale skin, hypothermia, and empty stomach are strong candidates for removal and hand-raising. Chicks taken from the nest after 1 week of age seem to thrive better.

Eggs of pairs that have previously broken or eaten eggs should be removed for artificial incubation. Pairs that have injured their hatchlings on previous occasions will most likely do it again. It is advisable to pull the nestlings of such pairs for hand-feeding. The visual presence of other pairs of toucans nearby is a factor of stress and aggressiveness during the breeding season, which may result in destruction of the offspring by the parents.

It is recommended to increase protein in the diet during the incubation and rearing period. Laboratory-raised insects, pinkie mice, or disease-free adult mice cut into small pieces with long and sharp bones removed, are good sources of protein. The author has seen a male toco toucan kill one chick and tear the body into pieces to feed the remaining siblings. Nutritional requirements for young ramphastids are not fully known and extrapolation from diets for psittacines is suggested.

Parent-raised spot-billed toucanet (*Selenidera maculirostris*), saffron toucanet (*B. bailloni*), and chestnut-eared toucanet (*Pteroglossus castanotis*) chicks have been fed successfully with a diet that consisted of poultry pellets, low-iron dog pellets, boiled egg whites, banana, papaya, cooked carrots, and beet root. Parents have also been seen ingesting free-flying insects that entered the cages and offering them to the chicks, which may add to the captive diet. Hand-rearing formulas for psittacines may be used in association with fruits such as papaya, banana, and applesauce.

Hygiene during food preparation is essential for successful rearing. Distilled or boiled water should be used to mix food items and the use of sterile and strained fruits are a routine procedure in a breeding facility (J. Jannings, personal communication, 1991). The author has used a noncommercial hand-rearing formula made of cereals and other products for human consumption (final nutrient content similar to commercial psittacine hand-rearing formulas) to feed young aracaris and toucanets. Food is administered carefully into the proventriculus using rubber or stainless steel feeding tubes. Because the skin of a young chick is thin, the food, of a yogurtlike consistency, can be easily seen advancing down the esophagus in the right side of the neck. Food administration should cease when food starts to accumulate and flow up into the esophagus. Small pieces of fruit, cooked egg white, and moistened pellets may be given from the first week of age.

The feeding response is elicited by touching the upper beak or the corner of the beak. Food is placed in the back of the mouth and moves by peristaltic movement down the esophagus into the proventriculus. Youngsters that do not show the feeding response are in critical condition and need urgent medical assistance. Chicks in deep sleep will not respond to feeding stimulation and should not be misinterpreted as being ill. Food is better accepted if given at warm temperatures; however, older chicks will promptly take food at room temperature. Ramphastids do not have crops in which to store food as psittacines do, and small amounts of food should be given at short intervals. Ramphastids less than 1 week old may need to be fed every 1–2 hours during the daytime. A good indication of when to give the next meal is to check if the stomach is empty and if the chick displays a food-begging behavior. Well-fed chicks have a distended and round abdomen and hungry chicks will accept food until fully satisfied. The feeding interval may be extended as the chicks grow. Storage of pureed food, even in the refrigerator, is not recommended, because pathogenic bacteria may grow quickly in this nutritive media.

In the first weeks of age, chicks are best maintained in a brooder at 33°C (91.4° F). The temperature is reduced to room temperature as down feathers start to grow and chicks become physiologically able to control the core body temperature. Shivering and lethargic birds may be an indication of a low temperature in the brooder. Restless chicks and reddish skin indicate a high temperature. Thermostats are recommended to prevent overheating in the brooder.

Dehydrated birds should be treated with fluids given subcutaneously and orally (Figure 19.6). Poorly developed chicks should be removed from the nest for treatment and hydration. Candidiasis, gram-negative bacteria, and aspergillosis are common causes of stunted growth.

FIGURE 19.6. Dehydrated toucanet chick showing wrinkled skin.

REFERENCES

1. Aho R.; Westerling, B.; Ajello, L.; Padhye, A.A.; Samson, R.A. 1990. Avian penicilliosis caused by *Penicillium griseofulvum* in a captive toucanet. Journal of Medical and Veterinary Mycology 28(5):349–354).
2. Altman, R.B. 1997. Beak repair, acrylics. In R.B. Altman, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 787–799.
3. Bartelett. C.M. 1987. In new avian filarioids (Nematoda: Splendidofilariinae): *Dessetfilaria guianensis, Aundersonfilaria africanus,* and *Splendidofilaria chandenieri*. Proceedings of the Helminthological Society of Washington 54(1):1–14.
4. Campbell, T.W. 1994. Hematology. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 176–198.
5. Charlton, B.R; Barr B.C.; Castro A.E.; Davis, P.L.; Reynolds, B.J. 1990. Herpes viral hepatitis in a toucan. Avian Diseases 34:787–790.
6. Clipsham, R. 1997. Beak repair, rhamphorthotics. In R.B. Altman, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 773–786.
7. Cornelissen, H.; Ducatelle, R.; and Roels, S. 1995. Successful treatment of a channel-billed toucan (*Ramphastos vitellinus*) with iron storage disease by chelation therapy: Sequential monitoring of the iron content of the liver during the treatment period by quantitative chemical and image analyses. Journal of Avian Medicine and Surgery 9:131–137.
8. Cornelissen, J.M.M.; van den Brink, M.E.; Bakker, M.H.; and Koopman, J.P. 1991. Cloacal microflora of healthy hornbills, toucans and aracaris. In Proceedings of the 1st conference of the European Association of Avian Veterinarians. Vienna, Austria, pp. 452–460.
9. Cubas, Z.S.; Lange, R.R.; Busetti, E.T.; Thomaz Soccol, V.; and Castro, E.A. 1987. Infecção por *Capillaria columbae* em *Ramphastos toco* em Curitiba-PR [Infection by *Capillaria columbae* in *Ramphastos toco* (toco toucan) in Curitiba, PR, Brazil], (summary 529). In Proceedings of the 14th Congresso Brasileiro de Zoologia, Juiz de Fora, p. 489.
10. Frankenhuis, M.T.; van Eyk, H.G.; Assink, J.A.; and Zwart, P. 1989. Iron storage in livers of birds of paradise. In Proceedings of the 2nd European Symposium of Avian Medicine and Surgery. Utrecht, the Netherlands, pp. 92–95.
11. Fudge, A.M. 1997. Avian clinical pathology(Hematology and chemistry. In R.B. Altman, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 142–157.
12. Fudge, A.M. Ed. 1999.Laboratory medicine: Avian and exotic pets. Philadelphia, W.B. Saunders.
13. Giddings, R.F. 1988. Treatment of flukes in a toucan. Journal of the American Veterinary Medical Association 193 (12):1555–1556.
14. Godoy, S.N.; Cubas, Z.S.; de Paula, C.D.; and Catão-Dias, J.L. 1999. Surto de S*arcocystis sp*. em aves exóticas e neotropicais mantidas em cativeiro [An outbreak of *Sarcocystis* sp. in captive neotropical and exotic birds]. In Proceedings of the 9th ENAPAVE (Encontro Nacional de Patologia Veterinária [Brazilian Meeting of Veterinary Pathology]), Colégio Brasileiro de Patologia Animal, Belo Horizonte, MG, pp. 69.
15. Kahler, J.; and Kornelsen, M.J. 1994. Sandostatin R (synthetic somatostatin) treatment for *diabetes mellitus* in a sulfur breasted toucan (*Ramphastos sulfuratus sulfuratus*). In Proceedings of the Association of Avian Veterinarians. Lake Worth, pp. 269–273.
16. Lainson, R. 1994. Observations on some avian coccidia (Apicomplexa: Eimeriidae) in Amazonian Brazil. In Memórias do Instituto Oswaldo Cruz 89(3):303–313.
17. Lowenstine, L.J.; and Munson, L. 1999. Iron overload in the animal kingdom. In M.E. Fowler and R.E. Miller, eds., Zoo and Wild Animal Medicine, Vol. 4. Current Therapy. Philadelphia, W.B. Saunders, pp. 260–268.
18. Southern, L.L.; and Baker, D.H. 1982. Iron status of the chick as affected by *Eimeria acervulina* infection and by variable iron ingestion. Journal of Nutrition 112:2353–2363.
19. Upton, S.J.; Ernst, J.V.; Clubb, S.L.; and Current, W.L. 1984. *Eimeria forresteri* (Apicomplexa: Eimeriidae)

from *Ramphastos toco* and redescription of *Isospora graculai* from *Gracula religiosa*. Systematic Parasitology 6(3):237–240.

20. Walker, R.L; Anderson, M.A.; Loretz, K.J.; Woods, L.W.; Johnson, B.; and Hess, P. 1996. Ulcerative enteritis in toucans. In Proceedings of the 39th American Association of Veterinary Laboratory Diagnosticians (AAVLD) Annual Meeting. Little Rock, Arkansas, p. 19.
21. Wilson, R.B. 1994. Hepatic hemosiderosis and klebsiella bacteremia in a green aracari (*Pteroglossus viridis*). Avian Diseases 38:679–681.
22. Worell, A. 1997. Toucans and mynahs. In R.B. Altman, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 910–917.
23. Worell, A. 1996. Medicine and surgery of toucans. In W.J. Rosskopf, Jr. and R.W. Woerpel, eds., Diseases of Cage and Aviary Birds, 3rd Ed. Baltimore, Williams and Wilkins, pp. 933–943.
24. Worell, A. 1988. Management and medicine of toucans. In Proceedings of the Annual Conference of the AAV (Associations of Avian Veterinarians). Chicago, Illinois, pp. 253–261.
25. Worell A. 1991. Serum iron levels in ramphastids. In Proceedings of the Annual Conference of the AAV (Association of Avian Veterinarians). Chicago, Illinois, pp. 120–130.

20 Order Passeriformes (Songbirds)

Marta Brito Guimarães
Silvia Neri Godoy

ANESTHESIA

Marta Brito Guimarães

Passeriformes (passerines) are difficult to anesthetize because of the stress involved with capture. Within this order, common birds seen in private South American clinics are hooded siskin (*Carduelis magellanicus*), chopi blackbird (*Gnorimopsar chopi*), red-crested cardinal (*Paroaria coronata*), lesser seed finch (*Oryzoborus angolensis*), green-winged saltator (*Saltator similis*), great-billed seed finch (*Oryzoborus maximiliani*), sayaca tanager (*Thraupis sayaca*),and thrushes (*Turdus* spp.).

INJECTABLE ANESTHETICS

Tiletamine/Zolazepam

The most frequently used injectable anesthetic agent in Brazil is tiletamine/zolazepam (Zoletil, Telazol), used to immobilize birds for noninvasive surgical procedures. Even in high doses, when used in quail,[7] an anesthetic plane was not reached. Of a group of 35 species tested, it was possible to anesthetize only a green-backed heron (*Butorides virescens*). It is common to combine this agent with atropine, which acts as a muscarine cholinergic receptor blocker, to reduce respiratory tract secretions, which may suffocate a bird. The atropine dose for passerines is 0.2 mg/kg. It is diluted in sterile isotonic saline and administered intramuscularly before the administration of Zoletil at a dose of 20 mg/kg.

During the procedure, the bird should be kept warm with a warm water heating pad or warm gel bags laid underneath it. Recovery may take from 30 minutes to a few hours, depending on the species. During this time, the bird should be kept wrapped in a piece of cloth or in a small paper or plastic tube to minimize self-trauma.

Ketamine

Ketamine is the drug most often used by veterinarians in Brazilian zoos because of its low cost and availability. With this agent, muscular tonicity is raised, causing quick contractions. Although it is a good analgesic, it is not recommended for laparoscopy because of the high dose required. Given with no other drug, it can be used for minor surgical interventions such as limb amputation in high-risk patients,[4] especially wings of aquatic birds. It is also used to facilitate handling of difficult patients during inhalant induction or intubation.[1]

Benzodiazepine

Diazepam is the most commonly used benzodiazepine. It increases the action time of other anesthetics and is commonly used in combination with ketamine to reduce the hallucinogenic effects of other dissociative drugs.[2] The recommended dose for this combination is 1 mg/kg of diazepam and 45 mg/kg ketamine.

In passerines, the doses used are low, and agents should be diluted with sterile isotonic solution. These birds have little muscular mass for injecting the drug.

INHALANT ANESTHESIA

Induction

Birds have an efficient air exchange system, associated with large air sac volumes, which may make them more susceptible than mammals to overdose with inhalant anesthetics.[1] To anesthetize a passerine it is necessary to apply a well-fitting mask. Because of their size, passerines are not intubated, but are kept anesthetized using a mask. It is possible to quickly remove the mask and allow the bird to receive oxygen from the environment or from an oxygen cylinder.

Inhalant Anesthetic Agents

Isoflurane

Isoflurane is the best anesthetic agent for use in passerines. It may be used for prolonged surgeries or to keep birds anesthetized to collect blood, administer fluids, perform radiographic or endoscopic examinations, or apply compresses.[6] The induction concentration is 3–4%, and anesthesia may be maintained at 1% with a flow of 1–2 L/min. No side effects are seen in passerines, such as the cardiac disease seen in chickens or turkeys anesthetized with isoflurane. This drug is considered to be a good anesthetic for emergency procedures.[5]

Halothane

Halothane gas in high concentration (2–4%) produces a rapid induction in all pet birds.[2] Anesthesia may be maintained using an average concentration of 1%. It may be necessary to vary the concentration, depending on the species and the health status of the patient. This anesthetic has the advantages of being inexpensive and having low tissue solubility. It produces moderate muscular relaxation.[1]

REFERENCES

1. Altman, R.B.; Clubb, S.L.; Dorrestein, G.M.; and Quesenberry, K. 1997. Avian Medicine and Surgery. Philadelphia, W.B. Saunders.
2. Hall, L.W.; and Clarke, K.W. 1991. Veterinary Anaesthesia. London, Bailliere Tindall.
3. Lin, H.C.; Thurmon, J.C.; Benson, G.J.; and Tranquilli, W.J. 1993. Telazol—A review of its pharmacology and use in veterinary medicine. Journal of Veterinary Pharmacology and Therapeutics 16(4):383–418.
4. Massone, F. 1999. Anestesiologia Veterinária (Farmacologia e Técnicas. Rio de Janeiro, Guanabara Koogan.
5. Lkowski, A.A.; and Classen, H.L. 1998. Safety of isofluorane anaesthesia in high-risk avian patients. The Veterinary Record 143:3.
6. Ritchie, B.W.; Harrison, G.J.; and Harrison, L.R. Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing.
7. Nicolau, Alexandra Alves. 1997. Efeitos da Associação tiletamin/zolazepam em codornas (*Coturnix coturnix japonica*) defendida por Alexandre Alves Nicolau. Masters thesis, Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo.
8. Warren, R.G. 1986. Anestesia de Animales Domesticos. Barcelona, Spain, Labor.

GENERAL MEDICINE

Silvia Neri Godoy

FUNGAL DISEASES

Yeast and fungi are part of normal bird flora; they inhabit the gastrointestinal tract and skin.[2,3,4,18,13] Prolonged antibiotic therapy, inadequate hygiene, nutritional deficiencies,[3,4,6,7,16,18] high-carbohydrate diets,[16,18] and concurrent diseases are conditions that predispose birds to yeast and fungal diseases.[3]

Birds in the wild are less susceptible to these diseases because they are not in contact with predisposing factors. Newly hatched offspring may be subject to infection, depending on the material with which the nest is made, the number of chicks hatched, and the local temperature and humidity.[15]

Fungal diseases are common in illegally traded birds, for they are subjected to stressful situations from the moment of capture, including dietary changes, malnutrition, and, often, poor sanitary conditions.

Candidiasis

The epidemiology, diagnosis, and management of candidiasis in passerine birds is the same as for other birds.

The most effective treatment is nystatin for 15 days at dosages of 100,000 IU/L in the drinking water or 200,000 IU/kg in the feed.[6,7] Supplementation with vitamins, immunostimulant drugs, and reduction of carbohydrate levels in the diet may improve the treatment.

In Brazil, candidiasis has been reported in many wild bird species, such as the lesser seed finch (O. *angolensis*),

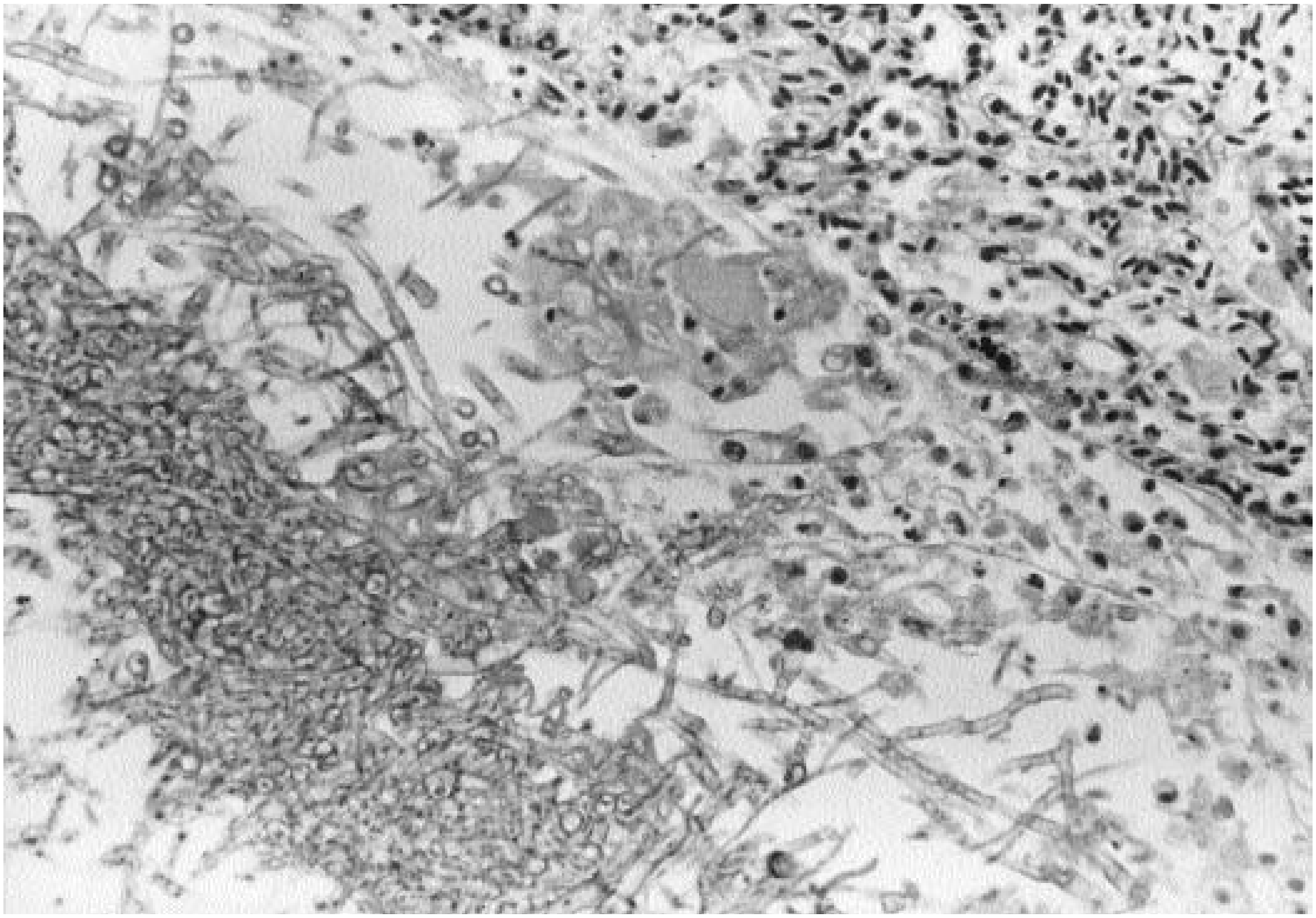


FIGURE 20.1. Photomicrograph of red-crested cardinal (*Paroaria coronata*) lung. Presence of a great quantity of *Aspergillus* spp. hyphae, associated with inflammatory cell infiltration and hemorrhage.

rufous-bellied thrush (*Turdus rufiventris*), ultramarine grosbeak (*Cyanocompsa cyanea*), saffron finch (*Silicalis flaveola*),[14] red-cowled cardinal (*Paroaria dominicana*), and red-crested cardinal (*P. coronata*).

Aspergillosis

Aspergillosis is a common fungal infection of stressed birds. The epidemiology, diagnosis, and management are similar to those of other avian species (see Figures 20.1 and 20.2).

In Brazil, in addition to the red-crested cardinal and red-cowled cardinal, the disease has been reported in wild canaries (*Serinus canarius*), lined seedeaters (*Sporophila lineola*), jays, large-billed seed finches (*Oryzoborus crassirostris*), black-throated grosbeaks (*Pitylus fuliginosos*), lesser seed finches (*O. angolensis*), tanagers, yellow-legged thrushes (*Platycichla flaviceps*), rufous-bellied thrushes (*T. rufiventris*), ultramarine grosbeaks (*C. cyanea*), and other seedeaters.[14]

VIRAL DISEASES

Poxvirus

Poxvirus may affect birds of any age; susceptibility varies according to the type of virus and the species of bird.[24] Transmission is related to direct contact with contaminated feed, water, discharges, and objects; indirect contact (e.g., insect bites) is also possible. Many *Diptera* spp. and *Dermanyssus gallinae* may be mechanical vectors of the agent.[24]

There are three variations of the disease: cutaneous, septicemic, and diphtheritic. The form developed depends upon the virulence of the virus strain, lesion distribution, and bird susceptibility.[13,24] A poor environment and continuous stress may contribute to the activation of a latent virus and to an increase in the pathogenicity of the disease. The illness seems to be autolimiting in passerines in the wild.[24]

The cutaneous form of the disease, also known as bouba, is the most common variation. Red-crested car-

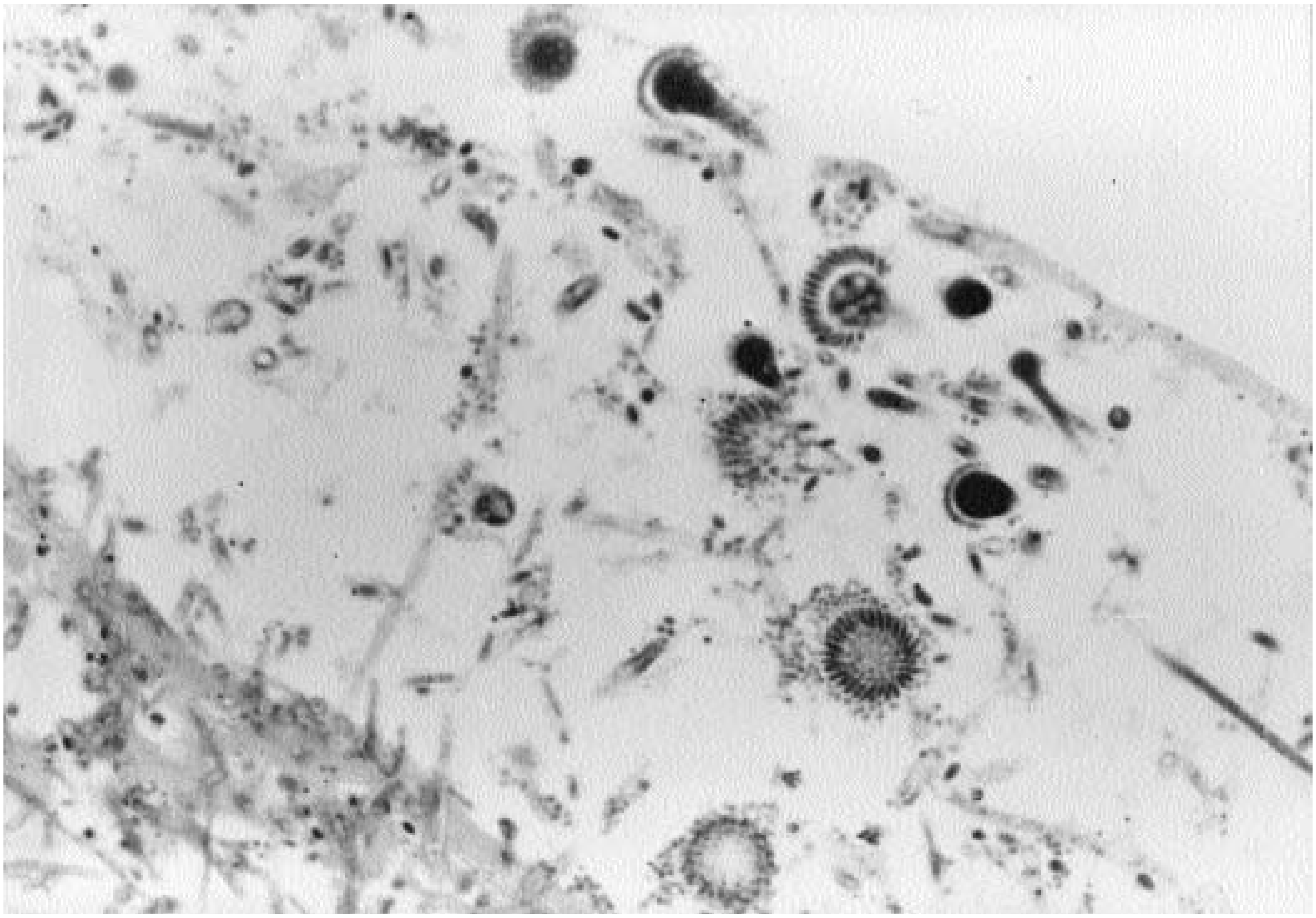


FIGURE 20.2. Photomicrograph of red-crested cardinal (*Paroaria coronata*) lung. Presence of structures similar to *Aspergillus* spp. conidiophores and hyphae.

dinals, red-cowled cardinals, and saffron finches are highly susceptible. Clinical manifestation of the disease is determined by the presence of proliferative nodular lesions in apteric regions, mainly located in the lower limbs, around the eyes, nostrils, and beak commissures (Figure 20.3). Sometimes, these nodules coalesce to produce large masses that affect the sight or the adequate opening of the beak. In mild forms of the disease, small cutaneous lesions may pass unnoticed.[24] Lesions are seldom infected by secondary agents;[21] however, they may become hemorrhagic and inflamed.[24] Microscopically, it is possible to view epithelial cell hyperplasia and large intracytoplasmatic eosinophilic inclusions called Bollinger bodies (Figure 20.4).

In the diphtheritic form of the disease, there are proliferative necrotic pseudomembranous lesions in the mucous membrane of the digestive and upper respiratory tract. These lesions may block the air flow and interfere with swallowing, causing anorexia, dyspnea, and possibly death. Signs are edema, hydropic degeneration and epithelial squamous metaplasia in the upper

FIGURE 20.3. Presence of nodular lesions around the beak and nostrils of a red-crested cardinal (*Paroaria coronata*).

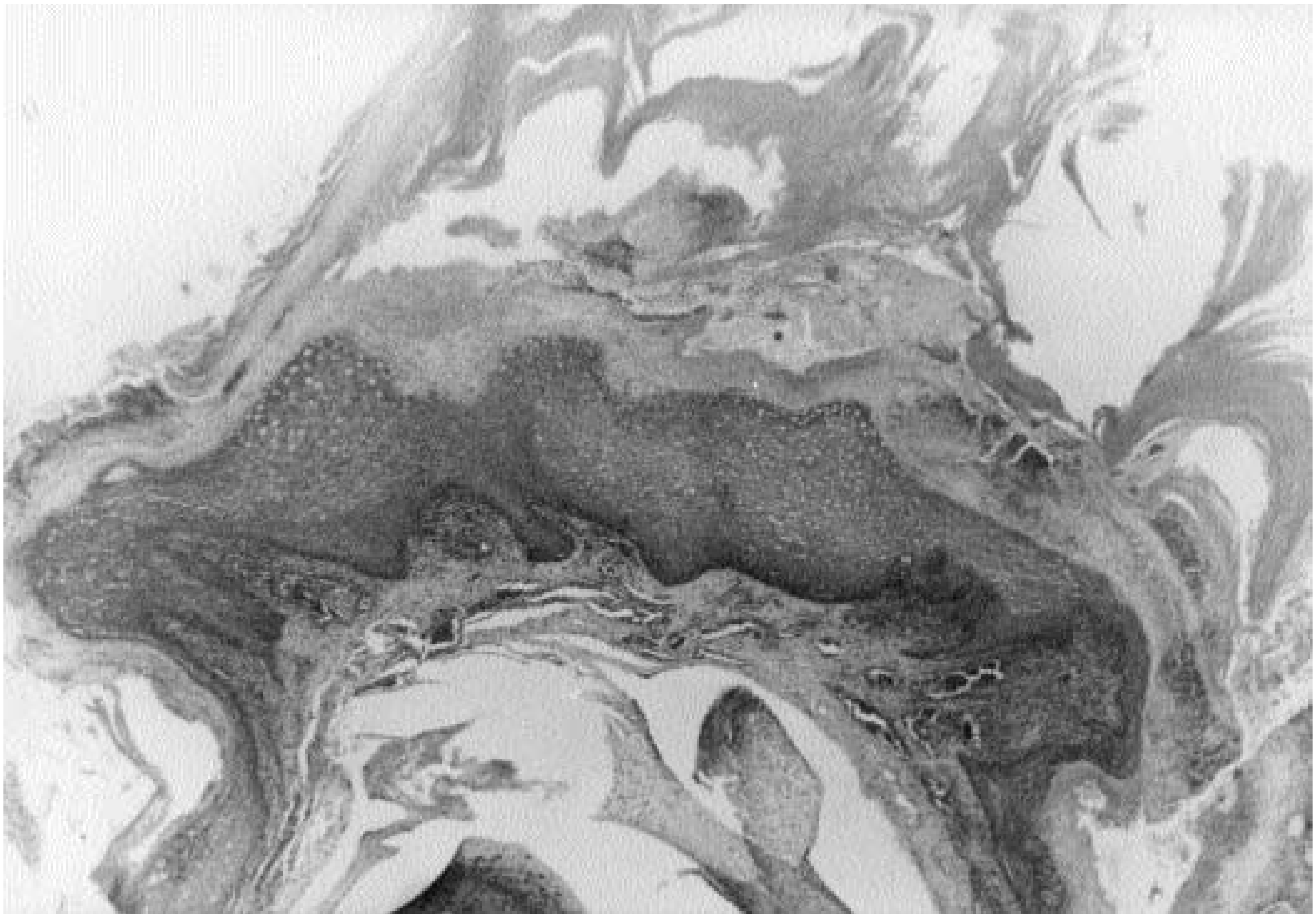


FIGURE 20.4. Photomicrograph of the skin of the saffron finch (*Silicalis flaveola*) presenting epithelial cell hyperplasia, associated with characteristic large intracytoplasmic inclusions.

respiratory tract, hyperplasia of the mucous glands, and epithelial cell inclusions.[24]

Canaries are highly susceptible to the septicemic form of the disease.[5,7,10,21,24] A high mortality rate may result from acute necrotic bronchiolar pneumonia.[7,10,13,21] Signs are prostration, dyspnea, and ruffled feathers; birds die in 3–4 days.[10]

In the septicemic form, inclusions may or may not be seen in the epithelium of the lungs or trachea, because of the rapid onset of the disease.[21] Findings may be limited to acute inflammation of the bronchial tubes. In less severe cases, inclusions may be seen in the epithelial cells of the lungs, skin, and respiratory tract.[10,13,20,21]

The most simple and common diagnostic technique is the demonstration of large intracytoplasmic inclusions in the epithelial cells (Figure 20.4). Inclusions may also be detected in smears stained by Giemsa.[24] Additionally, viral particles may be observed by electron microscopy. The virus will replicate in embryonated eggs.[24] Serologic tests are also employed.

The diphtheritic form must be differentiated from laryngotracheitis, herpesviruses, vitamin A deficiency, lesions caused by *Trichomonas* spp., and Newcastle disease.[5,24] The cutaneous form must be differentiated from insect bites.[13]

There is no effective treatment, and support treatment varies according to clinical signs. Antibiotic therapy, immunostimulant drugs, and vitamin supplementation may improve recovery.

Lymphoid Leucosis

Lymphoid leucosis is caused by one of the retrovirus-induced neoplasms in the leucosis/sarcoma complex.[19,21] Erythroid leucosis, myeloid leucosis, renal tumors, and hemangioma may also occur.[21]

Transmission is by direct or indirect contact with contaminated feces, nasal or oral discharges, semen, or blood. It may also be vertically transmitted.[21] Signs vary according to tumor location and the presence or absence of secondary infections.[21] Generally, the disease

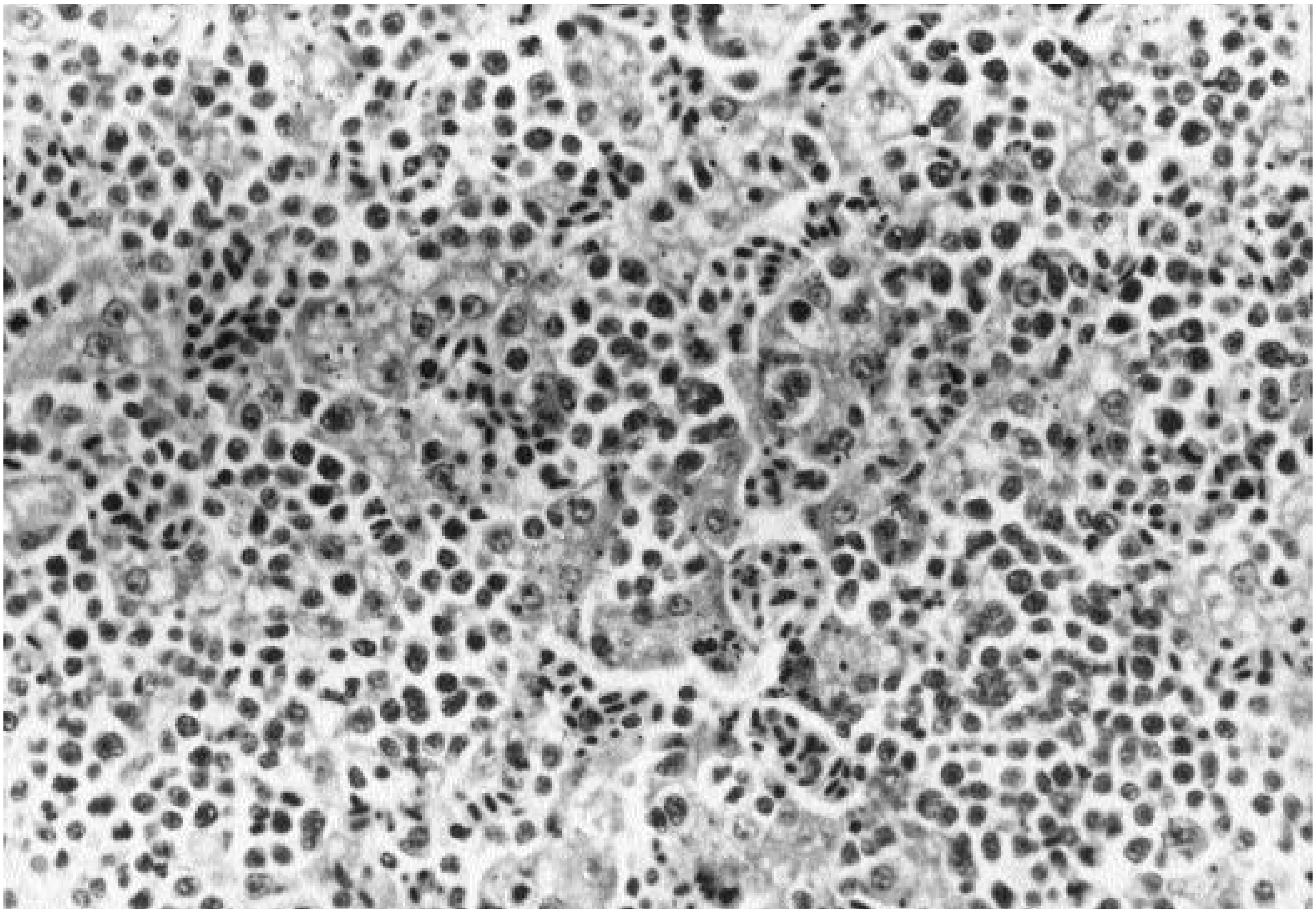


FIGURE 20.5. Photomicrograph of the liver of a zebra finch (*Taeniopyzia guttata*). Hepatic distrabeculation is associated with severe lymphoid cell infiltrate, presenting a high mitosis rate.

begins with prostration, ruffled feathers, and an increase in abdominal volume. Tumors primarily affect the liver; however, in a generalized infection, the spleen, kidneys, gonads, lungs, bone marrow, mesentery, and bursa are also affected. The liver and the bursa are generally palpable.[19]

At necropsy, the affected organs are increased in size and are pale;[10,20] however, variations occur according to the severity of the disease. Microscopic examination shows lesions that may affect every organ; in the beginning they are immature lymphoid cell infiltrates. These infiltrates coalesce and form large diffuse masses (Figure 20.5). A high mitosis rate is seen in these lymphoid cells.

Diagnosis may be made by serological tests or cytology, but it is usually confirmed at necropsy.[21] There are few findings related to the disease in Brazilian wild birds; most information has been developed from the detection of the virus in exotic birds, which are intensely bred in Brazil.

There is no effective treatment, but antibiotic therapy, immunostimulant drugs, and prednisolone have yielded good results in some birds.

Papovavirus

The genera papillomavirus and polyomavirus, which belong to the Papovaviridae family, cause different diseases in passerines. These diseases seem to occur more frequently than are diagnosed.[17,20]

Papillomavirus is associated with benign epithelial tumors in birds.[17,21] The legs are mainly affected, presenting hyperkeratinized lesions, distorted toes, excessive growth, and functional disorders.[17] Lesions may also be present in the commissures of the beak of young canaries.[21] Papillomatous lesions in the digestive epithelium may or may not be caused by the virus and must be differentiated from chronic irritation.[21]

Histologically, there is hyperkeratosis and acanthosis of the epidermis.[17,21] There is no metastasis or infiltration.[3] Intracellular inclusions may be seen with an electron microscope.

Recommended treatment is surgical removal of the lesions or cauterization with silver nitrate. The presence of lesions seems to be seasonal, and increases during the summer.

Poliomavirus affects immunosuppressed birds,[21] producing a high mortality rate among nestlings.[9] Young birds that survive the disease experience delayed growth and feathering and dirty feathers.[13]

Chronic disease may result in beak abnormalities and poor development.[7,13,21] Secondary infections are common, mainly caused by *Candida* spp.[13] Macroscopic findings include an enlarged liver that is pale and friable,[7,13,21] splenomegaly, enlarged kidney, and congestion of vessels in the intestinal serosa and brain.

Histologically, splenitis, hepatitis, myocarditis, bone marrow and liver necrosis, and hyperplasia of the spleen and liver macrophages are seen. Intranuclear inclusions may be seen in the kidneys, heart, spleen, intestines, and liver.[7,9,21] These inclusions may be identified by electron microscopy as poliomaviruses.

There is no effective treatment. Neither papillomavirus nor poliomavirus have been isolated in most South American countries; however, it is believed that they are common and have not been detected because of the lack of resources to make the diagnosis.

Paramyxovirus

Of the various types of paramyxovirus, types 1, 2, and 3 may affect passerines. Paramyxovirus type 1 is the agent of Newcastle disease, which is an important disease of poultry, but a few reports relate to passerines. Wild birds are considered to be agents of dispersion of the virus.[1] Affected birds exhibit respiratory, gastrointestinal, and neurological signs.[1,5,12] At necropsy, hemorrhagic lesions are found in various organs.[5,11]

Paramyxovirus types 2 and 3 are the most common in passerines.[1,21] Type 2 seems to be self-limiting or asymptomatic,[13] but may cause respiratory disorders. Type 3 is the most common in tropical birds, and causes conjunctivitis, followed by anorexia, diarrhea, dyspnea, and neurological signs, such as torticollis and circling gait.[1,7,13,21,22,23]

Transmission is by contact with contaminated feces and respiratory tract discharges. Macroscopic findings depend on the signs. Diagnosis is made by isolation of the virus.

Other Viral Diseases

Other viruses may affect passerines; however, they have not been diagnosed, probably because of the lack of resources in South American countries. There are some reports of such viruses as cytomegalovirus, which is a herpesvirus that causes depression, anorexia, conjunctivitis, dyspnea, and has a high mortality rate[13,5] and many arboviruses that cause encephalitis,[5,8,26] such as Venezuelan equine encephalitis (VEE) that is transmitted by insect bites and affects a great number of wild passerines of the Brazilian rain forests.[8,26]

PROTOZOAL DISEASE

Sarcosporidiosis

The most common causative agent of sarcosporidiosis in birds is *Sarcocystis falcatula,* a protozoan that requires two hosts (birds and opossums) to complete its life cycle. Birds are intermediate hosts and ingest oocysts present in opossum feces. They may present two types of clinical signs: cysts of the parasite in the muscles or the respiratory form of the disease (the most common cause of death of Old World psittacines).

Cysts are the most common manifestation of the disease in passerines, which are not detrimental to the health of the birds. However, intense and frequent exposure to the agent may lead to the pulmonary form of the disease and death as a result of massive lung hemorrhage, caused by the rupture of endothelial cells in the pulmonary vessels as a result of massive multiplication of the parasite. Generally, the disease is hyperacute, and there is no clinical sign, but sudden death.

Macroscopically, in the pulmonary disease edema of the lung is observed, along with congestion and hemorrhage, commonly associated with spleno- and hepatomegaly. Microscopically, the most significant findings are pear-shaped structures typical of *Sarcocystis* spp. inside or near endothelial cells of the blood vessels of the lung, associated with large areas of hemorrhage and, generally, pneumonia. It is also possible to observe hepatitis and splenitis associated with intense histiocytosis.

In Brazil, the disease has been reported in the bare-throated bellbird (*Procnias nudicollis*), scarlet tanager (*Ramphocelus brasilius*), blue-chevroned tanager (*Thraupis ornata*), and saffron finch.

Because diagnosis is difficult, treatment is seldom given. However, in highly infested sites, sulfadiazine combined with trimethoprim sulfa may be used as a preventative.

TRAUMA

Lower-limb fractures are common in passerines, either because they fall from their cages or because their long toenails become entangled in the bars of the cages. Bird bones may be immobilized using small pieces of wood or half an insulin syringe to help fracture fixation. Depending on the kind of lesion, needles may be used as pins to improve fracture coaptation. When the femur is fractured, the recommended procedure is restriction of the space in which the bird is kept, because immobilization is too difficult.

Necrosis in the extremities may result from constriction caused by threads from the nest. If the thread is not totally removed, an inflammatory process may be established and bacterial contamination may occur. In the most severe cases, the affected limb should be amputated before total necrosis occurs.

REFERENCES

1. Alexander, D.J. 1993. Paramyxovirus infection. In J.B. McFerran and M.S. McNulty, eds., Virus infections of Vertebrates, Vol. 4. Virus Infections of Birds. Amsterdam, Elsevier Science, pp. 321–340.
2. Bauck, L. 1994. Mycoses. In G.J. Harrison, L.R. Harrison, and B.W. Ritchie, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 997–1006.
3. Carcia, R.G.F; and Schönhofen, C.A. 1988. Ocorrência de candidíase em mainá (*Gracula riligiosa*). Revista do Setor de Ciências Agrárias 10(1–2):211–213.
4. Coles, B.H. 1985. Infectious diseases of birds: Mycotic diseases. In B.H. Coles, ed., Avian Medicine and Surgery. Oxford, Blackwell Scientific, pp. 230–231.
5. Coles, B.H. 1985. Infectious diseases of birds: Viral diseases. In B.H. Coles, ed., Avian Medicine and Surgery. Oxford, Blackwell Scientific, pp. 226–229.
6. Dorrestein, G.M. 1996. Medicine and surgery of canaries and finches. In W. Rosskopf and R. Woerpel, eds., Diseases of Cage and Aviary Birds. Baltimore, William & Wilkins, pp. 915–927.
7. Dorrestein, G.M. 1997. Passerines. In R.B. Altman, ed., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 867–885.
8. Ferreira, I.B.; Pereira, L.E.; Rocco, I.M.; Marti, A.T.; Souza, I.T.M.; Iversson, L.B.; and De-Souza, L.T.M. 1994. Surveillence of arbovirus infections in the Atlantic forest region, State of São Paulo, Brazil, I. Detection of hemagglutination-inhibiting antibodies in wild birds between 1978 and 1990. Revista do Instituto de Medicina Tropical de São Paulo 36(3):265–274.
9. Forshaw, D.; Wylie, S.L.; and Pass, D.A. 1988. Infection with a virus resembling papovavirus in Gouldian finches (*Errythrura gouldiae*). Australian Veterinary Journal 65(1):26–28.
10. Johnson, B.J.; and Castro, A.E. 1986. Canary pox causing high mortality in an aviary. Journal of the American Veterinary Medical Association 189(10):1345–1347.
11. Khalafalla, A.I.; Nayil, A.A.; Nimir, A.H.; and Hajer, I. 1990. Role of some Passeriformes birds in transmission of Newcastle disease, I. Susceptibility of some wild birds of Sudan to Newcastle disease virus. Bulletin of Animal Health and Production in Africa 38(1):45–49.
12. Khalafalla, A.I.; Nayil, A.A.; and Nimir, A.H. 1990. Role of some Passeriformes birds in transmission of Newcastle disease, II. Pathogenesis of Newcastle disease virus in Sudan house sparrows (*Passer domesticus arborius*). Bulletin of Animal Health and Production in Africa 38(1):51–54.
13. MacWhiter, P. 1994. Passeriformes. In G.J. Harrison., L.R. Harrison, and B.W. Ritchie, eds., Avian Medicine: Principles and Application. Lakeworth, Florida, Wingers Publishing, pp. 1172– 1199.
14. Nakano, M. 1977. Doenças de Aves do Estado de São Paulo. São Paulo, Instituto Biológico de São Paulo, Secretária da Agricultura e Abastecimento.
15. O'Meara, D.C.; and Witter, J.F. 1977. Aspergillosis. In J.W. Davis, L. Karstad, R.C. Anderson, and D.O. Trainer, eds., Infectious and Parasitic Diseases of Wild Birds. Ames, Iowa, Iowa State University Press, pp. 157–167.
16. O'Meara, D.C.; and Witter, J.F. 1977. Candidiasis. In J.W. Davis, L. Karstad, R.C. Anderson, and D.O. Trainer, eds., Infectious and Parasitic Diseases of Wild Birds. Ames, Iowa, Iowa State University Press, pp. 168–174.
17. Osterhaus, A.; and Moreno-Lopes, J. 1993. Papovavírus. In J.B. McFerran and M.S. McNulty, eds., Virus Infections of Vertebrates, Vol. 4, Virus Infections of Birds. Amsterdam, Elsevier Science, pp. 147–151.
18. Patgiri, G.P. 1987. Systemic mycoses. In E.W. Burr, ed., Companion Bird Medicine. Ames, Iowa, Iowa State University Press, pp. 102–106.
19. Payne, L.N. 1993. Avian leukosis/sarcoma. In J.B. McFerran and M.S. McNulty, eds., Virus Infections of Vertebrates, Vol. 4. Virus Infections of Birds. Amsterdam, Elsevier Science, pp. 411–435.
20. Real, F.; Acosta, B.; Deniz, M.S.; Rodrigues, F.; and Espinosa, A. 1994. Pathological study of two outbreaks of pox in canaries. Medicina-Veterinaria 11(4):232–234.
21. Ritchie, B.W. 1995. Avian Viruses: Function and Control. Lakeworth, Florida, Wingers Publishing.
22. Schemera, B.; Toro, H.; Kaleta, E.F.; and Herbst, W. 1987. A paramyxovirus of serotype 3 isolated from African and Australian finches. Avian Diseases 31(40):921–925.
23. Shihmanter, E.; Weisman, Y.; Lublin, A; Mahani, S.; and Panshin, A. 1998. Isolation of avian serotype 3 paramyxoviruses from imported caged birds in Israel. Avian Diseases 42(4):829–831.
24. Tripathy, D.N. 1993. Avipoxviruses. In J.B. McFerran and M.S. McNulty, eds., Virus infections of Vertebrates, Vol. 4. Virus Infections of Birds. Amsterdam, Elsevier Science, pp. 5–15.
25. Tsai, S.S.; Park, J.H.; Hirai, K.; and Itakura, C. 1992. Aspergillosis and candidiasis in psittacine and passeriform birds with particular reference to nasal lesions. Avian Pathology 21:699–709.
26. Vasconcelos, P.F.C.; Travassos da Rosa, J.F.S.; Travassos da Rosa, A.P.A.; Dégallier, N.; Pinheiro, F.P.; and Sá Filho, G.C. 1991. Epidemiologia das encefalites por arbovírus na Amazônia Brasileira. Revista do Instituto de Medicina Tropical de São Paulo 33(6):465–476.

SELECTED INFECTIOUS DISEASES

Marta Brito Guimarães

Chlamydiosis

This disease is caused by *Chlamydia psittaci* and can affect more than 100 avian species.[1] There is no confirmation of this disease in Brazilian passerines because of limited diagnosis techniques. Furthermore, Passeriformes are less susceptible to *C. psittaci* than psittacines[7]. Psittacosis is suspected when symptoms don't disappear after broad spectrum antibiotic treatment. It is possible to treat chlamydiosis with doxycycline treatment P.O. (50–100 mg/kg orally) once a day for 45 days. A successful treatment was achieved for a saffron finch (*S. flaveola*) suffering from conjunctivitis using this protocol only.

The birds may present anemia, leukocytosis, and heterophilia or monocytosis. There may be raised levels of enzymes such as aspartate aminotransferase (AST), lactate dehydrogenase (LDH), bile acids, and occasionally uric acid. The gross lesions are: hepatomegaly where the liver is congested and friable; hepatitis; splenomegaly with congested spleen and splenitis; air sack opacity and thickness with fibrin deposition; fibrinous pericarditis; bronchopneumonia; nephrosis; glomerulonephritis; enteritis; conjunctivitis; and keratitis. Not all birds present these classic symptoms. In histopathological exams the lesions vary within the host and the *Chlamydia* strain involved. It has been seen as a focal necrosis in lungs, spleen, and kidney, as well as monocyte proliferation, epithelioid granuloma in liver and lungs, bronchopneumonia, fibrinous airsacculitis; peritonitis, pericarditis, and orchitis. Nonsuppurative meningitis is seen in birds with central nervous system disorders.[1,6]

Mycoplasmosis

Mycoplasmosis is common in passerines such as lesser seed finch (*O. angolensis*), great-billed seed finch (*O. maximiliani*), green-winged saltator (*S. similis*), hooded siskin (*C. magellanicus*), and chopi blackbird (*G. chopi*). The isolation of *Mycoplasma gallisepticum* in these birds is not confirmed. However, it has been described in sparrows (*Passer domesticus*)in India.[3] These birds may present simple symptoms such as conjunctivitis, tracheitis, and syrinxitis, or have infraorbitary sinus, air sacks, and lungs compromised. It may also compromise what is most prized: the song.

The loss of vocalization or a simple hoarseness can be treated with specific antibiotics. Some antibiotics are not used to eradicate mycoplasma but to reduce clinical lesions. They are: erythromycin, tylosin, tiamulin, gentamicin, tetracycline, streptomycin, spiramycin, lincomycin, and spectinomycin. Erythromycin is used at a dose of 60 mg/kg every 12 hours for 15 days, although the symptoms may reoccur. It has been observed that these birds sing less after being handled for long periods of time. Therefore, instead of being injected in birds, the antibiotics are injected into larva, the main diet of these birds, to disguise the drug's displeasing flavor. It is also possible to manipulate the drug, adding to it flavors accepted by the animals. The administration of tylosin through drinking water at a dose of 1 mg/mL for at least 21 days and eye drops of cyprofloxacin were the best protocols to treat passerines presenting conjunctivitis caused by *M. gallisepticum*.[4]

Colibacillosis

This disease is caused by the bacteria *Escherichia coli* and is the major cause of mortality for imported[2] or wild[5] passerines. The animals develop sepsis that can be combined with many diseases. It can proliferate due to lack of hygiene, fecal contamination of water, food, perch, cage pavement, and environment by sick birds.[8] In a chopi blackbird (*G. chopi*) presenting purulent sinusitis, *E. coli* was isolated and showed enrofloxacin sensitivity. Depending on the species, a dose of 5–10 mg/kg every 12 hours for a week was used with great success when combined with improving the hygiene in the cages.

Salmonellosis

Salmonellosis is caused by *Salmonella* spp., with more than 2100 species. *Salmonella typhimurium* was isolated from two passerines observed in a study.[8] Salmonellosis in Brazil is responsible for the death of many birds in flocks of saffron finch (*S. flaveola*) and hooded siskin (*C. magellanicus*). The transmission may occur through ingestion of contaminated food or water or through direct contact with dirty feathers. The most common symptoms are prostration, depression, lethargy, diarrhea, and death. The gross lesions are hepatomegaly, splenomegaly, pneumonia, and hemorrhagic enteritis. Isolation must be done to confirm the diagnostic.

Enterobacteriaceae

Enterobacteriaceae have been isolated from normal aerobic flora of psittacine and domestic and wild Passeriformes' coproculture.

Normal Intestinal Flora

The smears made of feces from lesser seed finch (*O. angolensis*), great-billed seed finch (*O. maximiliani*), and hooded siskin (*C. magellanicus*) stained by gram method showed a small number of gram-positive bacteria (coccus and bacillus), standard for Passeriformes, such as the saffron finch (*S. flaveola*).

REFERENCES

1. Altman, R.B.; Clubb, S.L.; Dorrestein, G.M.; and Quesenberry, K. 1997. Avian Medicine and Surgery. Philadelphia, W.B. Saunders.
2. Bauck, L. 1989. Diseases of the finch as seen in commercial import station. In Proceedings of the Association of Avian Veterinarians. pp. 196–202.
3. Jain, N.C.; Chandiramani, N.K.; and Singh, I.P. 1971. Studies on avian pleuro-pneumonia-like organisms: Occurrence of Mycoplasma on wild birds. Indian Journal Animal Science 41:301–305.
4. Mashima, T.Y.; Ley, D.H.; Stoskopf, M.K.; Miller, E.A.; Welte, S.C.; Berkhof, F.J.E.; Degernes, L.A.; and Fleming, W.J. 1997. Evaluation of treatment of conjunctivitis associated with *Mycoplasma gallisepticum* in house finches (*Carpodacus mexicanus*). Journal of Avian Medicine and Surgery, 11:1, 20–24
5. Pennycott, T.W.; Ross, H.M.; Mclaren, I.M.; Park, A.; Hopkins, G.F.; and Foster, G. 1998. Causes of death of wild birds of the family Fringillidae in Britain. The Veterinary Record 6:143.
6. Randall, C.J.; and Rodney, R.L. 1996. Colour Atlas of Avian Histopathology.
7. Ritchie, B.W.; Harrison, J.G.; and Harrison, L.R. 1994. Avian Medicine: Principles and application. Lakeworth, Florida, Wingers Publishing.
8. Rosskopf, W.J., Jr.; and Woerpel, R.W. 1996. Disease of Cage and Aviary Birds.

ECTOPARASITES

Marta Brito Guimarães

Knemidocoptic Scabiosis

In passerines, scabies may be caused by mites(*Knemidocoptes* spp.) and several other less-common species. *Knemidocoptes jamaicensis* is the most common agent of lesions in birds. This mite produces nonpruritic hyperkeratosis of the beak, around the eyes, and on the feet, particularly of immunosuppressed birds. Lesions are similar in appearance to honeycombs, having a whitish color. Diagnosis may be confirmed by a deep scrape of the lesion, in which the agent may be seen with an optical microscope. Knemidocoptic scabies may be treated with 1:10 oral ivermectin and thiabendazole parasiticide ointments on the affected areas.

Respiratory Tract Mites

Sternostoma tracheacolum is a tracheal mite that causes changes in bird vocalization, beak chattering, and dyspnea. *Cytodites nudus* is another species that may cause the same signs. It may also cause death in severe cases. The recommended treatment is based on pyrethrin aerosols, which are sprayed over the cages, or the use of a 1:10 ivermectin/propylene glycol solution, administered orally, one or two drops every 15 days, until signs disappear. Chronic cases may develop if the mites have not been totally eliminated from the respiratory tract.

Feather Mites

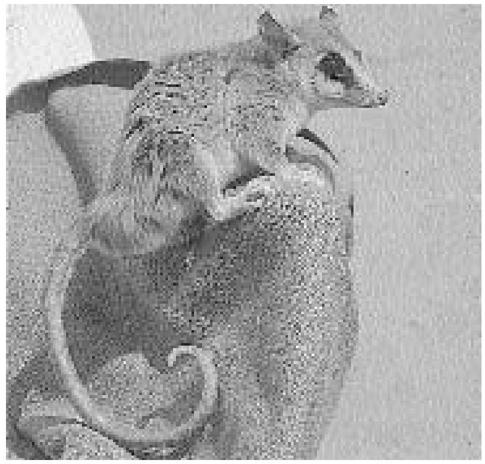
Dermanyssus spp. and *Ornithonyssys* spp. are mites that produce irritation and anemia. When the load is severe, death may occur.

Nonpathogenic feather mites are *Anlages, Megninia,* and *Rivoltasia* spp. Occurrence is not common, but these agents may cause bird disorders. Treatment is based on pyrethrins.

Nonspecific Host Mites

Genera that may affect some passerines are *Dermoglyphus, Syringophilus, Picobia,* and *Harpyhynchus.*

Mammals: Class Mammalia



21
Order Marsupialia (Opossums)

Murray E. Fowler

BIOLOGY AND MEDICINE

BIOLOGY

Marsupials are believed to have originated in North America and then dispersed to Central and South America and Australia.[4,7,10,13] The only North American species, the Virginia opossum, *Didelphis virginianum,* migrated back to North America via the Caribbean land bridge from South America. Australian marsupials have been studied more intensively than those of the Americas. Considerable information is available on a few New World species, such as the Virginia opossum and some others that have been used as experimental animals (*Didelphis, Marmosa, Monodelphis*). Little is known about the other South American marsupials (SAMs).[8]

Taxonomy

South American marsupials are classified in three families, with 11 recent genera and 75 species.[4,7,10,14] Table 21.1 lists some biological data on the genera and selected species. The family Didelphidae is the largest group, and members of the group are considered to be true opossums. The genus *Didelphis* is the most widely distributed mammalian genus, other than humans, in the Western Hemisphere and the most widely distributed marsupial in the world.[7,14] Caenolestidae and Microbiotheridae are primitive marsupials living in scattered pockets west of the Andes mountains. See Table 21.1 for the general distribution of SAMs.

Habitat Requirements

Marsupials have radiated into almost all the habitats of the Western Hemisphere, including the semiaquatic habitat. They are extremely adaptable, but relatively noncompetitive, and do not dominate any niche.[7,14] Some of the didelphids have adjusted to the presence of people and exploit the suburban environment. Most species are arboreal, thus they tend to be found more in forested regions. A few species are restricted to tropical regions (see Table 21.1).

Opossums are nomadic, solitary animals that are capable of exploiting varied food sources. Most SAMs are nocturnal or crepuscular.

Exploitation

None of the SAMs have particularly fine pelts and are generally not palatable, so they aren't hunted or trapped. Predators include all the larger raptorial birds and all the carnivore species.

TABLE 21.1. South American marsupials: biological data (Order: Marsupialia; Families: Didelphidae and Caenolistidae)

Scientific Name	Name (English)	Name (Spanish)	Name (Portuguese)	Distribution	Weight
Chironcetes minimus	Yapok, water opossum	Ratón de agua, comadreja de agua	Cuica-d'água, cachorro d'água	Southern Mexico to northeast Argentina	600–800 g
Didelphis spp.	Long-eared opossum Speckled opossum	Zarigueya, orejuda Comadreja overa	Gambá, mucura Gambá, ucura		
D. marsupialis	Crab-eating opossum	Zaregueya cangrejera, mbicuré cangrejero	Gambá, sarigüéa	Mexico to northern Argentina	2–5.5 kg
Dromiciops australis	Kongoy-kongoy	Monito del monte, colo colo		Chile	16.7–31.4 g
Glironia venusta	Bushy-tailed opossum, squirrel-tailed opossum	Glironia		Upper Amazon regions of Peru, Bolivia, Ecuador	
Lestodelphis halli	Patagonian opossum	Lestodelfo		Patagonian pampas	
Lutreolina crassicaudata	Thick-tailed opossum, little water opossum	Coligueso, comadreja colorado		East of the Andes in Bolivia, Southern Brazil, Paraguay, Uruguay and northern Argentina	200–500 g
Marmosa spp. 44 species	Murine or mouse marmosa	Marmosa ratón, achocaya ratón	Gambasinha	Throughout southern Mexico, Central and South America	♂ up to 130 g ♀ up to 70 g
Metacirops spp. *(Caluromys)*	Grey & black four-eyed opossums, wooly opossum	Cuica, zorrito de palo	Quaicuica, cuica	Northeastern Mexico to Peru and northeastern Argentina	240–400 g
Metachirus naudicaudatus	Rat-tailed opossum, brown four-eyed opossum	Cuica de cola rata, cuica cola de rata	Cuíca cauda-de-rato, Cuíca verdadeira		Up to 800 g
Minuania Subgenus of *Monodelphis*	Little red opossum	Minuania, comadreja colorada			
Monodelphis spp. 17 species	Short bare-tailed opossum	Colicorto	Catita, juptí	Panama, Brazil, Peru, Bolivia, Paraguay	
Philander spp.	Four-eyed opossum	cayopollín	Mucurachichica	Central America to northern Argentina	Up to 800 g
Caenolestes spp. 5 species	Shrew opossum	Ratón comadreja, ratón runcho	Ratões-gambás	Ecuador, western Colombia, Venezuela	♂ 25–41 g ♀ 17–25 g
Lesteros inca	Incan shrew opossum	Ratón uncho andino		Andean zone of southern Peru	26–31 g
Rhyncholestres raphanurus	Chilean shrew opossum	El rincolesta, Ratón runcho coligrueso		Chile	21 g

Thermoregulation

The body temperature of SAMs is lower than that of placental mammals. The mean body temperature of the Virginia opossum is 35.2°C (95.4°F). The body temperature of an individual opossum may vary from 17 to 37.4°C (62.6–99.3°F), depending on the ambient temperature, state of excitement, activity, and wakefulness.[7] Thermoregulation is controlled by avoidance behavior (nocturnal), evaporative cooling (salivating on forearms), vasoregulation, shivering, piloerection, and torpor.[7] Variable body temperature should be considered during physical examination when evaluating potential diseases with fever as a clinical sign.

Studies on Free-Ranging Populations

Few field studies have been conducted on SAMs. Most information on biology and disease susceptibility has been obtained from studies on the species maintained as laboratory animals.[8] SAMs are not endangered, so little attention has been paid to conserving these species.

MANAGEMENT OF ANIMALS MAINTAINED IN CAPTIVITY

Housing

The size of the enclosure is determined by the size of the species.[13] A 40 L (10 gal) aquarium tank suffices for *Marmosa* and *Monodelphis* species. Most species are arboreal, thus a climbing network is desirable. Outdoor enclosures may be glass or fine mesh wire. If the ambient temperatures drop below 0°C (32°F), a heated indoor enclosure should be provided. Tropical forms should be maintained indoors during cold weather. *Didelphis* is an exception, because they are quite hardy.

Humidity should be maintained at a minimum of 50% year round. The water opossums (*Chieronectes* and *Lutreolina*) require pools with constantly circulating water. All species should be provided with a nest box for each individual and with access to nesting material.

Feeding

All species are omnivorous or carnivorous and may be maintained on commercial complete meat mixes or omnivore or carnivore soft diets. Many species are partly insectivorous, so meal worms and crickets are usually added to the captive diet.[6,13]

Restraint

The North American opossum *D. virginianum* and its South American cousin *Didelphis marsupialis* will display an open mouth with a formidable array of 50 small sharp teeth when cornered or threatened. They may be netted, or a snare may be placed around the neck. As soon as the animal is snared, the tail should be grasped and held with the pole to avoid the animal twirling or thrashing in the snare. The latter species have prehensile tails; they may climb their own tail and bite an unwary handler, if the head is not restrained. These species may feign death by going into an apparent hypnotic state (playing dead or playing possum) if threatened by a predator.[5]

Most American opossums are small- to medium-sized animals that may be captured easily with a net and then grasped with a lightly gloved hand to avoid scratching from the toenails. Small rodentlike species should be handled in much the same way as wild rats. Gloves, towels, tubes, and other special tools may be improvised for handling.[5]

Chemical restraint may be accomplished using ketamine hydrochloride at 15-30 mg/kg intramuscularly, or tiletamine/zolazepam (Telazol, Zoletil) 5-10 mg/kg. The higher dose of Telazol provides good relaxation, but prolonged recovery should be expected.[5]

Anesthesia

The most suitable anesthesia is isoflurane administered by a cone attached to a precision vaporizer or an anesthetic chamber.[5,9,12] This may be the safest and easiest method of conducting an examination or carrying out diagnostic procedures. An open drop method of anesthesia should not be used because the vapor pressure of isoflurane and halothane may allow a concentration buildup to 25%. Two or three deep breaths at such a concentration may be lethal.

Surgery

Surgical procedures described for laboratory rodents are suitable for SAMs.

Clinical Examination

Anesthesia will facilitate examination similar to that carried out on laboratory rodents.

Collecting Samples

The femoral vein is the vein of choice for venipuncture in most species when the operator has minimal experience with the species. Its location in the femoral triangle is the same as in all mammals. Some of the larger species have accessible brachial and saphenous veins. The jugular vein is also suitable if restraint is adequate.[8]

Urine is collected by cystocentesis, being cautious to avoid penetration of the pouch.

Special diagnostic procedures include radiography, ultrasonography, and endoscopy, which are performed as in laboratory rodents.

DISEASES

Diseases in Free-Ranging Populations[3,11]

Free-ranging SAMs are susceptible to rabies (but are not a reservoir host); arborvirus infection (may or may not be a reservoir); serologic response to yellow fever virus, and vesicular stomatitis infection; tularemia; salmonellosis; leptospirosis; and spotted fever. See Table 21.2.

The most important parasite of free-ranging didelphids is *Trypanasoma cruzi* (Chaga's disease, trypanosomiasis; see Table 21.2).[2] Otherwise, SAMs harbor an array of external and internal parasites that cause little, if any, disease in free-ranging populations.

Infectious Diseases

South American marsupials are relatively free of infectious diseases except for ubiquitous opportunistic bacteria

TABLE 21.2. Selected infectious and parasitic diseases of South American marsupials

Name (English)	Agent	Name (Portuguese)	Name (Spanish)	Epizootiology	Signs of Disease	Diagnosis	Management
Rabies	Lyssavirus Rhabdoviridae	Raiva	Rabia	Bites from bat or carnivore reservoir hosts	Encephalitis, similar to all mammals	Necropsy, monoclonal antibody panel on serum.	No treatment. Prognosis unfavorable.
Tularemia	*Francisella tularensis*	Tularemia	Tularemia	Acquired from scavenging reservoir carcasses. Also by ingesting contaminated feed.	Septicemia	Necrosis in liver, spleen and lymph nodes	Prognosis is unfavorable. Sanitation and antibiotics
Bacterial endocarditis	β hemolytic streptococci	Endocaridose	Endocarditis	Unknown	Sudden collapse. Emboli may lodge in various organs or the limbs	Blood culture, necropsy	Sanitation, antibiotics
Leptospirosis	*Leptospira* spp.	Leptospirose	Leptospirosis	Infected by ingesting contaminated feed and water. Didelphids may serve as a reservoir host.	Hematuria, kidney failure, septicemia	Urine and blood culture, necropsy	Vaccination with appropriate serovar bacterin. Rodent control
Spotted fever	*Rickettsia rickettsii*	Ricketsiose	Ricketsiosis	Ticks are responsible for transmission. Didelphids may serve as a natural host	No reported clinical disease	Culture	Eliminate external parasites
Salmonellosis	*Salmonella* spp.	Salmonelose	Salmonelosis	Ingestion of contaminated feed and water. Direct contact	Enteritis, septicemia	Clinical signs, culture	Aggressive fluid therapy. Antibiotics based on sensitivity.
Toxoplasmosis	*Toxoplasma gondii*	Toxoplasmose	Toxoplasmosis	Scavenging hosts containing cysts.	Various organ systems may be involved. There may be no clinical disease	Necropsy	Therapy not used. Prognosis guarded
Trypanosomiasis Chaga's disease	*Trypanosoma cruzi*	Trypanosmose	Trypanosomiasis	Reduviid (triatome) insects are the vector. Some species of didelphids are natural hosts and may be involved in spreading infection to people.	No naturally occurring disease has been reported.	Xenodiagnosis: Blood of suspect is fed to triatomes and their feces examined for trypanosomes	
Cutaneous leishmaniasis	*Leishmania brazilienses*	Leishmanose	Leishmaniasis	The main vectors are flies, *Lutzomyia* spp.	Didelphids are natural hosts, but may develop ulcers at the base of the tail	Biopsy of dermal lesion	Fly control
Coccidiosis	*Eimeria* spp.	Coccidiose	Coccidiosis	Direct life cycle. Ingestion of contaminated feed	Diarrhea	Oocysts in fecal flotation	Sanitation, coccidiostats

common to other mammals.[3] The question is often asked whether or not they are susceptible to canine or feline viral infections. Because they are far removed from the order Carnivora, they are not susceptible to any of those diseases. They may become infected with the rabies virus, but do not constitute a reservoir for rabies. See Table 21.2 for some selected infectious diseases.

Parasitic Diseases

South American marsupials have a full complement of external and internal parasites, but clinical parasitism is rare in captive animals.[2,8,14]

Noninfectious Diseases

The most common condition in captive didelphids is tail ulceration (ring tail).[7] The etiology is a combination of low humidity, suboptimal ambient temperatures, and poor peripheral circulation. Once ulceration occurs, it is a problem to cure because self-mutilation becomes a constant challenge. Obviously environmental conditions must be corrected.

Dental and gingival problems may arise because of the soft diet provided. Plaque and tarter buildup may predispose the animals to gingivitis.

Metabolic bone disease (rickets) may be observed in juvenile SAMs if fed fruits and vegetables along with meat, but no calcium supplementation. Commercial carnivore and omnivore diets prevent this problem.

SAMs are subject to traumatic episodes as are all other animals.

REPRODUCTION IN CAPTIVITY[1,7,8]

Male and female South American marsupials should be housed separately, except during the breeding season. Larger didelphids may engage in active precopulatory chasing, so space should be provided if breeding is anticipated. Males do not participate in rearing of young, so males should be removed after breeding. Free-ranging male offspring are weaned and disperse earlier than females. Extra nest boxes should be provided for captives.

Reproductive anatomy and physiology is unique in marsupials.[1,4,7,8] Marsupials have a common opening for the urogenital system and the digestive system called the urogenital sinus. Proceeding cranially, a urogenital canal opens into two lateral vaginae. A central false vagina supports the lateral vagina which may dilate and arc laterally, returning to the midline before entering into a corresponding short uterus. The oviducts extend to the infundibulum near the ovary.

The reproductive cycle is as follows: Ova are released from the ovary and are collected by the infundibulum, hence into the oviduct where fertilization takes place. Embryonic development continues in the uterus. Marsupials do not have the chorioallantoic placenta characteristic of eutherian mammals, but rather a vitelline(yolk) allantoic placenta. Once a certain stage of development is reached, the embryo enters a diapause state and may remain so for a number of months, until the female weans the previous litter or environmental conditions become conducive to beginning another litter.

In many species of mammals the fetus is protected, sometimes at the expense of the mother. In marsupials, any threat to the survival of the female (lack of food, illness, stress) may be followed by discarding the pouched offspring. However, an evolutionary adaptation to cope with a possible loss of a season's offspring is to have embryos waiting in diapause, which can be born as soon as the stress period has changed.

Most SAMs, except *Marmosa* spp. and *Lutreolina* spp. have a marsupium or pouch[1] that provides a protected environment for the continued development of the embryos. At birth, the embryos of *D. marsupialis* may weigh as little as 0.13 g and be 10 mm in length. The embryo must crawl to the pouch by grasping hairs with special deciduous claws on its forelimbs. Once inside the pouch, it must find an unoccupied nipple and attach to it by grasping with the mouth. The mouth swells shut around the dilated tip of the nipple. If the embryo is physically pulled from the nipple it is impossible for it to be reattached and the embryo dies. Seven to nine embryos gain attachment. The embryo remains attached for 55-60 days, but continues to nurse after release from the nipple until weaning at approximately 100 days. This cycle varies for other species.

Neonatology[6-8]

Little is known about artificial rearing of SAMs, except for the Virginia opossum. It is known that marsupial milk composition changes with the stage of lactation. Toward the end of the nursing period, marsupial milk is higher in total solids, fats, and proteins, but lower in carbohydrates than eutherian mammal milk (Table 21.3). Galactose is

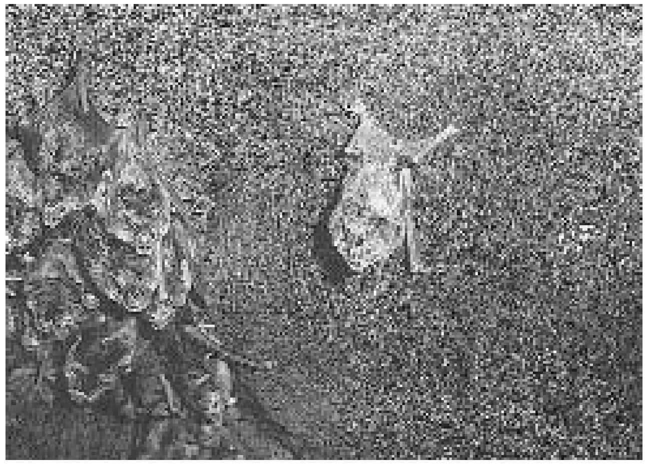
TABLE 21.3. Milk composition of selected mammals

Species	Water	Total Solids	Protein	Lipid	Carbohydrate
Didelphis marsupialis	76.8	23.2	8.4	11.3	1.02
Cow	87.0	13	3.0–4.0	3.5–5.0	4.5–5.0
Human	87.4	12.6	1.0–1.5	3.0–4.0	7.0–7.5

the primary milk sugar of marsupials, whereas lactose is the primary milk sugar in eutherian mammal milk.[7]

REFERENCES

1. Barnes, R.D. 1977. The special anatomy of *Marmosa robinsoni*. In D. Hunsaker, III, ed., The Biology of Marsupials. New York, Academic Press, pp. 387–389.
2. Beveridge, I. 1986. Parasitic diseases of marsupials. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 577–587.
3. Butler, R. 1986. Bacterial diseases of marsupials. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 572–576.
4. Finnie, E.P. 1986. Introduction to marsupials. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 558–566.
5. Fowler, M.E. 1995. Restraint and Handling of Wild and Domestic Animals, 2nd Ed. Ames, Iowa, Iowa State University Press, pp. 199–205.
6. Hume, I.D. 1986. Nutrition and feeding of monotremes and marsupials. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 566–570.
7. Hunsaker, D., III. 1977. Ecology of new world marsupials. In D. Hunsaker, III, ed., The Biology of Marsupials. New York, Academic Press, pp. 95–156.
8. Jurgelski, W. 1987. American marsupials. In T.B. Poole, ed., The UFAW Handbook on the Care and Management of Laboratory Animals, 6th Ed. New York, Churchill Livingstone, pp. 189–206.
9. Kohn, D.F.; Wixson, S.K.; White, W.J.; and Benson, G.J. Eds. 1997. Anesthesia and Analgesia in Laboratory Animals. San Diego, Academic Press, pp. 339–340.
10. Nowak, R.M.; and Paradiso, J.L. 1983. Walker's Mammals of the World, 4th Ed. Baltimore, John Hopkins University Press, pp. 11–26.
11. Potakay, S. 1977. Diseases of marsupials. In D. Hunsaker, III, ed., The Biology of Marsupials. New York, Academic Press, pp. 415–498.
12. Shima, A. 1986. Sedation and anesthesia in marsupials, In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 4th Ed. Philadelphia, W.B. Saunders, pp. 333–336.
13. Shoemaker, A.H. Ed. 1997. American Association of Zoos and Aquaria, Minimum Husbandry Guidelines for Mammals. Bethesda, Maryland, American Zoo and Aquarium Association.
14. Tyndale-Biscoe, H. 1973. Life of Marsupials. London, Edward Arnold, pp. 37–98, 189–209.



22
Order Chiroptera (Bats)

Carlos Esbérard
Luciana Hardt Gomes

BIOLOGY AND CAPTIVE MANAGEMENT

Carlos Esbérard

INTRODUCTION

Bats are classified in two suborders, the Megachiroptera and the Microchiroptera. All species of the suborder Megachiroptera belong to a single family (Pteropodidae) restricted to the Old World. The suborder Microchiroptera comprises 16 families. The Thyropteridae, Natalidae, Furipteridae, Mormoopidae, Phyllostomidae, and Noctilionidae families are restricted to the Americas, whereas the Emballonuridae and Molossidae families are distributed worldwide.[7] Table 22.1 discusses the species of South American bats suitable for captivity.

BIOLOGY

In Brazil, more than 140 species of bats, distributed in eight families, are known.[1] Of these families, only the Phyllostomidae have diverse feeding patterns differing from the insectivorous, with species presenting frugivorous, nectarivorous, carnivorous, and hematophagous patterns. One Central and South American species consumes fish and insects (family Noctilinidae, *Noctilio leporinus*).[6]

Bats have not been commonly exhibited by South American zoos, and this may be one of the reasons why only fragmented knowledge about the habits of the great majority of the South American species is available. Nevertheless, bats are brought to zoos by people who find injured or ill bats, orphaned nestlings, or individuals from destroyed colonies inside or near their homes.[4,5] Frugivorous species (Phyllostomid) are most likely to be successfully maintained in captivity, followed by nectarivorous, carnivorous, and hematophagous species.[8]

Several South American species live in large colonies, sometimes exceeding 100 individuals of all ages.[9] An ideal exhibition must take this into account. The maintenance of bats under conditions of reversed photoperiod is one of the best options, providing the opportunity for the bats to be seen by visitors in their peak level of activity. In reversed lighting, infrared, blue, or white lamps are used to simulate night lighting, and high-intensity common lamps simulate daylight.

Bat Physiology

The morphology of bats may provide clues about their feeding habits, because wing shape is correlated with flight patterns. Jaw properties may reveal dietary adaptations.

TABLE 22.1. South American species of bats suitable for captivity

Family	Species	Observations
Noctilionidae	*Noctilio leporinus* (Fisherman bat)	Gregarious, insectivorous, and piscivorous habits; roosts in hollows
Phyllostomidae	*Chrotopterus auritus* (Peter's woolly false vampire Bat)	Carnivorous species; roosts in caves in small groups
	Tonatia bidens (Round-eared bat)	Carnivorous and insectivorous habits; roosts in caves in small groups
	Phyllostomus hastatus (Spear-nosed bats)	Omnivorous habits; refuge in hollow trees, caves and houses; number of each group varies from 1–2 to more than 20.
	Artibeus sp. (Fruit bat)	Frugivorous habits; refuge in trees; groups composed by a harem of 8–12 females and young
	Sturnira lillium (Yellow-shouldered bat)	Frugivorous habits; refuge in hollow trees
	Glossophaga soricina (Long-tongued bat)	Nectarivorous species, refuge in caves, houses; groups composed of few animals, but larger groups can reach more than 50 adult animals
	Carollia perspicillata (Short-tailed bat)	Frugivorous habits; refuge in hollow trees; groups can reach more than 150 animals
	Desmodus rotundus (Vampire bats)	Hematophagus habits, prey on mammals and birds; roosts in caves and hollow trees; form harems of up to 10 females and young
Molossidae	*Molossus* sp. (Velvety free-tailed bat)	Insectivorous; refuge in rock crevices and roofs; groups present sexual segregation in part of the year; more than 50 animals can be seen in some roosts.
Vespertilionidae	*Myotis nigricans* (Little brown bat)	Insectivorous; refuge in rock crevices and roofs; the groups generally do not reach more than 30 animals.

The basal metabolic rates of various bat species may be classified into two groups. Vespertilionidae and Molossidae species have a low basal metabolism rate, whereas Phyllostomidae bats have a high basal metabolism rate. The lower rate may be viewed as an energy-conserving adaptation, permitting the maintenance of lower body temperatures during the roosting periods. Phyllostomid bats maintain a constant body temperature, although some smaller species are unable to maintain constant body temperature in a low-temperature environment.[3] Body temperatures of bats were measured in the wild, where it was noted that it varies during the night and within species with different feeding habits. Starving animals had lower body temperatures than those in good condition. The body temperature of captive molossid bats increased 1–2 minutes after ingesting food.

MANAGEMENT IN CAPTIVITY

Handling

All people handling bats should receive preventive antirabies vaccination. All bats bite when handled. Larger species, such as fruit bats (*Artibeus* sp.), spear-nosed bats (*Phyllostomus hastatus*), or vampire bats (*Desmodus rotundus*) can produce serious wounds. Leather gloves are an indispensable piece of equipment.

In large enclosures, where bats are able to fly, the capture of an individual may be made easier by using insect nets. Shelters made of wood appear as refuges to some species (mainly carnivorous and omnivorous species), facilitating the restraining of individuals during their daylight retreat.

Cotton sacks are used for containing bats for several hours. The ideal size of these sacks varies with the average size of the species, 19 cm (7.5 in.) deep for smaller species (primarily nectarivorous) to 30 cm (12 in.) deep for carnivorous bats. For all species, only one individual should be kept in each sack. Several sacks may be suspended inside cardboard or wooden boxes for transport, which provides adequate protection for the captured animals as long as there is adequate ventilation. Bats suffer in low temperatures, and exposure to an environment below 22°C (72° F) should be avoided.

Bats may also be restrained using cotton sacks. The bat may be grasped through the sack, with its wings folded. The sack is then opened, exposing the whole animal for administration of medicine or collection of samples.

Husbandry

Bats require large enclosures. Smaller species may fly in cages 1 m (3.5 ft) long, but larger species have difficulty moving about in enclosures less than 3 m (10 ft) long. It is important that enclosures provide shelters in which the bats may hang by the hindlimb claws. Animals forced to crowd into inadequate shelters (small in size

TABLE 22.2. Diets for bats used at Rio Zoo Foundation

Species	Diet or Items Offered (average quantity offered per day)	Observations
Noctilio leporinus	Fish or canned dog food supplemented with insect flour—20% of the live weight of the species	Sometimes we use shrimps. On Sundays we do not offer food to avoid obesity
Carollia perspicillata, Sturnira lilium, Artibeus lituratus, Artibeus fimbriatus, Rhinophyla pumillo	Chopped fruits (bananas, apples, avocados, tomatoes, pineapples, oranges, etc.)—50–100% of the live weight of the species	Supplemented once a week with insect flour, yolk of boiled eggs, honey, Meritene, or canned dog food.
Phyllostomus hastatus	Chopped fruits (same as above)—45% of the live weight of the species	Supplemented daily with newly killed mice or chicks, minced meat, insect flour, or yolk of boiled egg, and weekly with honey, Meritene, or canned dog food
Glossophaga soricina	Chopped fruits (mainly papaya) blended with egg yolk, Meritene, insect flour and honey—35% of the species live weight	Insect flour content was increased every time we noted loss of fur
Tonatia bidens	Killed mice or canned dog food with insect flour—30% of the live weight of the species	On Sundays we do not offer food
Chrotopterus auritus	Killed mice or chicks	On Sundays we do not offer food. If loss of fur is noted we add insect flour within the abdominal cavity of mice.
Molossus molossus, Myotis nigricans	Larvae of *Tenebrio molitor,* cockroaches and frozen insects (beetles and butterflys)—20% of the live weight of the species	
Desmodus rotundus	Defibrinated blood—60% of the live weight of the species	Substitute live chickens and rabbits

and in number) suffer wounds and loss of fur produced by close contact with other individuals and their claws. In each enclosure, there should be three or four appropriate places from which the animals may hang. In spite of their social behavior, individuals of several species use isolated locations to feed; young-carrying females assemble in small numbers or tend to isolate themselves during part of the night.

Dry leaves or wooden scrapes are routinely placed in concrete-floored enclosures to decrease the risk of possible wounds when the animals land to pick up pieces of fruit. For large groups, it is necessary to place several food trays in separate locations throughout the enclosure to facilitate access to food and adequate feeding by all the bats; this also avoids the risk of possible collisions. Wire or screened cages are used in the Rio Zoo for lodging of 12 different species of bats.

Cannibalism has been seen among males of *Tonatia bidens,* as has frequent fighting among frugivorous bats. The most suitable social group is a harem. For *Artibeus lituratus* and *Artibeus fimbriatus,* 1 or 2 males with 10–15 females and their young may be placed in a 25 m^2 (82 ft^2) enclosure. Bachelor male groups of *Phyllostomus hastatus* have been formed without observing any fights. Better adaptation is obtained when newly acquired bats are housed in pairs or in small groups. *D. rotundus* consumes more food when housed in groups than when kept isolated.

For molossid bats, heated cages are used to provide daily ambient temperatures in the range of 38°C (100.4°F). At night the heater should be turned off. Under these conditions, bats have longer survival rates and higher consumption of food.

For permanent marking, most researchers use forearm rings. To observe individuals, several methods may be used, including punch marking and plastic bracelets or collars with colored plastic bands.

Feeding

Inasmuch as some bats capture their prey or pick fruit during flight, recently captured animals may have great difficulty when housed in small cages. In the first days after capture, food may be presented with tongs or placed on the cage floor. It is common for recently captured animals to die the very first night unless they are fed.

Water should always be available, but some species, such as the vampire bats, do not drink under normal conditions. Recently netted bats should be given a saline dextrose solution (1 mL/10 g) two to three times per day, by syringe, until they accept food. On some occasions, fur loss was noted in *N. leporinus* and *T. bidens,* species that use insects as part of their diet. The addition of insect flour to the diet eliminated this loss of fur.

Table 22.2 presents diets used at the Rio Zoo Foundation.

TABLE 22.3. Hematologic values for *Artibeus* and *Desmodus* species of bats analyzed

	A. lituratus	*A. fimbriatus*	*A. obscurus*	*A. jamaicensis*	*D. rotundus*
RBC	4.77–12.30 (9.22)	3.54–12.62 (7.33)	3.25–10.40 (7.33)	6.53–9.35 (7.94)	3.76–12.58 (9.18)
Hb	7.30–23.00 (16.42)	7–25.60 (13.18)	4.20–18.00 (13.12)	10.60–15.20 (12.90)	9.00–27.70 (15.45)
Ht	13.04–62.00 (49.15)	21.00–48.00 (34.59)	12.00–39.00 (30.75)	26.00–44.00 (35.00)	24.00–50.00 (39.47)
MCV	15.40–62.60 (48.44)	34.70–118.00 (52.59)	35.50–62.60 (42.97)	39.80–47.00 (43.40)	29.60–70.90 (45.57)
MCH	15.2–20.3 (17.62)	12.7–39.5 (18.84)	12.9–20.9 (17.32)	16.20 (16.20)	9.90–23.80 (17.33)
MCHC	24.3–57.2 (37.28)	23.3–54.6 (37.42)	33.3–49.4 (41.57)	36.8–40.7 (38.75)	32.0–55.4 (38.62)
WC	3.20–9.90 (5.91)	2.50–18.80 (8.70)	2.70–10.10 (5.35)	3.80–13.30 (8.55)	1.30–26.80 (12.06)

Source: See reference 2.
RBC, red blood cells; Hb, hemoglobin; Ht, hematocrit; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; WC, white cells.

Diseases and Causes of Mortality

Mortality in recently netted animals (0–3 days after capture), is caused primarily by the stress of capture and starvation. Animals adapted to confinement may develop *Salmonella* infection or sustain wounds from fighting. Ectoparasites are common. Infant animals of groups maintained in small cages have been found dead as the result of several bites; injuries are less common in groups maintained in cages with more appropriate dimensions or that provide areas for nursing females to remain isolated from the rest of the group.

Breeding

Most Neotropical species are seasonal breeders, with one to two births per female per year. For a review of the seasonality of phyllostomid bats, see Wilson.[10]

Desmodus seems to be a seasonal breeder, and other phyllostomid species are polyestrous seasonal breeders, ensuring that young are independent at the most suitable season, generally in the months with a high number of rainy days.

Euthanasia and Anesthesia

Halothane, nitrous oxide, and oxygen or isoflurane have been used to induce anesthesia in vampire bats. The rapid sedation necessary for the collection of vaginal smears may be achieved by a mixture of xylazine and ketamine (1:2), at a dosage of 25 mg/kg, intraperitoneally. Pentobarbitone is effective for euthanasia of fatally wounded animals, such as those with a broken forearm.

Hand-Rearing

It is common for zoos to receive orphaned nursing bats. For phyllostomid bats, evaporated milk (1:1 in water) or powdered baby formula (Nestogeno) may be fed. Each bat receives milk four times a day, each meal corresponding to 5% of the body weight. It is important to keep infant bats warmed to 36–40°C (96.8–104°F), for at least 2 hours before they are fed.

Hematologic Values

Blood samples were collected by cardiac puncture in animals used in terminal experiments.[2] The values are summarized in Table 22.3. Small amounts of blood may be withdrawn by venipuncture of the brachial or interfemoral vein.

REFERENCES

1. Aguiar, L.M.; and Taddei, V.A. (1995) Workshop sobre a conservação dos morcegos brasileiros. Chiroptera Neotropical 1(2):24–29.
2. Baptista, M.; and Esbérard, C.E.L. 1997. Valores hematológicos de *Artibeus* sp. e *Desmodus rotundus* (Mammalia, Chiroptera). Revista Cientifica do Instituto de Pesquisas Gonzaga Gama Filho 3(2):11–22.
3. Eisenberg, J.F. 1991. Mammalian Radiations—An Analysis of Trends in Evolution, Adaptation, and Behaviour. Chicago, University of Chicago Press.
4. Esbérard, C.E.L. 1995. Morcego: Uma vitima das superstições. Ciência Hoje 18(105):71–72.
5. Esbérard, C.E.L.; Chagas, A.S.; and Luz, E.M. 1999. Uso de residências por morcegos no estado do Rio de

Janeiro (Mammalia: Chiroptera). Revista Brasileira de Medicina Veterinária 21(1):17–20.
6. Gardner, A.L. 1977. Feeding habits. In R.J. Baker, J.K. Jones, Jr., and D.C. Carter, eds., Biology of Bats of the New World Family Phyllostomid, Pt. 2. Lubbock, Texas, pp. 293–350. [Special Publications of the Museum of Texas Technical University 13:1–316.]
7. Nowak, R.M. 1991. Walker's Mammals of the World, 3rd Ed., Vol. 1. Baltimore, Johns Hopkins University Press.
8. Rasweiler, J.J., IV. 1975. Maintaining and breeding neotropical frugivores, nectarivorous and pollenivorous bats. International Zoo Yearbook 15:18–30.
9. Tutle, M.D. 1976. Collecting techniques. In R.J. Baker, J.K. Jones, Jr., and D.C. Carter, eds., Biology of Bats of the New World Family Phyllostomid, Pt. 1. Lubbock, Texas, pp. 71–88. [Special Publication of the Museum of Texas Technical University 10:1–218.]
10. Wilson, D.E. 1979. Reproductive patterns. In R.J. Baker, J.K. Jones, Jr., D.C. Carter, eds., Biology of Bats of the New World Family Phyllostomid, Pt. 2. Lubbock, Texas, pp. 317–378. [Special Publication of the Museum of Texas Technical University 16:1–364.]

PUBLIC HEALTH

Luciana Hardt Gomes

Various etiological agents have been isolated from bat species of South America,[3] but more studies are needed concerning the role of these animals in the epidemiology of diseases and, consequently, about the public health risk. Histoplasmosis and rabies are the most important zoonoses transmitted by bats. Editions of *Zoo and Wild Animal Medicine*[4,5], contain chapters referring to the role of bats in the epidemiology of these diseases. This chapter emphasizes the Latin American situation and updates general information.

Histoplasmosis

Environments such as caverns, abandoned construction sites, poultry yards, and trees are generally associated with the accumulation of bat, chicken, and other gregarious bird feces. Such fecal accumulations, along with favorable conditions of temperature and humidity, foster the proliferation of a saprophytic fungus, *Histoplasma capsulatum.*

Domestic and wild mammals are susceptible to fungal infections, and act as accidental hosts. Some species of bats, particularly colonial species, develop subclinical infections, shedding fungus through feces and consequently becoming carriers, serving an active role in the agent's diffusion.[2]

Histoplasmosis begins as a respiratory infection as the result of spore inhalation. Most cases are subclinical. Clinical cases may be pulmonary or generalized. The occurrence of histoplasmosis in Brazil has been demonstrated by the observation of autochthonal clinical cases or by epidemiological surveys with a histoplasmin cutaneous test.[12]

Rabies

Rabies is caused by a *Lyssavirus,* family Rhabdoviridae, and is important in public health because of its near 100% mortality. Every species of bat is susceptible to rabies and may transmit the disease to humans, domestic animals, and other wild mammals.

The vampire bat, *D. rotundus,* is the primary vector for transmission of rabies to herbivores, inhibiting the development of cattle raising in certain areas of Latin America. The range of geographic distribution of vampire bats is from northern Mexico to Uruguay, northern Argentina, and central Chile. An estimated 1 million cattle deaths are caused by rabies annually.[4] Acha and Malaga, in the years 1983-84, estimated a loss of US $64,820,000.[1,2] In Brazil, a population of 42 million cattle are at risk for rabies.

In many countries of Latin America, the number of human rabies cases transmitted by bats is rising in recent years. Bats have become second only to the dog as a source of rabies infection in humans.

Between 1929 and 1996, 585 cases of human rabies were reported in 13 countries of Latin America as having been transmitted by bats, possibly vampire bats. Peru reported the largest number of cases (179), followed by Mexico (160), and Brazil (95).[8]

In the period from 1990 to 1996, nine Latin American countries reported cases of human rabies transmitted by bats; Peru reporting almost half of them, 49.4%; Brazil, 25.3%; and Mexico, 19.5% of the total.[8] In Brazil, from 1986 to 1998, bats were responsible for transmission of 10.5% of the number of cases of rabies, second only to dogs (Fundação Nacional de Saúde). According to the Health Department, of 29 cases of human rabies transmitted by bats in the period from 1992 to 1998, the northeast region of the country contributed 14 (48.3%), followed by the southeast region with 7 cases (24.1%), the north with 5 (17.3%), and the west central with 3 (10.3%). In the southern region no cases of human rabies were reported. The majority of reports do not supply information about the species of bat involved or the circumstances of occurrence.

According to Schneider et al.[9] many cases occur because of human penetration in the forests, developing small villages for mining, which include no cattle. People in these areas become a new source of food for vampire bats.[9]

In 1990, seven cases of human rabies transmitted by bats occurred in the Amazon Forest in the state of Mato Grosso in an area of gold mining with a population of

40 people, all of them attacked by vampire bats. Some residents were reported to suffer as many as 25 bites. Uieda suspected that in certain regions, young and single males of *D. rotundus,* expelled from their colonies, attacked humans as an alternative and temporary source of food.[11]

According to Schneider and collaborators, urban rabies cases that occurred in Brazil were transmitted by non-vampire bats.[9] In Brazil, of the 144 bat species recorded, 29 have been observed exploiting shelters in buildings used by people,[10] which raises the possibility of humans, dogs, and cats having contact with them, and favoring the transmission of rabies. Cats may kill bats in their roosts, another important link between rabies of bats and carnivores.

In 1973, São Paulo, the largest city in Brazil, developed a Zoonosis Control Center that was appointed by the World Health Organization (WHO) as a collaborating center of training and research in zoonosis and is working on the epidemiology of rabies in wild and domestic species. They have found that insectivore bats are becoming important as a reservoir of the rabies virus. Six cases have been recorded: two in *Nyctinomops macrotis,* two in *Tadarida brasiliensis,* one in *Lasiurus cinereus,* and one in *Myotis nigricans*. All of these bats were found in atypical situations, active at inappropriate times of the day or in unusual localities.

REFERENCES

1. Malaga Alba, A. 1988. Economic losses due to *Desmodus rotundus*. In A.M. Greenhall and U. Schmidt, eds., Natural History of Vampire Bats. Boca Raton, Florida, CRC Press, p. 246.
2. Acha, P.N.; and Szyfres, B. 1989. Zoonosis y Enfermedades Transmisibles Comunes al Hombre y a los Animales [Zoonoses and diseases transmissible between people and animals],. 2nd Ed. Washington, D.C. Organizacion Panamericana de la Salud, Publicacion Cientifica No. 503, p. 989.
3. Constantine, D.G. 1970. Bats in relation to the health, welfare and economy of man. In W.A. Wimsatt, ed., Biology of Bats, Vol. 2. New York, Academic Press, pp. 319–449.
4. Fowler, M.E. 1986. Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, Saunders, p. 1127.
5. Fowler, M.E. 1993. Zoo and Wild Animal Medicine: Current Therapy 3. 3rd Ed. Philadelphia, Saunders, p. 617.
6. Mitchell, G.C.; Burns, R.J.; Flores Crespo, R.; and Fernanez, S.S. 1973. El control del murcielago vampiro 1934–1971 [The control of vampire bats]. In Agernia para el Desaoviollo International (A.I.D.). Combate Químico de los Murcielagos Vampiros. Buenos Aires, Centro Regional de Ayuda Tecnica, p. 40.
7. Moreno, A.J.; and Baer, G.M. 1980. Experimental rabies in the vampire bat. American Journal of Tropical Medicine and Hygiene 29:254–259.
8. Peralta, E.A. 1997. Rabia transmitida por vampiros, distribucion, frecuencia e importancia [Rabies transmitted by vampire bats: distribution, frequency, importance]. Técnica Pecuavia en México 35(2):93–104.
9. Schneider, M.C.; and Santos-Burgoa, C. 1995. Algunas consideraciones sobre la rabia humana transmitida por murciélago [Some considerations about human rabies transmitted by vampire bats]. Salud Publica Mexico 37:354–362.
10. Taddei, V.A. 1996. Sistemática de quirópteros [Systematics of bats]. Boletim do Instituto Pasteur 1(2):17–36.
11. Uieda, W.; Hayashi, M.M.; Gomes, L.H.; and Silva, M.M.S. 1996. Espécies de quirópteros diagnosticadas com raiva no Brasil [Species of bats diagnosed with rabies in Brazil]. Boletim do Instituto Pasteur 1(2):17–36.
12. Zancope-Oliveira, R.M.; and Wanke, B. 1987. Distribuição das fontes de infecção do Histoplasma capsulatum var. capsulatum em Rio da Prata—Município do Rio de Janeiro (RJ) [Distribution of histoplasmosis in Rio da Prata]. Revista Instituto de Medicina Tropical de São Paulo 29:243–250.

23
Order Rodentia (Rodents)

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BIOLOGY AND MEDICINE

INTRODUCTION

The order Rodentia is the largest order of the class Mammalia and comprises approximately 1700 species, distributed in three suborders (Sciuromorpha, Myomorpha, and Hystricomorpha), more than 30 families, and more than 350 genera. Rodents are mammals of cosmopolitan geographical distribution. South America began to be settled by rodents in the Eocene epoch of the Tertiary period (Cenozoic era), and they have diversified enormously. The largest and more disparate of these animals are among the components of the Neotropical fauna.

The many species of this order are extremely variable, but basically they are characterized by the presence of large incisor teeth, two in the upper arcada and two in the lower. These incisor teeth grow continuously and are worn down constantly by counterabrasion, which maintains the teeth's permanently sharp edges. The diastema is a gap between the incisors and the premolars. A mobile temporomandibular joint allows wide lateral movement of the jaws.

The order Rodentia represents more than 40% of the mammal species of South America,[9] but few rodents are maintained in Brazilian zoos. Even so, certain rodents are important enough to justify individual medical care,[53] and many of them belong to endangered species. Capybaras, pacas, agoutis, acouchis, nutrias, and porcupines may be exhibited in zoos and have a long life expectancy under appropriate management. Many rodent species have been bred commercially as laboratory animals.

There are excellent general texts on medicine and handling of captive wild rodents,[11,55] as well as on the basic biology of the Neotropical rodents.[8,9,10,12,13,50] The objective of this chapter is to report supplemental information resulting from observations and studies conducted in Brazilian institutions.

BIOLOGY

Some South American rodent species kept in zoos and city parks have been studied intensively because they are attractive to the public. Studies of captive rodents have obtained physiologic and biomedical data. Capybaras, pacas, agoutis, acouchis, nutrias, and porcupines are among the well-known rodents.

Agoutis (*Dasyprocta* spp., Family Dasyproctidae)

Biological data about agoutis (*Dasyprocta azarae*) were obtained in studies performed between 1993 and 1996 at a scientific breeding institution located in the city of Curitiba (Paraná, Brazil).[19] During that time, 159 young were born in 77 litters from 19 females. The annual litter average per female was 1.74. The mean number of young per litter was 2.09 ± 0.69, most of the births being twinned, but some litters had three or four. The neonate mean weight was 193.20 g and the male/female proportion was 1:1. The mean interval between births was 179 ± 93 days, and the gestation period was 103 days. The females presented estrous cycles of 34 days continually during the year, but births were concentrated in August and September (winter and spring). The estrous cycle of *Dasyprocta primnolopha* is a little shorter, with a duration of 31 days. Sexual maturity was reached in 325 ± 102 days for female, and in 266 ± 14.85 days for males. The mortality rate was 28.93%; 19.5% due to infanticide, with evidence of cannibalism.

In a recent paper, focal registers were used to evaluate the alimentary behavior of two groups of *D. azarae* and verify whether bird and egg predation is a learned or innate behavior in this species. One group was formed exclusively of individuals kept in captivity and the other only of free-ranging individuals. Eggs and live chicks from *Gallus gallus* and *Cothurnix japonica* were offered. In both groups immediate predation was noticed when eggs and chicks were offered. The results indicate that zoophagy revealed by *D. azarae* is innate, since all captive animals were from the second or third generation born in captivity and had never received eggs or birds as a part of their diets. The conclusion was that in the wild *D. azarae* may prey upon birds, eggs, and nests.[29]

Acouchis (*Myoprocta* spp., Family Dasyproctidae)

The acouchis belong to the family Dasyproctidae, together with the agoutis. They are smaller, however, reaching a maximum length of 38 cm and a maximum weight of 1.3 kg. The hair coat is yellowish to orange. Sexual maturity is reached between 8 and 12 months. Gestation is 99 days, after which one or two young are born. The lactation period is 2–3 months.[11,55]

Pacas (*Agouti paca*, Family Agoutidae)

Pacas are large Neotropical rodents, reaching 80 cm in length and 12 kg in weight. They have four digits on the thoracic limbs and five on the pelvic limbs. The hair is short and bristled, varying in color from brown to red, with interpolated longitudinal white strips or spots. They have a digestive physiological adaptation represented by a highly functional cecum that facilitates the digestion of vegetable matter rich in cellulose. Pacas are nocturnal. During the day they stay in a burrow dug among roots of trees, under stones, or in sandbanks. They are excellent swimmers and when alarmed or threatened seek shelter in ponds or rivers. Sexual activity begins at the age of 1 year for both males and females.[34] Like the agoutis, pacas are continuously polyestric, with estrous cycles of 31–33 days.[34] Gestation averages 150 days and the interval between births varies from 180 to 250 days.[34] Twinned births are less frequent than in agoutis. Neonates weigh an average of 600 g and suckle for up to 90 days.[34] Births occur throughout the year, except February to March (end of the summer) and August (end of the winter).[34]

Capybaras (*Hydrochaeris hydrochaeris*, Family Hydrochaeridae)

The capybara (*Hydrochaeris hydrochaeris*) is the largest living rodent. Body length of an adult capybara commonly ranges from 1 to 1.5 m (3.3–5.0 ft), and the average body weight is usually around 50 kg (110 lbs). Size and weight increase with the latitude, and in some Brazilian and Uruguayan regions this animal can reach up to 90 kg (198 lbs). It is a semiaquatic species, capable of watching its surroundings while keeping itself almost entirely submerged. There are four digits on the forelegs and three on the rear legs, and the digits are connected by skin forming natatorial membranes.

There is no obvious sexual dimorphism except for a sebaceous gland on the upper head, between the eyes and the nose, appearing to be a dark lump. This "nose gland" produces a sticky white secretion used to mark the group territory. All capybaras have the gland, but it is more visible in males after their first year of age, becoming highly developed in dominant adults. Some dominant females also have a prominent nose gland.

The capybara is a gregarious species; the size and stability of the social group varying seasonally.[16,49] A typical family group is constituted of three to four males and six females. One male leads the group, marks the territory, and exercises dominance.

The number of young varies according to the birth season. Old, sick, or aggressive animals are generally expelled from the families. The group structure is formed of a central block of females with their offspring and a peripheral guard of males, protecting the group. The dominant male is attentive to any transgression of the territory by an individual that does not belong to his family group. An interloper is aggressively repelled or even killed. Subordinate males frequently move to other groups.

The young are nursed by all the lactating females. Besides suckling, they eat solid food from birth onward, depending upon mother's milk for only 5 weeks. The family provides protection from predators. Young males and females generally reach puberty in the first year of life and form new group units, very few remaining with the original family group.[16]

The territory of a group comprises zones for resting, defecating, bathing, and grazing. To mark the territory, the dominant males rub their nose gland against vegetation, and all the members of the group drag the ventral part of their bodies on vegetation, pressing genitalia against it and urinating on it.[22] Frequently, territories lack well-defined limits, leading to true battles among the groups. Absence of vegetation and pasture and lack of water lead capybaras to migrate to other areas.

Grazing and resting activities generally take place in the morning, from 7 A.M. to 9 A.M., and in the evening from dusk to 10 P.M. Capybaras may also graze during the early dawn. In the warmer hours of the day, until about 3 P.M., they bathe, swim, play, and rest in the water. This pattern may be modified according to the season and hunting pressure.

Mastication reduces the forage to fine particles, contributing to a high digestive efficiency. The stomach of the capybara is simple, and the intestines are similar to those of other mammals, except for a well-developed cecum (representing 37% of the total weight of the digestive tract and containing 74% of the ingested food). This is the higher relative capacity for this organ.[15] Active fermentation takes place in the cecum, similar to what occurs in ruminants, with production of volatile fatty acids. With respect to nitrogen metabolism, there is evidence that this species has coprophagous behavior, and it is possible that capybaras have a mechanism for colic-cecal reflux, similar to that seen in other rodents.[15,17]

The diet of the capybara varies with the season and the rainfall, being composed of grass (75%) and other forages rich in proteins. In zoos the diets are varied and are usually composed of high-quality grass plus a balanced concentrated supplement.[16]

Female genitalia and anus are located within a pouch formed by a cutaneous fold. There are six pairs of mammae distributed from the pectoral to the inguinal area. The female reaches sexual maturity at the age of 10–12 months, when its body weight is between 15 and 20 kg (33–44 lbs). Females have spontaneous ovulation and are continuously polyestric. The estrous cycle is 7.5 ± 1.2 days and estrus lasts for 8 hours.[20]

The testicles are adhered to the abdomen, and males do not have a scrotum. Males reach sexual maturity at the age of 15–24 months, weighing 30–40 kg (66-88 lbs).[36] Copulation usually occurs in the water, both in nature and in captivity. Gestation is 5 months, and 1 to 8 young (average 4) are born. Neonates weigh from 1.3 to 2.2 kg (2.8–4.8 lbs) with an average of 1.75 kg (3.8 lbs).[16] Fifteen to 20 days after birth, while still nursing, the female enters estrus and may become pregnant. Thus, it is possible to have two births per year.

The exploitation of this species in South America is from both subsistence and commercial hunting. Attempts are being made to establish rules for the rational breeding of capybaras, under extensive, semi-intensive, and intensive systems. National laws forbid hunting in Brazil, Panama, Colombia, Paraguay, Uruguay, and in many provinces of Argentina. In Peru, Suriname, and some other provinces of Argentina, regulated hunting is allowed.[16,37] In Venezuela, because of the custom of consuming capybara meat during the Catholic Lenten period, an exploitation system developed that is supplied by systematic and regulated wide-scale hunting.[37] Brazilian law allows the slaughter of farmed capybaras weighing 20–40 kg (44–88 lbs), from 6 months to 1 year of age, only in registered abattoirs with regular sanitary inspection.

Nutrias (*Myocastor coypus*, Family Myocastoridae)

The nutria (*Myocastor coypus*) is a semiaquatic rodent varying in color from yellowish to reddish brown. Head and body length vary from 0.43 to 0.63 m (1.4-2.1 ft), and body weight varies from 7 to 9 kg (15–20 lbs). Sexual maturity is reached at 3–7 months, gestation and lactation have durations of 128–132 days and 6–10 weeks, respectively. A female may have two or three litters in 1 year, giving birth at any time of the year.[55] There are 5 to 6 (sometimes up to 12) young in each litter and the neonate's body weight is about 225 g (0.5 lbs). Growth is fast during the first 5 months.

Porcupines (*Sphiggurus* spp. and *Coendou* spp., Family Erethizontidae)

Porcupines (*Coendou* spp. and *Sphiggurus* spp.) are small to medium-sized nocturnal arboreal rodents. Their movements are slower than those of other rodents of the same size. The dorsal and lateral areas of the body are covered by yellowish cornified spines and long dark hair, giving them a yellowish-gray appearance. The spines are an efficient defense against predators.[50] Porcupines have a truly prehensile tail.[11] The body length of *Coendou* doesn't surpass 50–90 cm (20–35 in.), and its weight is 1.5–2.0 kg (3.3–4.4 lbs), whereas *Sphiggurus* has a body length of 36–38 cm (14–15 in.) and weighs 1.1–1.3 kg (2.4–2.9 lbs).[10] *Coendou* females reach sexual maturity around 19 months and usually mate immediately postpartum. The gestation period is about 203 days, with birth of one or two young. At birth the young weigh about 0.4 kg (0.88 lbs) and are able to climb. A female may be reproductively active for 11–12 years.

Supplemental biologic data for agoutis, acouchis, pacas, capybaras, and nutrias are presented in Table 23.1.

TABLE 23.1. Biological data for some South American rodents

Parameters	*Agouti paca*	*Dasyprocta azarae*	*Hydrochaeris hydrochaeris*	*Myocastor coypus*	*Myoprocta acouchi*
Food habits	Herbivore (vegetables, fruits)	Herbivore/ zoophagous	Herbivore (vegetables, aquatic plants)	Herbivore (plants, aquatic plants)	Herbivore (seeds, fruits)
Rectal temperature (°C)	38.36 ± 0.68[a]	36.38 ± 2.04[b]			36.20 ± 1.76[b]
Respiratory rate (mpm)	93.11 ± 17.73[a]	80.63 ± 29.09[b]			74.15 ± 30.23[b]
Heart rate (bpm)	199.56 ± 27.32[a]	150.93 ± 31.48[b]			196.42 ± 30.20[b]
Life span (years)	10–16	10–18	10–12	6–15	
Litters/year	1–2	1–2	1	2–3	1
Lactation	90 days	28–30 days	16 weeks	6–10 weeks	2–3 months
Number of nipples (pairs)	2	4	4–5	4–6	4–5
Dental formula (×2)	I1/1 C0/0 PM1/1 M3/3				
References	11, 34, 39, 55	19, 29, 39, 55	11, 55	11, 55	11, 55

[a] Data obtained under anesthesia with ketamine HCl, acetylpromazine maleate, and atropine sulfate (reference 39).

[b] Data obtained under anesthesia with ketamine HCl, xylazine HCl, and atropine sulfate (J.R. Pachaly & R.R. Lange, unpublished data).

mpm, movements per minute; bpm, beats per minute; I1/1, incisor teeth, one tooth in each hemiarcade; C0/0, canine teeth, no tooth in each hemiarcade; PM1/1, premolar teeth, one tooth in each hemiarcade; M3/3, molar teeth, three teeth in each hemiarcade.

RESTRAINT, ANESTHESIA, AND SURGERY

RESTRAINT AND HANDLING

Handling of several species of rodents may be difficult because of differing biological and behavioral characteristics. Even when adapted to captivity, these animals are susceptible to stress and react intensely, with risks of injuring themselves or the handlers. Thus, the restraint method should be carefully selected on the basis of knowledge of the reactions of each species, to ensure safety to both the animal and personnel.

Physical Restraint

Several methods of physical restraint of wild animals may be used for rodents. Capybaras are strong and may react aggressively to restraint. Nutrias, capybaras, and pacas seek shelter in the water and must be confined before capture.

Physical restraint of capybaras may be applied with snares or nets. Squeeze cages are also useful. In general, pacas, agoutis, nutrias, and young capybaras should be captured with a net attached to a wire loop and transferred to a cotton fabric bag or to a squeeze cage. Within the bag or cage, the animal can be weighed, examined, medicated, and/or anesthetized by intramuscular injection.

Porcupines are not aggressive, but their spines may inflict painful injuries. The best method to capture a South American porcupine is to catch the animal by its tail. After capture, a plastic tube with appropriate diameter may be placed in front of the animal, into which the animal is directed. The method is similar to that usually used for snakes. Once the anterior half of the body is within the tube, it is possible to give intramuscular injections and sex the animal. The handler should continue to hold the tail until the procedure is finished.

Chemical Restraint and Anesthesia

As in several other wild animal groups, the use of pharmacological methods to restrain rodents is often indispensable to the success of selected medical or management procedures. Although restraint is the most important limiting factor in wildlife practice, a complete review of anesthesia techniques for wild rodents does not exist.[46] Thus, many investigators working with rodents tolerate extremely long recovery periods and high anesthesia-related mortality rates.[46] For South American rodents, the anesthesia literature is even more scarce.[46] There are some reports of the effects of sedative and anesthetic drugs in capybaras,[53] nutrias,[7] pacas,[38,39,41,46] agoutis,[6,40,43,42] and chinchillas (*Chinchilla laniger*).[33]

The general recommendation for chemical restraint of South American rodents is the administration of a sedative with analgesic and muscle-relaxing properties and an agonist of α_2 adrenoceptors (such as xylazine hydrochloride , detomidine hydrochloride or medetomidine hydrochloride) combined with an anticholinergic agent (atropine sulfate) and a dissociative anesthetic. Ketamine hydrochloride is the classic “zoo anesthetic,” and its use in combination with the above-mentioned drugs produces good results.

Recent studies demonstrate that excellent surgical anesthesia for agoutis, pacas, porcupines, and capybaras is obtained when combining α_2 agonists, atropine, and tiletamine hydrochloride plus zolazepam (Zoletil or Telazol). Correct dosages are a challenge to zoo clinicians since rodents vary widely in size and weight. For this reason, allometric scaling is recommended as a safe and efficient method for calculating anesthetic, sedative, and neuroleptic doses.[42] (See Chapter 40, Allometric Scaling). The drugs should be mixed in a single dart or syringe and given by intramuscular route. The blowgun is a good choice for large animals such as the capybaras. For smaller animals, the drugs are usually injected with the animal under physical restraint.

SURGERY

Surgical procedures performed in rodents are usually limited to abscess drainage, amputations, fracture repair, and laceration suturing. In agoutis, preparatory hair clipping is not recommended. When the dorsal hair of several animals was clipped, severe sunburn with scaling and secondary self-mutilation occurred.

DIAGNOSIS

Available laboratory data about rodents are scarce[11] because of the difficulty in obtaining adequate samples of body fluids or tissues. Besides the small size of some animals, blood and urine collection generally demands chemical restraint, making frequent samplings unfeasible.

Clinical Pathology

HEMATOLOGY Blood samples from rodents may be obtained by venipuncture of the cephalic, saphenous, jugular, and femoral veins.[11] For laboratory animals the orbital venous plexus and cardiac puncture are often used.[11] It is possible to obtain blood drops for a smear by clipping the tip of a claw or tail.

Venipuncture in rodents of the family Dasyproctidae is usually difficult, especially when the clinician tries to

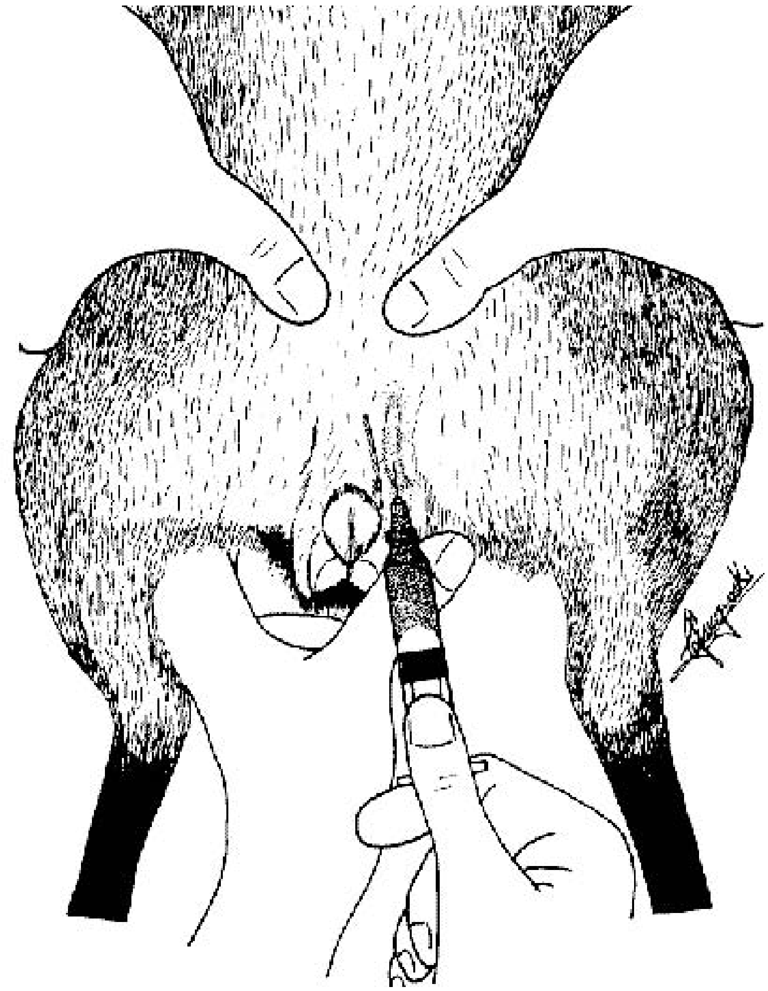


FIGURE 23.1. Diagram showing blood collection in an agouti from the right lateral saphenous vein. The site of venipuncture is the last segment of the vein's course, which is lateral to the median inguinal line. (Courtesy of J.R. Pachaly. Art: Thiago Cavalieri Luczinski, scientific illustrator.)

use the same veins used for domestic species. The jugular veins of these rodents have a small diameter, and the femoral and cephalic veins, besides being small, collapse easily.[44] To solve this problem, studies were conducted to evaluate a new site for venipuncture in Dasyproctidae rodents. Paired veins were found in the inguinal area on both sides and parallel to the genitalia.[44] If pressure is applied as shown in Figure 23.1, the vein expands greatly, allowing blood collection with the same needles used in dogs and cats, without collapsing the vein. The site was used successfully in 158 animals of the following groups: 119 agoutis (*Dasyprocta azarae*) of both sexes, weighing 2.12 ± 0.70 kg; 12 agoutis (*Dasyprocta aguti*) of both sexes, weighing 5.28 ± 0.72 kg; and 27 acouchis (*Myoprocta acouchi*) of both sexes, weighing 1.12 ± 0.19 kg. All the patients were chemically restrained with xylazine hydrochloride, ketamine HCl and atropine sulfate. The technique was used for blood sampling, allowing the collection of an average 0.90 mL of blood per animal.[44] It was also successful for intravenous injections.

Anatomical studies were made to identify that vein. In dissected animals "the lateral saphenous vein begins in the lateral aspect of the distal third of the leg, where it runs for a short distance, crosses the caudal aspect of the thigh over the popliteal region and reaches the medium aspect of the thigh, where it runs over the gracilis muscle in a medial-cranial direction closely to the median line which divides both inguinal regions and reaches the caudal epigastric vein".[23] Venipuncture is done in the last segment of its course, which is lateral to the median inguinal line. Observations made on those

TABLE 23.2. Mean hematological values for some South American rodents

Species	PVC (%)	Hgb (g/dL)	MCHC (%)	MCHC (g/dL)	RBC ($\times 10^6/\mu L$)	MCV (μm^3)	MCV (fl)	MCH (%)	MCH (pg)	WBC ($\times 10^6/\mu L$)
Dasyprocta azarae	42–54	13.6–16.5			5–6	80.1–93.7		29.7–33.2		2.6–9.1
Dasyprocta primnolopha (males)	42–59	11.5–20.4	27.4–34.6		4.7–6.4		89.4–92.2		24.4–31.9	3.0–11.3
Hydrochaeris hydrochaeris	33–37	10.2–11.1			3.1–3.2					6.1–7.9
Myocastor coypus	42–58	13.1–14.9			3.9–4.2					5.2–11.3
Myoprocta acouchi	33–49	10.3–18.0			5.3–8	45.3–77.8		27.4–46.5		3.2–7.9
Agouti paca	41.81 ± 2.47[a]	15.12 ± 1.37		36.96 ± 3.12	4.86 ± 0.65 [b]		87.8 ± 10.4		32.1 ± 3.49	9.81 ± 1.5 [c]

Species	Segmented Neutrophils (%)	Segmented Neutrophils ($\times 10^6/\mu L$)	Band Neutrophils (%)	Band Neutrophils ($\times 10^6/\mu L$)	Lymphocytes (%)	Lymphocytes ($\times 10^6/\mu L$)	Eosinophils (%)	Eosinophils ($\times 10^6/\mu L$)	Monocytes (%)	Monocytes ($\times 10^6/\mu L$)	Basophils (%)	Basophils ($\times 10^6/\mu L$)
Dasyprocta azarae	51–56		0–5		7–40		0–4		1–2		0	
Dasyprocta primnolopha (males)	25–82		0–5		19–36		2–8		1–4		0	
Hydrochaeris hydrochaeris	55–69				25–44				1–5		0–1	
Myocastor coypus	47–61				28–45		2–9		6–10		0	
Myoprocta acouchi	31–65		0–2		30–66		0–1		0–1		0	
Agouti paca		3.39 ± 0.77[d]				5.94 ± 0.93[e]		0.33 ± 0.13		0.16 ± 0.063		

Source: *Dasyprocta azarae*, reference 27; *Dasyprocta primnolopha*, reference 47; *Hydrochaeris hydrochaeris*, reference 11; *Myocastor coypus*, reference 11; *Myoprocta acouchi*, reference 28; *Agouti paca*, reference 34.

[a] Significant difference between males (42.84 ± 2.19%) and females (40.16 ± 1.84%) and adults (40.58 ± 2.39%) and young (43.66 ± 1.67%).

[b] Significant difference between females ($4.67 \pm 0.69 \times 10^6/\mu L$) and males ($5.06 \pm 0.61 \times 10^6/\mu L$).

[c] Significant difference between adults ($9.28 \pm 0.87 \times 10^6/\mu L$) and young ($10.86 \pm 2.15 \times 10^6/\mu L$).

[d] Significant difference between adults ($3.09 \pm 0.51 \times 10^6/\mu L$) and young ($3.82 \pm 0.96 \times 10^6/\mu L$).

[e] Significant difference between males ($6.10 \pm 0.77 \times 10^6/\mu L$) and females ($5.54 \pm 0.56 \times 10^6/\mu L$).

PVC, hematocrit; Hgb, hemoglobin; MCHC, mean corpuscular hemoglobin concentration, RBC, red blood cells; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; WBC, white blood cells.

animals indicate the lateral saphenous vein as ideal for blood collection in anesthetized agoutis and acouchis, as well as for intravenous drug administration.

Hematological values for agoutis, pacas, acouchis and capybaras are presented in Table 23.2.

BLOOD CHEMISTRY Blood glucose values in *Dasyprocta* sp. are 104.5 ± 33.4 mg/dL for females and 130 ± 13.89 mg/dL for males.[26,45] There is a report of bilateral cataract in an adult male *D. azarae,* caused by a hyperglycemic state (617.1 mg/dL) characteristic of diabetes mellitus.[31]

In captive agoutis serum levels of cortisol were determined.[2] Levels were higher in females (82.25 × 58.14 μg/dL), especially in younger ones, than in males (45.11 × 13.35 μg/dL). The highest level was measured in a pregnant female, near delivery (201.48 μg/dL).[2] In the same study, cortisol was measured by radioimmunoassay, and the hormone's circadian rhythm was not detected. Other serum biochemical values for pacas, agoutis, and acouchis are presented in Table 23.3.

URINALYSIS Urine may be collected easily from sedated or anesthetized animals, by vesical compression or urethral catheterization. In males, the exposed penis facilitates the procedure and avoids waste of urine. In agoutis, cystocentesis may be performed as it is in domestic cats. Acouchi urine is clearer than that of agoutis, but in both genera the color varies from light to golden yellow. The most commonly encountered substances are crystals of triple phosphate, calcium carbonate, amorphous phosphate, and amorphous urate, as well as spermatozoids, epithelial cells, hematic cylinders, leukocytes, and tyrosine crystals.[1] Other urinalysis physical and biochemical values for agoutis and acouchis are presented in Table 23.4.

Special Diagnostic Procedures

Some studies were done in agoutis (*Dasyprocta azarae*) to determine other physiological parameters. With an aplanatic tonometer it was determined that the intraocular pressure is higher in adults (18.20 ±(2.70 mm Hg) than in younger animals.[30] With a pulse oximeter placed in the left ear of agoutis it was determined that the partial oxygen pressure (SpO_2) reaches values of 84.25 ± 6.89% during the first 45 minutes of ketamine hydrochloride plus xylazine hydrochloride and atropine sulfate anesthesia.[54] Embryonic vesicles were observed and measured by ultrasonography in a female agouti in the first trimester of pregnancy.[5]

DISEASES

The primary medical problems encountered in captive South American rodents are related to trauma induced by incorrect handling and poor husbandry (capture, restraint, housing). Ectoparasites, endoparasites, and bacterial infections are also important.

Trauma

Being unable to adapt to the floor of enclosures is a common cause of medical problems in South American rodents, especially in species adapted to the soft soils of tropical forests. A group of Amazonian acouchis developed severe problems related to sand or concrete substrates just after arrival at Curitiba Zoo. They presented two distinct clinical syndromes, acute and chronic. In the acute cases, severe wearing of the claws and skin erosions in the footpads and metatarsal areas were observed, with hemorrhages and granulation tissue formation. Affected animals became anorexic, prostrate, and died within a few days. In chronic cases, hard, dry calluses formed in the skin of the metatarsal, tarsal and calcaneal areas, causing severe locomotion deficits, as well as inappetence, apathy, and weight loss. The condition was aggravated with fissure formation, secondary contamination and development of abscesses, as well as erosions, abrasions, and severe hemorrhages.

No results were obtained with tentative medical and surgical treatment. The problem was solved when the animals were housed in enclosures provided with a 5-cm layer of foliage. Once adapted to captivity, the recovered animals did not develop related problems, even when housed in enclosures with sand or concrete floors, without foliage coverage.

Similar problems were observed in pacas and agoutis. Erosions and ulcerations developed in the skin of the footpads and metatarsal areas. In agoutis, large, hard, horny calluses formed in the metatarsal and calcaneal areas. In several cases the callus formation seemed to be associated with the flooring and the animals' weight. A higher incidence was observed in *Dasyprocta leporina* (mean weight 7 kg) than in *D. azarae* (3–4 kg). The calluses tended to bulge, producing lateral expansions and making locomotion difficult. Surgical extirpation failed to alleviate the condition, because severe hemorrhage and failure of the borders of the wound to close ensued. Calluses with the same characteristics were also observed in older agoutis.

Pacas and agoutis suffered from loss of the claws as a result of trauma caused by hard flooring, especially when animals would try to excavate burrows. Exposition of phalanxes, necrosis, and subsequent loss of digits were seen in these cases. In captive or semicaptive agoutis, severe periungual inflammation with digit tumefaction and lameness was seen. In these cases digit loss was also common.

Some cases of paresis with loss of sensitivity and subsequent abrasive lesions caused by dragging the central digits on the floor were observed in the pelvic limbs of agoutis that had been injected with ketamine hydrochloride. All the animals recovered after 7–14 days, but it was necessary to protect the extremities with bandages. It is important to note that this problem occurred only after injection of ketamine hydrochloride or mixtures containing ketamine hydrochloride, and did not develop with any other drug.

Cutaneous lesions in acouchis and agoutis may be caused by fights between males, in which the aggressor jumps over the other male, biting and kicking its back

TABLE 23.3. Mean serum biochemical values for some South American rodents

Parameters	*Agouti paca*	*Dasyprocta primnolopha*	*Dasyprocta azarae*	*Myoprocta acouchi*
Uric Acid (mg/dL)		3.76 ± 0.71		
Glucose (mg/dL)	131.3 ± 15.15		± 33.4–female 130.0 ± 13.89–male	
Urea (mg/dL)	23.84 ± 6.79	42.6 ± 7.41		
Creatinine (mg/dL)		1.4 ± 0.27		
Total Lipids (g/dL)	212.6 ± 77.89			
Cholesterol (mg/dL)		134.88 ± 17.94 (males) 118.41 ± 14.58 (females)		
Triglycerides (mg/dL)		108.97 ± 21.54 (males) 105.36 ± 24.66 (females)		
Plasmatic Proteins (g/dL)	6.27 ± 0.45	7.43 ± 0.77	5.0–6.6	5.9–6.7
Albumin (g/dL)	2.85 ± 0.35	3.84 ± .032		
LDH (IU/L)			259 ± 153.34	
Sodium (mg/dL)	353.54 ± 55.16			
Potassium (mg/dL)	25.94 ± 5.77			
Calcium (mg/dL)	14.3 ± 2.56			
Magnesium (mg/dL)	3.02 ± 0.33			
Zinc (mg/dL)	0.34 ± 0.22			
Copper (mg/dL)	0.24 ± 0.6			
Iron (mg/dL)	0.52–0.24			
References	34	3, 14, 51, 52	2, 26, 27, 45	28

TABLE 23.4. Urinalysis values for agoutis (*Dasyprocta azarae*) and acouchis (*Myoprocta acouchi*)

Species	Glucose	Ascorbic Acid	Protein	Blood	pH	Nitrite	Urobilinogen	Bilirubin	Specific Gravity	Ketone	Reference
Dasyprocta azarae	Negative	Not tested	Negative or +	Negative	5.8–8.1	Negative	Negative	Negative	1018–1033	Negative	1
Myoprocta acouchi	Negative	Negative or +	Negative or +	Negative	5.0–6.5	Negative	Not tested	Not tested	1005–1027	Not tested	25

with the hind paws. This behavior causes dorsal cutaneous lacerations that suffer secondary contamination, resulting in abscess formation, fistulae, and necrosis, frequently climaxing with death. In pacas and agoutis, ear lacerations may be caused by fights among enclosure mates.

Traumatic dental fractures are common in rodents. Treatment is discussed in Chapter 38, The Oral Cavity.

Infectious Diseases

PARASITIC DISEASES Ectoparasites include fleas, ticks (*Amblyoma* spp.), lice, sarcoptic mange (*Sarcoptes scabei*), and trombiculidae mites. Therapy using fipronil (Frontline) topically has been effective. Ivermectin, administered intramuscularly, has been effective for sarcoptic mange.

Endoparasites include species of Ascaris, Ancylastoma, Trichuris, Capillaria, and Strongyloidia: *Paraspdodera uncinata* and *Longistriata brevispicula*. Protozoan parasites include *Ballantidium coli, Eimeria agouti, Babesia* spp., *Tryanosoma* spp., and *Leishmania* spp.

BACTERIAL DISEASES Bacterial infections include ubiquitous species of staphylococcus, streptococcus, corynebacteria, and pasteurella.

Many species of rodents are carriers of *Leptospira* spp. and may serve as sources of infection for other animals and humans. The clinical disease has not been reported in South American rodents. In capybaras there are reports of different rates of positive reaction to several serotypes of *Leptospira,* with predominance of *Leptospira canicola, Leptospira ballum, Leptospira hardjo, Leptospira hendomadis,* and *Leptospira wolffi. L. canicola* was isolated from the renal tissue of capybaras, but no histological changes or previous signs of disease were detected.

Brucellosis is an important disease identified in capybaras from groups that share pastures with cattle. Serological data are variable, but *Brucella abortus* and *Brucella suis* were isolated from tissue samples.[21] Clinical brucellosis has not been recognized in capybaras.

The parenteral route is indicated for giving antibiotics to rodents. Oral antibiotics should be avoided because they can cause serious imbalances in the intestinal flora. Several antibiotics may be used. It is advisable to take precautions when using certain penicillins to prevent allergic reactions. Enrofloxacin has become the antibiotic of choice in Brazilian zoos and gives good results against the majority of treatable infections.

FUNGAL DISEASES Some dermatomycoses may affect rodents. The most common fungi isolated from cutaneous lesions of South American rodents are *Trichophyton* and *Microsporum* spp. The treatment drug of choice is ketoconazole. In severe or refractory cases, such drugs as itraconazole or fluconazole are indicated.

REFERENCES

1. Acco, A.; Mangrich, R.M.V.; Pachaly, J.R.; Lange, R.R.; and Margarido, T.C.C. 1997. Determinação dos parâmetros de urinálise parcial em cutias (*Dasyprocta azarae*)(Relato preliminar [Urinalysis in *Dasyprocta azarae*(Preliminary report]. In Anais 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. Curitiba, ANCLIVEPA-PR, p. 20.
2. Acco, A. 1998. Mensuração dos Níveis Séricos de Cortisol e de Lactato Desidrogenase como Indicadores de Estresse em Cutia (*Dasyprocta azarae*) [Measurement of cortisol and serum lactate dehydrogenase levels and their roles as stress indicators in *Dasyprocta azarae*]. Masters thesis, Universidade Federal do Paraná.
3. Amaro, K.M.; and Souza, M.S.N. 1996. Determinação de proteínas e fraçães séricas em cutias (*Dasyprocta primnolopha*) mantidas em cativeiro. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 74.
4. Arias, J.F.; Garcia, F.; Rivera, M.; and Lopez, R. 1997. *Trypanosoma evansi* in capybara from Venezuela. Journal of Wildlife Diseases 33(2):359–61.
5. Augusto, A.Q.; Pachaly, J.R.; and Lange, R.R. 1995. Diagnóstico ultrassonográfico de gestação em cutia (*Dasyprocta azarae*)—Retato de caso [Ultrasonographic pregnancy diagnosis in *Dasyprocta azarae*—(Case report]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 10.
6. Bacher, J.D.; Potkay, S.; and Baas, E.J. 1976. An evaluation of sedatives and anesthetics in the agouti (*Dasyprocta* sp.). Laboratory Animal Science 26(2):195–198.
7. Bo, R.F.; Palomares, F.; Beltrán, J.F.; De Villafañe, G.; and Moreno, S. 1994. Immobilization of coypus (*Myocastor coypus*) with ketamine hydrochloride and xylazine hydrochloride. Journal of Wildlife Diseases 30(4):596–598.
8. Cabrera, A. 1961. Catalogo de los mamiferos de America del Sur [Catalog of South American Mammals]. Buenos Aires, CONI.
9. Cabrera, A.; and Yepes, J. 1940. Mamiferos Sud-americanos: Vida, Constumbres y Descripción [South American mammals: life, behavior and description]. Buenos Aires, Compañia Argentina de Editores, p. 230.

10. Cimardi, A.V. 1996. Mamíferos de Santa Catarina [Mammals of Santa Catarina]. Florianópolis, Fundação do Meio Ambiente de Santa Catarina, pp. 98–99.
11. Clark, J.D.; and Olfert, E.D. 1986. Rodents (Rodentia). In M.E. Fowler, ed., Zoo and Wild Animal Medicine 2nd Ed. Philadelphia, W.B. Saunders, pp. 728–737.
12. Eisenberg, J.F. 1989. Mammals of the Neotropics—The Northern Neotropics. Chicago, The University of Chicago Press.
13. Emmons, L.H. 1990. Neotropical Rainforest Mammals—A Field Guide. Chicago, The University of Chicago Press, pp. 166–210.
14. Goldbarg, M.; Reis, R.K.; Queiroz, P.V.S.; Medeiros, C.P.S.; and Souza, M.S.N. 1996. Determinação dos lipídeos séricos em cutias (*Dasyprocta primnolopha*) na região do semi-árido nordestino [Values for serum lipids in *Dasyprocta primnolopha*]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Pan American Association of Veterinary Sciences, p. 74.
15. González-Jiménez, E. 1977. Digestive physiology and feeding of capybaras. In M. Rechcige, ed., Diets for Mammals, Vol. 1. Cleveland, Ohio, CRC Press, pp. 163–177.
16. González-Jiménez, E. 1995. El Capybara (*H. hydrochaeris*): Estado Actual de su Producción [The capybara: present status of its production]. Roma, FAO.
17. Herrera, E.A. 1985. Coprophagy in the capybara, *Hydrochaerus hydrochaeris*. Journal of Zoology Sér. A 207(4):616–619.
18. Jelambi, F. 1976. Leptospirosis en Chigüires [Leptospirosis in capybaras]. Maracay, Informe Centro Investigaciones Veterinarias.
19. Lange, R.R. 1998. Criação e relocação de cutias *Dasyprocta azarae* Lichtenstein, 1823 (Dasyproctidae, Mammalia) em área verde urbana, Curitiba [Breeding and relocation of agoutis *Dasyprocta azarae* in the city of Curitiba]. Masters thesis, Universidade Federal do Paraná.
20. López, S. 1982. Una contribución al estudio de la fisiología reproductiva del chigüire (*H. hydrochaeris*) en cautiverio, 1. Ciclo estral [Contribution to the study of the capybara in captivity, 1. Estrous cycle]. Acta Cientifica Venezolana 33: 487–501.
21. Lord, V.R.; and Flores, C.R. 1983. *Brucella* spp. from the capybara (*H. hydrochaeris*) in Venezuela: Serologic studies and metabolic characterization of isolates. Journal of Wildlife Diseases 9(4):308–314.
22. MacDonald, D.W.; Krantz, K.; and Aplin, R.T. 1984. Behavioral, anatomical and chemical aspects of scent marking among capybaras (*H. hydrochaeris,* Rodentia: Caviomorpha). Journal of Zoology, London 202(3):341–360.
23. Machado, G.V. 1999. Personal communication. Umuarama.
24. Mangrich, R.M.V.; Acco, A.; Pachaly, J.R.; Lange, R.R.; and Margarido, T.C.C. 1997. Comparação dos parâmetros bioquímicos e físicos da urina de dois grupos de cutia (*Dasyprocta azarae*) [Comparison of urine biochemical and physical parameters between two groups of *Dasyprocta azarae*]. Archives of Veterinary Science 2(Suppl.):62.
25. Mangrich, R.M.V.; Pachaly, J.R.; Lange, R.R.; and Werner, P.R. 1995. Avaliação dos parâmetros bioquímicos e físicos da urina de cotiaras (*Myoprocta acouchi*) [Evaluation of biochemical and physical parameters of agouti urine]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 14.
26. Mangrich, R.M.V.; Pachaly, J.R.; Lange, R.R.; and Werner, P.R. 1995. Avaliação da glicemia em cutias (*Dasyprocta azarae*) [Blood glucose evaluation in agoutis]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 13.
27. Mangrich, R.M.V.; Pachaly, J.R.; Lange, R.R.; Silva, S.F.C.; Dittrich, R.L.; and Werner, P.R. 1996. Avaliação dos valores de hemograma de cutia (*Dasyprocta azarae*) [Hematological values for agoutis]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Pan American Association of Veterinary Sciences, p. 162.
28. Mangrich, R.M.V.; Pachaly, J.R.; Silva, S.F.C.; Dittrich, R.L.; Lange, R.R.; and Werner, P.R. 1996. Avaliação dos valores de hemograma de cotiara (*Myoprocta acouchi*) [Hematological values for acouchis]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Pan American Association of Veterinary Sciences, p. 70.
29. Monteiro-Filho, E.L.A.; Margarido, T.C.C.; Pachaly, J.R.; Mangini, P.R.; Montiani-Ferreira, F.; and Lange, R.R. 1998. Comportamento zoofágico inato de cutias—*Dasyprocta azarae* Lichtenstein, 1832 (Rodentia: Mammalia) [Innate zoophagous behavior in agoutis (*D. azarae*]. Arquivos de Ciências Veterinárias e Zoologia da Unipar 2(2):135–142.
30. Montiani-Ferreira, F.; Pachaly, J.R.; and Ciffoni, E.M.G. 1997. Valores para pressão intra-ocular em cutias (*Dasyprocta azarae*) anestesiadas pela associação cloridrato de xilazina, cloridrato de ketamina e sultafo de atropina [Values for intra-ocular pressure in agoutis anesthetized with ketamine HCl and atropine sulfate]. In Anais 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. Curitiba, ABRAVAS/AVEPER, p. 23.
31. Montiani-Ferreira, F.; Pachaly, J.R.; Lange, R.R.; Mangrich, R.M.V.; Mangini, P.R.; and Werner, P.R. 1996. Catarata e diabetes mellitus em cutia (*Dasyprocta azarae*) [Cataract and diabetes mellitus in an agouti]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Pan American Association of Veterinary Sciences, p. 71.

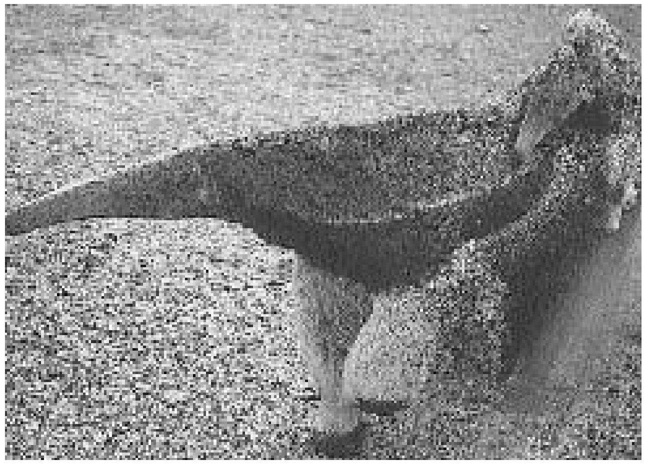
32. Morales, G.A.; Wells, E.A.; and Angel, D. 1976. The capybara (*H. hydrochaeris*) as a reservoir host for *Trypanosoma evansi*. Journal of Wildlife Diseases 12:572–574.
33. Morgan, R.J.; Eddy, L.B.; Solie, T.N.; and Turbes, C.C. 1981. Ketamine-acepromazine as an anaesthetic agent for chinchillas (*Chinchilla laniger*). Laboratory Animals 15:281–283.
34. Nogueira, T.M.R. 1997. Alguns Parâmetros Fisiológicos e Reprodutivos da Paca (*Agouti paca,* Linnaeus, 1766), em Cativeiro [Some physiological and reproductive parameters for captive pacas]. Masters thesis, Universidade Estadual Paulista.
35. Nunes, V.L.B.; Oshiro, E.T.; Dorval, M.E.C.; Garcia, L.A.M.; Da Silva, A.A.P.; and Bogliolo, A.R. 1993. Investigação epidemiológica sobre *Trypanosoma (Trypanozoon) evansi* no Pantanal Sul-Mato-Grossense: Estudo de reservatórios [Epidemiologic survey on *T. evansi* in the Pantanal Sul-Mato-Grossense: Reservoirs study]. Revista Brasileira de Parasitologia Veterinária 2(1):41–44.
36. Ojasti, J. 1973. Estudio Biológico del Chigüire o Capybara [Biological Study of the Capybara]. Caracas, Ediciones del Fondo Nacional de Investigaciones Agropecuarias.
37. Ojasti, J. 1991. Human exploitation of capybara. In J.G. Robinson and K.H. Redford, eds., Neotropical Wildlife Use and Conservation. Chicago, University of Chicago Press, pp. 236–252.
38. Pachaly, J.R. 1991. Chemical restraint and anesthesia in the paca (*Agouti paca*—Rodentia). In Abstracts of the 24th World Veterinary Congress. Rio de Janeiro, p. 196.
39. Pachaly, J.R. 1992. Estudo da utilização da associação cloridrato de cetamina, maleato de acetilpromazina e sulfato de atropina na contenção de *Agouti paca* (Linnaeus, 1766 Rodentia: Mammalia) [Study of the use of ketamine HCl, acetylpromazine maleate and atropine sulfate in the restraint of *Agouti paca*]. Masters thesis, Universidade Federal do Paraná.
40. Pachaly, J.R. 1992. Utilização da associação cloridrato de ketamina e maleato de acetilpromazina na contenção de *Dasyprocta* spp. [The use of ketamine HCl and acetylpromazine HCl in the restraint of *Dasyprocta* spp.] (summary). In 22° Congresso Brasileiro de Medicina Veterinária. (Curitiba, Sociedade Paranaense de Medicina Veterinária, p. 46.
41. Pachaly, J.R. 1996. Estudo da utilização da associação cloridrato de cetamina, maleato de acetilpromazina e sulfato de atropina na contenção de *Agouti paca* (Linnaeus, 1766 Rodentia: Mammalia) [Study of the combination of ketamine HCl, acetylpromazine maleate and atropine sulfate in the restraint of *Agouti paca*]. Archives of Veterinary Science 1(1):15.
42. Pachaly, J.R. 1998. Contenção da Cutia, *Dasyprocta azarae* (Lichtenstein, 1823 Rodentia: Mammalia), Pela Associação de Cloridrato de Cetamina, Cloridrato de Xilazina e Sulfato de Atropina—Definição de Protocolos Posológicos Individuais com Base em Extrapolação Aalométrica Interespecífica [Restraint of the agouti, *Dasyprocta azarae* with the combination of ketamine hydrochloride, xylazine hydrochloride and atropine sulfate—Individual dosages defined by interspecific allometric scaling]. Doctoral thesis, Universidade Federal do Paraná.
43. Pachaly, J.R.; and Brito, H.F.V. 1995. Emprego de cloridrato de detomidina, em associação a cloridrato de ketamina e sulfato de atropina, na contenção de cutias (*Dasyprocta azarae*), com base em extrapolação alométrica [Detomidine HCl, ketamine HCl and atropine sulfate in the restraint of *Dasyprocta azarae*—Allometric calculations]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 1.
44. Pachaly, J.R.; Lange, R.R.; and Farias, E.L.P. 1998. A new technique for venipuncture in agoutis (*Dasyprocta* spp.) and acouchis (*Myoprocta* spp) (summary). In 16th Panamerican Congress of Veterinary Sciences. Santa Cruz de la Sierra, p. 162.
45. Pachaly, J.R.; Mangrich, R.M.V; and Lange, R.R. 1994. Avaliação da glicemia em cutias—Relato preliminar [Blood glucose evaluation in agoutis—Preliminary report]. In Anais 49° Conferência Anual da Sociedade Paulista de Medicina Veterinária. São Paulo, Sociedade Paulista de Medicina Veterinária, p. 28.
46. Pachaly, J.R.; and Werner, P.R. 1998. Restraint of the paca (*Agouti paca*) with ketamine hydrochloride, acetylpromazine maleate and atropine sulfate. Journal of Zoo and Wildlife Medicine 29(3):303–306.
47. Queiroz, P.V.S.; Reis, R.K.; Goldbarg, M.; and Souza, M.S.N. 1996. Aspectos hematológicos das cutias (*Dasyprocta primnolopha*) da região do semi-árido nordestino [Some hematological values for *Dasyprocta primnolopha*]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 67.
48. Riviera, M.A. 1983. Sarna sarcoptica en chigüires (*H. hydrochaeris*) [Sarcoptic mange in *H. hydrochaeris*]. Revista de la Facultad de Ciencias Veterinarias de UCV 30(1-8):99–115.
49. Schaller, G.B.; and Crawshaw, P.G., Jr. 1981. Social organization in a capybara population. Saugetierk. Mitt. 29:3–16.
50. Silva, F. 1984. Mamíferos Silvestres do Rio Grande do Sul [Wild mammals of Rio Grande do Sul]. Porto Alegre, Fundação Zoobotânica do Rio Grande do Sul. pp. 71–209.
51. Souza, M.S.N. 1996. Avaliação da função renal de cutias (*Dasyprocta primnolopha*) mantidas em cativeiro [Evaluation of renal function in captive agoutis]. In Anais 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 68.
52. Souza, M.S.N.; Amaro, K.M.N.; Reis, P.F.C.C.; and Oriá, R.B. 1996. Determinação da concentração de ácido úrico em cutias (*Dasyprocta primnolopha*) mantidas em cativeiro [Evaluation of uric acid concentrations in captive agoutis]. In Anais 15° Congresso Panamericano de

Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 73.

53. Stoskopf, M.K. 1979. Anesthesia of zoo rodents. In Proceedings of the American Association of Zoo Veterinarians. Philadelphia, pp. 68–69.

54. Vilani, R.G.D'O.C.; Pachaly, J.R.; and Ciffoni, E.M.G. 1997. Valores de oximetria em cutias (*Dasyprocta azarae*) anestesiadas pela associação cloridrato de xilazina, cloridrato de ketamina e sultafo de atropina—Relato preliminar [Pulse oximetry values for agoutis anesthetized with xylazine HCl, ketamine HCl and atropine sulfate]. In Anais 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. Curitiba, ABRAVAS/AVEPER, p. 23.

55. Wallach, J.D.; and Boever, W.J. 1983. Rodents and lagomorphs. In J.D. Wallach and W.J. Boever, eds., Diseases of Exotic Animals—Medical and Surgical Management. Philadelphia, W.B. Saunders, pp. 135–147.



24

Order Xenarthra (Edentata) (Sloths, Armadillos, Anteaters)

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BIOLOGY AND CAPTIVE MANAGEMENT OF ARMADILLOS AND ANTEATERS

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FAMILY DASYPODIDAE

Introduction

The armadillos, one of the few original South American groups that successfully spread to Central and North America during the Miocene and Pliocene periods, comprise a group of 20 species divided into eight genera and two subfamilies.[10]

They differ from other mammals in that their bodies are shielded by cornified plates, with a varying number of bands. Movable or not, these bands are located in the dorsal region. The ventral area, on the other hand, is soft and unprotected. Body mass ranges from 90 g in the small hairy armadillo (*Chlamyphorus truncatus*) to 60 kg in the giant armadillo (*Priodontes maximus*). Armadillos grow hair between the shields, that is, in some species, dense. The basic color varies from gray to brown. Their vision is poor, but their sense of smell is excellent. There are three to five digits in the fore feet, which are powerful in the fossorial species, and five in the hind feet. The number of teeth is highly variable, from 7-8/7-8 to 18/19. Some species retain the incisors, but all have lost the canines.

Biology

Subfamily Chlamyphorinae

***CHLAMYPHORUS* (TWO SPECIES)** *Chlamyphorus retusus* is found in the Grán Chaco of southeastern Bolivia, western Paraguay, and northwestern Argentina,[29] whereas *C. truncatus* ranges in the Argentinean provinces of Mendoza, San Luis, La Pampa, eastern La Rioja, southern Catamarca, Córdoba, and western Buenos Aires.[29]

Members of this genus are the smallest edentates. *C. retusus* has a body length of 140–180 mm and *C. truncatus,* 125–150 mm. In the latter species, the carapace is loosely attached to the body by a thin membrane along the spine and butt plate, a distinguishing characteristic. Its tail is spatulate at the end and covered by plaques, which gives the animal support while feeding.[20] It inhabits rainless and sandy soil areas, whereas *C. retusus* prefers dry and arid soil or well-drained grasslands.

C. retusus is not a good digger like *C. truncatus*, which is fossorial. To avoid predators, *C. retusus* flattens on the ground. It has been reported that both species feed on insects and larvae, as well as plants;[19,24] however, Minóprio[20] reports that vegetable matter is ingested accidentally when foraging. Additionally, the form and texture of the teeth are not appropriate for plant ingestion.

There is limited data on *C. truncatus*. Endemic to Argentina's central region, it has become rare as a consequence of human encroachment, land cultivation, and predation by domestic dogs. Its status is considered endangered by the U.S. Endangered Species Act, and is insufficiently known by The World Conservation Union (IUCN). The same is true of *C. retusus*.

Subfamily Dasypodinae

***CABASSOUS* (FOUR SPECIES)** *Cabassous centralis* ranges from Mexico to Colombia.[29] *Cabassous chacoensis* ranges from the Gran Chaco of northwestern Argentina to western Paraguay and southeastern Bolivia, and, possibly, into adjacent areas of Brazil.[38] *Cabassous tatouay* is found in southern Brazil, Uruguay, and the Misiones province of Argentina, and, perhaps, as far south as the province of Entre Rios.[29] The range of *Cabassous unicinctus* is east of the Andes in Venezuela to the Amazon's Solimães River.[38]

Distinguishing characteristics of the *Cabassous* species are a flexible carapace with 11 bands and an unshielded tail that is covered only by skin and is soft to the touch, hence the common name, naked-tailed armadillo. They are a fossorial, solitary, and nocturnal species that inhabit grassland and upland plateaus. In Paraguay they are found in semiarid lowland and moist palmettos; in Panama they are found in highland areas and along embankments adjacent to roadways, as well as in moist lowland forest. In northern Argentina, burrows and live animals were found along the Pilcomayo River.[18] *C. unicinctus* and *C. tatouay* are, likewise, not limited to the cerrado (savanna) region of Brazil.[27] Active burrows have a musky odor, making naked-tailed armadillo burrows easily recognizable.

C. tatouay has large and prominent ears, whereas *C. unicinctus* has the smallest. It also has powerful claws with large nails, which allows it to dig rapidly.[27] *C. tatouay* changes burrows every day and does not return to them.[9,33] These animals are part of the ant-eating/termite-eating group. Only one young is born at a time.

***CHAETOPHRACTUS* (THREE SPECIES)** The distribution of *Chaetophractus nationi* is poorly known because it has frequently been confused with *Chaetophractus vellerosus*. It is found in the puna of Bolivia and extends into the highlands of Chile.[29] *C. vellerosus* is found in the Grán Chaco, western Paraguay, and Argentina, extending south to Mendoza province and the middle latitude of Buenos Aires province where its distribution is disjunct.[29] *Chaetophractus villosus* is found in the chaco of Paraguay and Argentina (south at least to Santa Cruz province) and in Chile along the eastern edge of the province of Bío-bio south to Aisén province.[29]

Chaetophractus spp. are found at both low and high altitudes. *C. nationi* and *C. villosus* are found in grasslands. *C. nationi* also inhabits dry savanna, and scrub desert. *C. vellerosus* is usually found in areas with yearly rainfall between 200 and 600 mm, but there is a population in eastern Buenos Aires province, in Argentina, where the annual rainfall is 1000 mm.

Chaetophractus spp. are commonly known as hairy armadillos because they have more hair than other genera of armadillos. The shield has 6–8 movable bands. *C. villosus* has scent glands in the middorsum of the pelvic shield, as does *Euphractus sexcinctus*.[38] Usually they are nocturnal, but during the winter they are diurnal, probably limited by temperatures. They are solitary, although *C. nationi* may form groups while feeding on carcasses. The gestation period for this genus is between 60 and 75 days; usually two young are born. Hairy armadillos are considered to be carnivorous omnivores. Their diet consists mostly of insects, plants, tubers, small rodents, lizards, fruits, vegetables, and carrion.[24,28]

The status of these animals is uncertain. Humans frequently eat them and, sometimes, the armadillos die because many burrows are destroyed and the land used for farming.

***DASYPUS* (SIX SPECIES)** *Dasypus hybridus* is found from eastern Paraguay, eastern Argentina, southern Brazil, and Uruguay, west to Jujuy province and south to Mendoza and Rio Negro provinces in Argentina.[29] It inhabits dense forest and savanna, from sea level to about 300 m.

Dasypus kappleri ranges in the lowland rain forests of the Orinoco and Amazon basins.[29]

Dasypus novemcinctus is found from the southern United States to southern Uruguay, in eastern and western Paraguay, and Argentina south to the provinces of Santiago del Estero, Santa Fé, and Entre Rios.[29] It prefers forests and disturbed woodlands over other habitats. This armadillo has the broadest distribution area.

Dasypus pilosus is known only from the Peruvian Andes in San Martin, La Libertad, Huánuco, and Junin.[10] It prefers open grassland and rocky upland areas.

Dasypus sabanicola ranges in the Colombian and Venezuelan savannas.[38]

Dasypus septemcinctus is found in the lower Amazon River basin, south through eastern Brazil to Rio Grande do Sul, and west to Mato Grosso, southeastern Bolivia, and northern Argentina.[38] It prefers the cerrado (savanna) areas and other open habitats.

Long-nosed armadillos are animals that have, as the name implies, a characteristic long and thin muzzle. The carapace has 6–11 moveable bands. They are excellent swimmers, especially when running from predators. Good diggers, their burrows have many entrances and exits and may be visited by several individuals, probably of the same sex.[24] Although they have predominantly nocturnal habits, they sometimes forage during daylight.

D. kappleri has about 8-12 kg of body mass, *D. novemcinctus* weighs up to 6 kg, *D. sabanicola* 4 kg, *D. septemcinctus* 3 kg, and *D. hybridus* 2 kg. *D. pilosus* has a body length of 370–400 mm and a tail length of 250–270 mm.

D. septemcinctus usually expands burrows made by other armadillos and is easily confused with *D. novemcinctus*. Both are common in their natural habitats. The gestation period for this genus is between 120 and 260 days. The number of young varies. *D. hybridus* gives birth to 8–12 young, *D. septemcinctus* 4–8, *D. novemcinctus, D. sabanicola,* and *D. klapperi* about 4–5 young, all of the same sex.

The diet is that of a generalist insectivore.[28] They feed mostly on insects, ants, and beetles, but also may eat small vertebrates, such as small reptiles, amphibians, birds, and small mammals. This genus does not have a special protected status, even though humans commonly use their meat as food. In some areas their presence is welcome because they eat vermin insects.

D. novemcinctus is the species of this genus that is most studied, both in the United States and in South America. Armadillos are the only mammals other than humans known to be highly susceptible to leprosy. For this reason, they are valued by researchers against *Mycobacterium lepra,* especially *D. novemcinctus,* because this species is common, has a lifespan longer than 10 years, and bears a large litter.[25] Studies have found this species to be a natural reservoir for *Trypanosoma* spp., *Paracoccidioides brasiliensis,* and *Leishmania* spp.[35]

***EUPHRACTUS* (ONE SPECIES)** This genus is comprised of only one species, *Euphractus sexcinctus,* found in the savannas of Suriname and adjacent areas of Brazil, south to Uruguay, eastern and western Paraguay, and in Argentina south to Buenos Aires province.[29] The species is commonly found in savannas (cerrado) and forest edges.[30] It appears to prefer higher, drier habitats and is rarely seen in marshy areas.[34]

E. sexcinctus is one of the largest armadillos; only *P. maximus* and *Dasypus kappleri* are larger. Its head and body are covered with armored plates, with 6 to 8 moveable bands, sparsely covered with long, stiff hairs. It is a good digger and builds single-entrance burrows that are frequently reused. One male used a single burrow for 18 days.[1] They are solitary and nocturnal animals, but are occasionally active during the day.[34] The gestation period is between 60 and 64 days, after which one to three young are born.

In diet, *E. sexcinctus* may be classified as an omnivorous carnivore, because it eats predominantly small vertebrates, snails, worms, insects, and carrion.[24,28] It is common and therefore not threatened.

***PRIODONTES* (ONE SPECIES)** *P. maximus* is found from Colombia and south Venezuela to Paraguay and northern Argentina.[29] Reported to prefer undisturbed forests near water, they are also found in grassland or brush land with wooded patches nearby.

As the name implies, giant armadillos are the largest members of the family, weighing up to 55 kg. They are solitary, nocturnal, extremely powerful diggers, and highly fossorial. Their burrows may be as long as 5 m and reach a depth of 1.5 m, each with several entrances and exits. They are destructive when foraging, destroying whole termites mounds, and, in wooded areas, uprooting small trees and shrubs. The gestation period is about 120 days, with one or two young born. *P. maximus* is considered the most myrmecophagous of the armadillos, its diet consisting of adult ants and termites, their eggs and larvae.[28]

The giant armadillo is rare in Paraguay and appears to have no resident population. There is no law protecting this species and no immediate plan to implement such legislation.[17] Studies conducted in the chaco region of Argentina showed that this armadillo's distribution has declined to 60% of its historical range. Not only this species, but also other armadillos rank first or second in the list of the wildlife most hunted for subsistence throughout their habitats.[40] It is classified as vulnerable by IUCN and endangered by the United States and is listed in Appendix 1 of Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES).

***TOLYPEUTES* (TWO SPECIES)** *Tolypeutes matacus* ranges from southeastern Bolivia, through the Paraguayan chaco, to the Argentine province of Santa Cruz.[29] *Tolypeutes tricinctus* has only an undefined range in the northeastern highlands of Brazil. Only a few specimens have been seen, and scientists lack information on the present status of this endangered species.[38]

Armadillos are easily recognized because of their peculiar ability to roll up and form a ball when threatened, in order to protect the soft underparts. Other than their geographic distribution, these two species may be differentiated by the number of digits on the limbs. *T. matacus* has three or four digits, whereas *T. tricinctus* has five digits. Neither species is a strong digger, so they tend to use empty burrows made by other armadillos during the day. At night they forage. They never return to the same burrow, probably to avoid predators. The gestation period is 120 days, with only one birth.

Although *T. matacus* is used as food by locals throughout its distribution area, it is not threatened.[37] On the other hand, *T. tricinctus* is classified as "indeterminate" by IUCN and "threatened" by the Brazilian official list of endangered species. It is not listed in CITES appendices. *T. tricinctus* was once common throughout the Brazilian caatinga region, but, unfortunately, its population has been drastically reduced. Because the area's climate is rough, with little rainfall, agricultural activities tend to fail and locals depend upon wildlife as a source of protein.[32]

For 20 years the scientific community had no records of sightings of *T. tricinctus* in the wild, until Santos et al.[33] found remains of burnt carapaces in northern Bahia. They discovered that eating this species was a common practice in the region, probably the main reason for its decline. Another population was recently found in the cerrado area close to the borders of the Brazilian states of Goias and Bahia, expanding the species distribution.[14] Little is known about this species.

Both species of three-banded armadillos are considered to be strict insectivores. However, studies have shown that in addition to its preferred food items, termites and ants,[11] *T. tricinctus* also eats fruits, spiders, beetles, and centipedes.[32] *T. matacus* is also highly myrmecophagous, but takes other soft-bodied invertebrates as well.

***ZAEDYUS* (ONE SPECIES)** *Zaedyus pichiy* is found in Argentina from the provinces of Mendoza, San Luis, and southern Buenos Aires to Rio Santa Cruz, west to the puna of the Andes, and in southern Chile from the province of Aconcagua to the Strait of Magellan.[29] They inhabit the higher areas of grassland and pampas and prefer warm, dry, sandy soil. *Z. pichiy* is a small-bodied species, with a body length of 250 to 400 mm and a body mass of about 1 kg. There are usually 9 movable bands. It is solitary and most active at dusk and dawn. This species is a highly sought-after food item for the native population in its distribution areas. *Z. pichiy* is also kept as a pet.[24]

In some areas of its range, this armadillo is reported to hibernate during the winter.[24] The gestation period is about 60 days, after which one to three young are born. It is considered a carnivorous omnivore, with a preference for the larvae and cocoons of ants, but also consumes tarantula spiders, a species of soft-bodied larvae, and plants.[16]

Management

The management of armadillos requires strict attention to detail. It is necessary to understand their habits in the wild in order to offer similar care in captivity. The most difficult problem is the diet, which, for most species, is highly specialized.

To build an adequate enclosure for armadillos requires that every detail be carefully checked. For example, for some species of *Dasypus, Euphractus,* and *Priodontes,* it is not advisable to use wire screen, because animals may scale it and escape. They may also traumatize their feet and muzzles while trying to escape. Customarily, the material used as bedding is composed of dry leaves, fine sand, or shredded paper, but these may cause eye problems and also dehydrates the carapace. Hay should not be used for some species, such as *Cabassous* and *Dasypus,* because they consume it and it causes problems in the digestive tract, sometimes resulting in rapid death. Nocturnal species may be exhibited in dark enclosures (nocturnal house), using reverse photoperiod, if proper humidity and temperature are provided.

C. villosus is exhibited in the Pozan Zoo, Poland, in an enclosure of 3.5 m^2, with a concrete floor and a variety of wooden maternity or resting boxes. All floors are covered with leaves and/or tree bark. Because winters are extremely cold in Poland, heating is necessary. There were 21 births between 1991 and 1996, all from the same female. The diet consists of raw or cooked minced beef or horsemeat mixed with chopped fruits, raw, grated, or cooked vegetables; baby cereal or boiled rice with milk; and boiled eggs or raw yolks.[26]

Enclosures for *C. nationi* and *C. vellerosus* can be similar to that of *C. villosus,* but the diet must include day-old chicks or mice.

E. sexcinctus is well adapted to captivity. However, it is important to supply nesting material for females, such as leaves, shredded paper, or tree bark. At São Paulo Zoo, births have been recorded, but, unfortunately, the young were abandoned shortly after birth and died. The diet consists of manioc, yams, carrots, eggs, peanuts, day-old chicks, and crushed necks of chicken.

D. novemcinctus is probably the species most reproductively successful in captivity. They are maintained in laboratories and used for medical research. Enclosures in medical laboratories are smaller than zoo and park enclosures. For display it is necessary to provide small water pools where it can swim or just wet its feet.

A young, 25-day-old female donated to the São Paulo Zoo was fed milk from a nursing bottle. The formula of reconstituted dried milk, honey, egg yolk, and vitamin and mineral supplements was offered several times during the day and twice at night. After 20 days, baby food was offered in bowls, consisting of an assortment of fruits and vegetables, plus milk mixed with flour, sugar, dried milk, vitamin supplements, and salt. Gradually, the same diet fed to *E. sexcinctus* was offered, with the addition of tenebrio larvae, twice a week. *D. novemcinctus* is kept in enclosures with a 1-m high fence of concrete and wire mesh and a bedroom. Concrete floors are covered with sand.

P. maximus is difficult to keep in captivity. Because the species is an extremely fossorial and insectivorous animal, it is necessary to build a special enclosure, which should be made of concrete with no screens, because the animal will climb. Additionally, it must have a pool and an appropriate area for digging to wear off the nails. The London Zoo occasionally displayed this species, feeding it with a canned carnivore diet of minced meat, along with carrots, cabbage, a bit of milk, and vitamin supplements, mixed to a solid consistency.[15]

There is little information about the care and handling of *Z. pichiy.* There is a record of a birth in 1972.[15] Its diet should probably be the same as for *Chaetophractus*.

Chlamyphorus may be displayed in nocturnal houses. At Michigan State University, *C. truncatus* was kept in an aquarium 50 × 25 × 30 cm, with a floor covered with fine sand and a wooden maternity box. The diet was bread and milk, whole oats, and, occasionally, beetles and beetle larvae.[31] *T. tricinctus* is kept at Brasilia Zoo in an enclosure with a soil floor, a water pool, stones, shrubs, and burrows. Its diet consists of larvae, adult tenebrios, pupae, dog food, termites, small eggs, papaya, melons, other fruits, and seeds, all mixed and mashed, offered twice a day. There is no record of births.[12]

FAMILY MYRMECOPHAGIDAE

Introduction

The family Myrmecophagidae[7] is represented by three genera with four species of anteaters. The four species show great similarities: lack of teeth, poor vision, and excellent olfaction. All are considered nocturnal or crepuscular and solitary.

Adult sizes are different; *Myrmecophaga* may weigh 40 kg, *Tamandua,* 7 kg, and *Cyclopes* just 400 g. *Tamandua* and *Cyclopes* are arboreal and have a prehensile tail, whereas *Myrmecophaga* is believed to be terrestrial. The three genera are insectivorous, eating almost exclusively ants and termites. They have some characteristics that differentiate them from all other mammals: additional lumbar vertebrae and rather simple skulls, with no canines, incisors, or premolars. The family Myrmecophagidae is the only one that is strictly toothless and that possesses a double posterior vena cava vein (single in other mammals), which returns blood from the hindquarters of the body to the heart. Females have a primitive divided womb, only a step removed from the double womb of marsupials, and a common urinary and genital duct, whereas males have internal testes and a small penis with no glans.[13] As a result of the low energy value of its diet, anteaters have a low metabolic rate as well as a low and variable body temperature (33–35.5°C), which prevents a loss of energy.

Biology

CYCLOPES *Cyclopes didactylus,*[39] the silky anteater, is found in forests from tropical Mexico (Veracruz), to South America west of the Andes, possibly as far south as northwestern Peru; east of the Andes through the forests of the Orinoco and Amazon basins to extreme eastern Brazil in coastal Pernambuco and Alagoas; western Brazil (Amazonas) and Amazonian Peru to southeastern Bolivia (Santa Cruz and Buenavista).[23] Adult head and body length varies from 360 to 450 mm, tail length varies from 180 to 262 mm, and the body mass is about 400 g.[5] The pelage is dense and golden brown. The upper parts are usually darker, and a darker line runs along the top of the head, neck, and back. The tail is prehensile and naked underneath.

The silky anteater is strictly nocturnal,[22,36] arboreal, and solitary. Its diet consists of termites and ants, especially those from the genera *Crematogaster, Solenopsis, Camponotus,* and *Zacryptocerus*.[21] The gestation period lasts between 120 and 150 days, after which a single young is born.

MYRMECOPHAGA *Myrmecophaga tridactyla,*[39] the giant anteater, is found from Belize and Guatemala south through the Paraguayan chaco and northern Argentine provinces of Missiones, Formosa, Salta Jujuy, and probably Chaco and Santiago del Estero. This species is probably extinct in Uruguay.[6] Giant anteaters are relatively large-bodied animals, with a long tail and long, coarse, and stiff hair. Adult measurements vary as follows: head and body length from 1000 to 1200 mm, tail length from 650 to 900 mm, and body mass from 18 to 39 kg[23] (one animal kept at São Paulo Zoo weighed 51.7 kg). The ears and eyes are small. The third front claw is greatly enlarged and powerful. The upper parts are grayish with a diagonal black stripe bordered in white.

The species is found in a great variety of habitats, ranging from evergreen and deciduous forests to dry (cerrado) and wet/swampy (pantanal) savannas.[4] Giant anteaters are solitary, and not strictly nocturnal. They feed predominantly on termites and ants. However, in Serra da Canastra National Park the species was considered to be a flexible specialist, because it used resources according to availability.[4] Although they are said to be terrestrial, adult giant anteaters can climb trees up to 8 m, when needful, or just for rest (K. Cassaro, personal observation). The gestation period is about 160 days, after which usually one young is born, but twins have been reported.[15]

***TAMANDUA* (TWO SPECIES)** *Tamandua mexicana*[39] is found from the southeastern edge of the Mexican plateau to South America west of the Andes and from northwestern Venezuela to northwestern Peru.[22] *Tamandua tetradactyla*[39] is found from Venezuela south through Paraguay to northern Uruguay and the northern Argentine provinces of Santa Fé, Chaco, Salta, and Jujuy.[6]

Adult measurements vary as follows: head and body length from 470 to 770 mm, tail length from 402 to 672 mm, and body mass from 2 to 7 kg[23] (one specimen kept at São Paulo Zoo weighed 12 kg). The hair is short and dense, and the upper parts have a golden coloration. A black vest may be found over the belly and from the lower back forward in a band over the shoulder and around the chest. Depending upon the geographic area, the vest may be absent or only partially present.[8] Collared anteaters inhabit savannas, thorn scrub, and a wide range of wet and dry habitats. They specialize in eating termites and ants. Gestation lasts about 150 days, after which a single young is usually born, but twins have been reported.[15]

Management

Data obtained by the Brazilian Zoo Society from 1996 to 1998[2] have shown an increase in the reproduction of captive giant and collared anteaters. The percentage of captive giant anteaters that were reproducing in 1996 was only 6.67%, whereas in 1998 reproductive success had doubled to 13.64%. In the same period, the reproductive success of captive collared anteaters increased from 2.5% to 6.6%. Since 1983, 25 births of giant anteaters have occurred at São Paulo Zoo. Nine of these young survived, three of which have already produced the second generation.

Adults have been adequately maintained in enclosures of 250 m^2, with two holding areas of 10 m^2. The animals dig shallow holes next to major chumps of vegetation, where they rest. The enclosures have natural soil and abundant vegetation, and only the holding areas have floors of concrete.

Giant anteaters are kept in pairs, but the male should be removed after pregnancy is detected (the female remains in the enclosure). The young remain with the mother until they are about 1 year old, when they are completely weaned and totally independent. They are born at any time of the year as a miniature adult, with the eyes open, and weighing from 1.2 to 1.5 kg. At about 3 months of age, they start eating solid food, encouraged by the mother. At this time they also begin exploring the surroundings of the enclosure. If they are handled from an early age, they tend to become more docile and less stressed when handled as adults. Once a compatible pair has been kept together it is not advisable to split them, because there seems to be some sort of selection among pairs.

Adults are fed satisfactorily with a mixture of soybean milk, dog food, boiled egg, ground beef, and vitamin and mineral supplements. Termites are provided weekly, because in addition to providing environmental enrichment, they also help intestinal motility.

Hand-raised young animals are fed a mixture of cow's milk, cream, and egg yolk. This diet should be changed gradually to the mixture fed to adults, which usually happens at the age of 6 months. Young raised by their mothers showed a faster growth rate than hand-raised animals.

Collared anteaters may be kept in pairs in enclosures of 25 m^2, with heated holding areas. The species is selective when pairings are made. There has been one instance in which an animal was fatally injured by its prospective mate. If animals had been handled from an early age, they were more easily handled as adults (for weighing, pregnancy detection, etc.).

Births take place at any time of the year. At São Paulo Zoo, 10 births were registered during 8 different months. A female collared anteater started mating at about 2 years of age and raised an offspring for 7 consecutive years. This female, after the fifth parturition, developed problems with milk production, which necessitated hand-raising the remaining young. At present, São Paulo Zoo's collared anteaters are in their second generation. Young are not carried by their mothers all the time. They are born with a lighter coloration than adults, without a well-defined vest, weighing 240–590 g, and with the eyes completely open. Adults are fed with the same mixture as that reported for giant anteaters, adding fruits, and the young eat the same foods as that reported for baby giant anteaters.

REFERENCES

1. Carter, T.S.; and Encarnação, C. 1983. Characteristics and use of burrows by four species of armadillos in Brazil. Journal of Mammology 64:103–108.

2. Sociedade de Zoológicos do Brasil. 1996. Censo da Sociedade de Zoologicos do Brasil 1996–98 [Census of Brazilian Zoo Society 1996–1998]. Brazilian Zoo Society.
3. Crandall, L.S. 1964. The Management of Wild Mammals in Captivity. Chicago, University of Chicago Press, pp. 183–187.
4. Drumond, M.A. 1982. Padrães de Forrageamento do Tamanduá-bandeira (*Myrmecophaga tridactyla*) no Parque Nacional da Serra da Canastra: Dieta, Comportamento Alimentar e Efeito de Queimadas [Foraging patterns of the giant anteater in the National Park of Serra da Canastra: Diet, feeding behavior and effects of burns]. Masters thesis, Universidade Federal de Minas Gerais, Brasil.
5. Eisenberg, J.F. 1989. Mammals of the Neotropics, Vol. 1. The Northern Neotropics. Chicago, University of Chicago Press, pp. 50–55.
6. Eisenberg, J.F.; and Redford, K.H. 1992. Mammals of the Neotropics, Vol. 2. The Southern Cone. Chicago, University of Chicago Press, pp. 47–50.
7. Eisenberg, J.F.; and Redford, K.H. 1999. Mammals of the Neotropics, Vol. 3. The Central Neotropics. Chicago, University of Chicago Press, pp. 90–94.
8. Emmons, L.H. 1990. Neotropical Rainforest Mammals—A Field Guide. Chicago, University of Chicago Press, pp. 31–35.
9. Encarnação, C. 1986. Contribuiçào à Biologia Dos Dasypodídeos da Serra da Canastra, Minas Gerais [Contribution to biology of the Dasypodidae at Serra da Canastra, Minas Gerais]. Masters thesis, Universidade Federal do Rio de Janeiro.
10. Gardner, A L. 1993. Order Xenarthra. In D.E. Wilson and D.M. Reeder, eds., Mammal Species of the World: A Taxonomic and Geographic Reference. Washington, D.C., Smithsonian Institution Press, pp. 64–67.
11. Guimarães, M.M. 1997. área de Vida, Territorialidade e Dieta do Tatu-bola, *Tolypeutes trincinctus* (Xenarthra, Dasypodidae): Num Cerrado do Brasil Central [Three-banded armadillo: Life area, territoriality and diet at Brazilian Central Cerrado]. Masters thesis, Universidade de Brasília.
12. Juarez, K.M.; and Pereira, G.N. 1999. Registro de ocorrência e manejo ex-situ do tatu-bola (*Tolypeutes trincinctus)* no Zoológico de Brasília [Three-banded armadillos occurrence record and management at Brazilia Zoo]. Pers. comm.
13. Macdonald, D. 1985. The Encyclopedia of Mammals. New York, Facts On File, pp. 770–775.
14. Marinho-Filho, J.; Guimarães, M.M.; Reis, M.L.; Rodrigues, F.H.G.; Torres, O; and de Almeida, G. 1997. The discovery of the Brazilian three banded armadillo in the cerrado of central Brazil. Edentata 3(1):11–13.
15. Merrett, P.K. 1983. Edentates. Zoological Trust of Guernsey, p. 88.
16. Meritt, D.A., Jr. 1973. Edentate diets, 1. Armadillos. Laboratory of Animal Science 23(4):540–542.
17. Meritt, D.A., Jr. 1973. Observations on the status of the giant armadillo, in Paraguay. Zoologica 58(3(4):103.
18. Meritt, D.A., Jr. 1985. Naked-tailed armadillos, *Cabassous* spp. In G.G. Montgomery, ed., The Evolution and Ecology of Armadillos, Sloths, and Vermilinguas. Washington DC,, Smithsonian Institution Press, pp. 389–391.
19. Meritt, D.A., Jr. 1985. The fairy armadillo, *Chlamyphorus truncatus* Harlan. In G.G. Montgomery, ed., The Evolution and Ecology of Armadillos, Sloths, and Vermilinguas. Washington DC, Smithsonian Institution Press, pp. 393–395.
20. Minóprio, J.D.L. 1945. Sobre el *Chlamyphorus truncatus* Harlan [About the *Chlamyphorus truncatus* Harlan]. Acta Zoologica Lilloana Lilloana 3:5–58.
21. Montgomery, G.G. 1983. *Cyclopes didactylus* (Tapacara, Serafin de Platanar, Silky Anteater). In Costa Rica Natural History. Chicago, University of Chicago Press, pp. 461–463.
22. Montgomery, G.G. 1985. The Evolution and Ecology of Armadillos, Sloths and Vermilinguas. Washington DC, Smithsonian Institution, p. 451.
23. Nowak, R.M. 1991. Walker's Mammals of the World. Baltimore, Johns Hopkins University Press, pp. 522–525.
24. Nowak, R.M. 1991. Walker's Mammals of the World, 5th Ed. Baltimore, Johns Hopkins University Press, pp. 525–535.
25. Opromolla, D.V.A; de Arruda, O.S.; and Fleury, R.N. 1980. Manutenção de tatus em cativeiro e resultados de inoculação do *Mycobacterium leprae* [Captivity armadillos maintenance and *Micobacterium leprae* inoculation results]. Hansenologia Internacionalis 5(1):28–36.
26. Ratajszczak, R.; and Trzesowska, E. 1997. Management and breeding of the Larger Hairy Armadillo, *Chaetophractus villosus,* at Pozan Zoo. Zoologische Garten 67(4):220–228.
27. Redford, K.H. 1994. The edentates of the cerrado. Edentata 1(1):4–10.
28. Redford, K.H. 1985. Food habits of armadillos (Xenarthra: Dasypodidae). In G.G. Montgomery, ed., The Evolution and Ecology of Armadillos, Sloths, and Vermilinguas. Washington DC, Smithsonian Institution Press, pp. 429–437.
29. Redford, K.H.; and Eisenberg, J.F. 1992. Mammals of the Neotropics, Vol. 2. Chicago, University of Chicago Press, p. 430.
30. Redford, K.H.; and Wetzel, R.M. 1985. *Euphractus sexcinctus*. Mammalian Species 252:1–4.
31. Rood, J.P. 1970. Notes on the behavior of the pygmy armadillo. Journal of Mammology 51(1):179.
32. Santos, I.B. 1993. Bionomia, Distribuição Geográfica e Situação Atual do Tatu-bola, no Nordeste do Brasil [Bionomia, geographic distribution and real situation of three-banded armadillo Brazilian northeastern]. Masters thesis, Instituto de Ciências Biológicas, Universidade Federal de Minas Gerais.
33. Santos, I.B.; Fonseca, G.A.B.; Rigueira, S.E.; and Machado, R.B. 1994. The rediscovery of the Brazilian three banded armadillo and notes on its conservation status. Edentata 1(1):11–15.
34. Schaller, G.N. 1983. Mammals and their biomas on a Brazilian ranch. Arquivos Zoologia 31:1–36.

35. Silva, E.A.; Arruda, M.S.P.; R£bio, E.M.; and Rosa, P.S. 1999. Estudo dos sistemas sangüineos ABO, Rh(D), MN e Duffy em *Dasypus novemcinctus* (Sanguine system studies ABO, Rh(D), MN and Duffy in *Dasypus novemcinctus*). Pers. comm.
36. Sunquist, M.E.; and Montgomery, G.G. 1973. Activity pattern of a translocated silky anteater (*Cyclopes didactylus*). Journal of Mammology 54:782.
37. Vizcaíno, S.F. 1997. Armadillos del noroeste Argentino (Provincias de Jujuy y Salta) [Argentinian northeastern armadillos, Jujuy and Salta provinces]. Edentata 3(1):7–10.
38. Wetzel, R.M. 1985. Taxonomy and distribution of armadillos, Dasypodidae. In G.G. Montgomery, ed., The Evolution and Ecology of Armadillos, Sloths, and Vermilinguas. Washington D.C., Smithsonian Institution Press, pp. 23–46.
39. Wilson, D.E.; and Reeder, M.D. 1992. Mammal Species of the World, 2nd Ed. Washington D.C., Smithsonian Institution Press, pp. 67–68.
40. Zuleta, G.; and Bolkovic, M.L. 1994. Conservation ecology of armadillos in the Chaco region of Argentina. Edentata 1(1):16.

BIOLOGY AND CAPTIVE MANAGEMENT OF SLOTHS

Carlos Esbérard

Until recently the five living sloth species were classified in the family Bradypodidae. Now sloths are classified in two families, Bradypodidae (three-toed sloths) and Choloepidae (two-toed sloths). All species are restricted to Central and South America (Table 24.1).[6]

The maned sloth is an endemic species of the Atlantic Forest of Brazil in the states of Bahia, Espírito Santo, and Rio de Janeiro.[2,8] This species is distinguished from others by its fur, which is a uniform brown, except for black on the throat and shoulders. It weighs up to 6.5 kg.

The brown-throated three-toed sloth has the widest distribution of the five species, occurring from Costa Rica south to Ecuador, Colombia, and Venezuela, continuing east of the Andes to Peru, Bolivia, and northern Argentina. It is also found in the forests of Brazil (except Amapá) to Rio Grande do Sul. This species is distinguished from other species by the brown fur of the throat and face.[8] It weighs up to 5 kg.

The pale-throated three-toed sloth is limited to the Amazon forest, being found in Venezuela, Guyana, Surinam, French Guiana and northern Brazil, from Amapá to Pará (east of the Rio Negro), and south of the Rio Amazonas, where it shares the range of *Bradypus variegatus*. This species may be identified by the white or pale yellow fur of the throat and head.[8] It weighs 3.5–4.5 kg.

Linné's two-toed sloth can be distinguished by a throat similar in color to the pectoral hair and its 6 to 8 cervical vertebrae. It occurs from Venezuela (delta of Rio Orinoco) to Colombia, Guyana, Surinam, French Guiana, Brazil (from Maranhão west along the Rio Amazonas), Ecuador, and Peru.[8] It weighs 7 to 9 kg.

Hoffman's two-toed sloth has pale throat fur contrasting with a darker pectoral pelage, and five or six cervical vertebrae. This species is found from northern Nicaragua south on the west side of the Andes from Colombia to northwestern Ecuador and western Venezuela, and east of the Andes from Rio Solimães to at least 11° south. In Brazil it occurs in the states of Amazonas (southwestern) and Mato Grosso (northern), and probably in Acre.[8] It weighs 7–9 kg.

The adult males of the two species of the genus *Bradypus* have a speculum, nonexistent in the maned sloth.

Three-toed sloths have not adapted well to captivity, even though they are often captured and sold in the illegal pet trade.

FEEDING

Sloths of the genera *Scaeopus* and *Bradypus* are strict folivores. Their habitat is limited to primary and secondary forests. *Choloepus* spp. feed on fruits, flowers,

TABLE 24.1. Species of sloths

Famíly	Genus	Species	Common Name
Bradypodidae	*Bradypus*	*Bradypus tridactylus*	Pale-throated three-toed sloth
		Bradypus variegatus	Brown-throated three-toed sloth
	Scaeopus	*Scaeopus torquatus*	Maned sloth[a]
Choloepidae	*Choloepus*	*Choloepus didactylus*	Linné's two-toed sloth
		Choloepus hoffmanni	Hoffmanni two-toed sloth

[a] Classified as rare by IUCN (1974) and included in the Brazilian List of Endangered Animals (see reference 1).

leaves,[4] and probably small animals. In captivity they accept meat, dry dog food or primate food, and raw or cooked eggs. Captive animals should also be given green vegetables.

Three-toed sloths have traditionally been thought to feed mainly, if not exclusively, on embaúba leaves (*Cecropia* spp.), but recent studies demonstrated that several other species of leaves are also consumed by the sloths, such as figs (*Ficus* spp.) and cashew (*Anacardium excelsum*).[5] In the absence of these preferred species, captive sloths were offered bamboo and grape leaves. Sloths accepted the new leaves. All leaves offered should be fresh. Leaves kept in a refrigerator for even a short time dehydrate, decreasing palatability.

Because different sloths have different food preferences, a variety should be offered daily. Captive animals consume large quantities of food (250–400 grams of new leaves each). Normally, these mammals do not drink water, obtaining necessary liquid directly from food. However, two-toed sloths should have water available at all times.

HOUSING

Sloths are strictly arboreal, going down to the ground only to move to another area if branches don't meet or to defecate.[3] Three-toed sloths defecate in average intervals of 4–8 days. They use a vertical trunk of small diameter to move down to the ground and to return. Animals in a semifree enclosure of 400 m^2 used only three sites for defecation. For resting, they mainly use forks, sitting down in these, maintaining contact with one of the branches by an arm or a leg, while the dorsal surface is supported against the other branch. They prefer to rest in large trees, containing several lianas (hanging vines), or with large amounts of leaves.

Captive sloths need areas of both shade and sun. In the morning, captive sloths may spend some hours exposed to the sun, especially after cold nights; even so, they avoid intense sunlight. Screened enclosures allow the animals to use the whole available area, but their use can make it difficult to contain animals. Adult males may fight and should be kept isolated.

Three-toed sloths (*Bradypus* spp.) are primarily diurnal, with minor night activity, whereas *Choloepus* spp. are strictly nocturnal.[3] In Rio Zoo, *Choloepus* are maintained in an inverted photoperiod, using red lamps during the visitation hours. *Choloepus* are also housed with other mammals, such as fruit bats (*Artibeus* spp.) and marsupials (*Caluromys philander*). Two-toed sloths have been observed using wooden boxes as shelters. No problems have been encountered with cohabitation.

REFERENCES

1. Bernardes, A.T.; Machado, A.B.M.; and Rylands, A.B. 1990. Fauna brasileira ameaçada de extinção. Belo Horizonte, Fundação Biodiversitas.
2. Coimbra-Filho, A.F. 1972. Mamíferos ameaçados de extinção no Brasil. Anais da Academia Brasileira de Ciências 44(Suppl.):13–98.
3. Goffart, M. 1971. The Form and Function of the Sloth. New York, Pergamon Press.
4. Merrit, D. 1976. The nutrition of the edentates. International Zoo Yearbook 16:38–46.
5. Montgomery, G.G.; and Sunquist, M.E. 1978. Habitat selection and use by two-toed and three-toed sloths. In G.G. Montgomery, ed., The Ecology of Arboreal Folivores. Washington D.C., Smithsonian Institution Press, pp. 329–359.
6. Nowak, R.M. 1991. Walker's Mammals of the World. Baltimore, Johns Hopkins University Press.
7. Waage, J.K.; and Montgomery, G.G. 1976. Cryptoses cholepi: A coprophagus moth that lives on a sloth. Science 193:157–158.
8. Wetzel, R.M.; and Avilla-Pires, F.D. 1980. Identification and distribution of the recent sloths of Brazil (Edentata). Revista Brasileira de Biologia 40(4):831–836.

HUSBANDRY

Antônio Messias-Costa
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Sloths require large spaces for captive maintenance. The environment must provide all the necessary conditions for the maintenance of homeostasis, primarily proper temperature and humidity.

The enclosure must be high, have a solarium area, and if there are predators in the area, it must be protected by a screen of fine mesh. Branches in several positions (vertical, horizontal, and parallel), with platforms at different levels, as well as places of refuge should be provided for the animals. Bedrooms may be smaller, but should be brightly lit. Antagonistic reactions in Bradypodidae are rare and insignificant, although they may occur in feeding territories and among males in the reproduction phase.

Cloth bags or boxes are used to move sloths. A crossed branch alongside provides support to enable the animals to maintain their normal upside down position. For animals that will be handled continually, as for serum administration, the fingernails of the feet and hands should be trimmed.

Recently received animals from the field may have ticks and other arthropods in the fur. The volume occupied by the stomach is large, which may decrease after a few days in captivity, and the fur may become drier.

Animals maintained in inadequate conditions of temperature or humidity may have loss of fur, most frequently on the limbs. Temperature extremes should be avoided, and the ideal humidity should be above 60%. In Amazonia, the daily average body temperature of a captive *Bradypus tridactylus* juvenile (1 year old) was 33.5°C (outdoor temperature 29.5°C), with a maximum of 34.5°C and a minimum of 30.8°C (outdoor temperature was 18°C). An adult female in the same facility had an average temperature of 32.5°C, with a maximum of 33.2°C and a minimum of 30.4°C. When the outdoor temperature was 25°C, the inactive juvenile had a temperature of 32.7°C, which rose quickly to 33.2°C with activity. Enteric and respiratory disturbances may occur in stressed animals.

A small water tank aids thermoregulation. The tank must be placed close to the base of a branch and must be shallow and large enough for the animal to evacuate in it. Animals in nature or in semifree enclosures produce large amounts of feces (more than 150 g). Captive *Bradypus* may have shorter intervals between defecations. The water tank must be disinfected when feces are in it, approximately every 3-7 days. In the absence of an appropriate place, animals will defecate on cold concrete surfaces or soil areas, which is not sanitary.

MEDICINE AND NEONATAL CARE OF SLOTHS

Antônio Messias-Costa

TRAUMA

Sloths are well adapted to an arboreal system, nevertheless it is not uncommon for them to fall, sometimes with serious consequences. When they fall, they bend their bodies and protect their heads with their arms, and this, with their dense fur, lessens the risk of injury. There is documentation of a fall of an adult animal from the height of 12 m without serious injury. However, in another case, an adult female fell with her infant onto a concrete surface. The infant sustained no injury, but the mother's tongue and left arm were paralyzed. Recovery and recuperation took 3 months. During that period the mother was unable to chew leaves. She was given peeled bananas and other soft foods.

Conventional techniques of reducing fractures are applicable in sloths, although the prognosis may be poor. Sloths that fall onto unshielded electric wires may be seriously burned or killed.

RESTRAINT AND HANDLING

Three-toed sloths, when threatened, freeze as if paralyzed and are then easily manipulated by holding them from the back or by the arm extremities. Nets are also appropriate for physical capture, but snares, besides being unnecessary, may damage the cervical column, with serious consequences. Two-toed sloths are strongly agile and aggressive, and their bites can cause serious damage. They should be contained with nets and squeeze cages.

Chemical Restraint

Ketamine hydrochloride (Vetalar; Aveco Company) at 5-10 mg/kg intramuscularly (IM) may be used with good results. Diazepam (Valium; Roche) at 0.1-0.3 mg/kg IM may be used in addition to prevent the hypertonicity associated with ketamine use. The tiletamine hydrochloride plus zolazepam hydrochloride (Telazo; Parke Davis) at a dose of 5 mg/kg IM was efficient, and the effects lasted 30 minutes. Salivation was not observed.

DISEASES

The three-toed sloth is difficult to maintain in captivity, so little information is available about diseases. Malnutrition and dehydration are important considerations in sick sloths. Annually, more than 300 three-toed sloths that have been rescued from forest fires or confiscated from the illegal pet trade are brought to the Zoobotanical Garden of the Emílio Goeldi Museum in Belém, Amazonia; 70% have malnutrition associated with respiratory diseases. Acute edema of the lungs and bronchopneumonia were the main causes of death in 95% of the necropsied animals.

Clinical signs of disease include weight loss, ocular globe retraction, opaque and damp fur (as if there is intense sweating), and, in some cases, bilateral nasal discharge. In youngsters, dyspnea becomes evident, characterized by intermittent opening and closing of the mouth.

Constipation and tympanism are gastrointestinal problems seen frequently in sloths, almost always associated with stress factors. The clinical picture is characterized by excessive swelling of the abdomen that becomes rigid, with tympanic sounds, or with massive sounds in cases of impaction, frequently accompanied by respiratory distress. In serious cases of bloating, it is advisable to release the gas by abdominocentesis, using proper antiseptic technique.

A three-toed sloth in captivity evacuates every 3-5 days. While adapting to captivity, animals may not evacuate for as long as 10 days, and when they do defecate, the fecal material is dark with large amounts of mucus. If intervals between defecation exceed 7 days, it is advisable to administer a soft laxative after the last feeding of the day. This regimen should continue until that animal has adapted to the environment and its intestinal function has become regularized. The process may last as long as 3 weeks.

Parasitic Diseases

Sloths are hosts to a wide variety of external parasites such as lice, ticks, and algae. In the genera *Bradypus* and *Choloepus,* ticks identified as *Amblyoma geayi* have been described. Most of these parasites are well adapted and cause little pathology in natural conditions. However, in captivity, the host/parasite balance may become disturbed, requiring treatment.

Wounds must be cleansed and the dense fur clipped to avoid myiases. The anus must be examined carefully, because it is a preferential site for the deposition of fly eggs.

Endoparasites are rare.[2] Coccidia are frequently discovered in free-living edentates. *Eimeria choloepi* has been found in the two-toed sloth *Choloepus didactylus*.[2] However, in several verified cases there was no manifestation of disease.

Acute and fulminate toxoplasmosis has been described in *Bradypus tridactylus* in captivity, characterized by interstitial pneumonia and necrotic lesions in several organs. The source of infection was unclear (E. Tury and E.J. Gimeno, and A. Messias-Costa, personal communication, 2000). The evolution of these animals in biotopes that make it difficult to contact pathogens impedes the development of the mechanisms of resistance to parasites acquired by most other terrestrial mammals.

B. tridactylus and *Choloepus didactylus* are important reservoirs of *Leishmania* (V) *shawi*. Vectors are the sandflies *Lutzomia whitmani* and *Lutzomia umbratilli*s, which inhabit logs in the primary forests of Amazonia.[3]

THERAPY

Sloths have a low metabolic rate and a large rumenlike stomach that accounts for 30% of the body weight, which may require the reduction of systemic drug dosages by 25%. The low metabolic rate increases the time it takes for drugs to be cleared from the body. Thus, drugs should be given less frequently than indicated for mammals with higher metabolic rates.[1]

For treatment of ectoparasites, it is recommended that ticks be removed by hand and lice removed by means of a bath with deltamethrin shampoo. Treatment of respiratory infections must be based on the etiologic agent. Ampicillin, at a dosage of 30 mg/kg, every 8 hours for 8 days has been successful, and sulfamethoxasole/trimethoprim 0.5–1 mL, 12/12 h (Bactrim; Roche) have been effective. For weakened orphan cubs, it is recommended trihydrated amoxicillin given at dosages of 25–75 mg in the food to prevent respiratory diseases. Treatment by injection may also be performed. For impaction in the three-toed sloth, milk of magnesia given by stomach tube has shown good results.

For trauma in an adult animal, the intramuscular injection of dexamethasone (Azium-Schering), with declining daily doses, beginning with 1.0 mL (200 mg), have been successful.

BREEDING AND REPRODUCTION

"Three-toed sloths in southeastern Brazil give birth in July to October. The young grasp the chest or abdomen of the mother by using hooked fingernails. Reproductive activity varies with the latitude, so that young become independent in the spring. Infants weigh about 150–250 g at birth, and in few days they begin to nibble the leaves ingested by the adult female. Young sloths begin to eat leaves at 4 weeks of age, but stay with mother for more than 5 months." (Carlos Esbérard)

The reproduction of sloths in captivity is rare and is extremely difficult to observe in the wild. Sloths are polyestrus, but little is known about the reproductive cycle.

Usually, they give birth every 3 years. Observations with captive *B. tridactylus* showed that births occurred during the months of April and May in Brazil. Heat ensued 30-40 days after the birth.

Three-toed sloths have sexual dimorphism. A male that has reached puberty acquires a brown hairless patch on its back, which is a glandular epithelium that secretes a yellowish substance with an ammoniacal scent. Two-toed sloths and maned three-toed sloths lack this patch. The penis in the adult animal is less than 1 cm in length, located ventral to the border of the anus, protected by a superior fold. The female possesses two tiny longitudinal lips on the border of the anus, which continue as a trough leading to the cervix of the uterus.

Estrus is characterized by high whistles emitted by the female to attract the excited male that then follows and protects her for 3 days. Coitus occurs throughout this period. To carry out copulation the male rests in dorsal decubitus, holding the pelvis of the female, which remains seated for 10-25 minutes.

The gestation period is 322 days. In the final trimester, the sloth becomes quieter, usually resting on a platform. Labor usually occurs in a vertical position with the arms fixed. As in many other mammals, the placenta is ingested by the mother. The presence of the male doesn't interfere with labor. Mothers suffering from malnutrition may abandon newborn cubs. In captivity, abandoned cubs may be accepted by another mother.

NEONATAL BEHAVIOR AND CARE

Newborn *B. tridactylus* weigh approximately 180 g and are 18 cm long. The coloration pattern is like that of the adult, although in much clearer tones. The eyes are open at birth. They are also born with teeth, favoring feeding with leaves in the first days of life. They also seek frequent oral contact with the mother, possibly to create a necessary intestinal flora for the digestion of leaves or replacement of baby liquids. Young suckle from the single pair of mammary glands, located on the chest, usually six times per day. Suckling time decreases as ingestion of leaves increases. A young sloth clings to its mother constantly for as long as 3-4 months, initially urinating and defecating on the mother's body, but later on using the same substratum as the mother for excretion.

By the end of the first month of life, sloths weigh more than twice their birth weight and have grown 5 cm. The nursing period lasts about 6–8 months. At six months of age they weigh approximately 715 g and are 33 cm long. Close association with the mother continues after weaning. The paternal relationship is indifferent, although there is no rejection by the father when he has access to the cub. By 1 year of age, the cub is independent of the mother, although occasional physical contacts with the parents may occur.

In addition to being suckled by the mother, young captive sloths may be given supplemental feedings with evaporated milk and a multivitamin mixture. Infants may be fed with a syringe twice a day, given 2–5 mL at each feeding. This amount should be gradually reduced as the infant grows and begins to eat leaves.

An orphaned sloth infant is usually weak in addition to other problems. It may be advisable to give orphaned infants 0.25 mL of trihydrated amoxicillin in the formula to prevent respiratory infections. Problems of constipation and tympanism may be resolved with 2–4 drops of milk of magnesia. This regimen was followed with a pale-throated three-toed sloth at Emilio Goeldi Park, the first successful rearing of this species in captivity. This experience shows that it is possible to breed this species if the biology is understood and special care is given.

ACKNOWLEDGMENTS

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REFERENCES

1. Divers, B.J. 1978. Edentates. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 439–448.
2. Lainson, R.; and Shaw. 1982. Coccidia of Brazilian edentates. *Eimeria cyclopei n. sp* from two-toed sloth, *Choloepus didactylus, Linn)*. Systematic Parasitology, The Hague 4:269–278.
3. Silveira, F.T.; Lainson, R.; Brito, A.C. 1997. Leishmaniose tegumentar americana. In R.N.C. Leão, coord., Doenças Infecciosas e Parasitárias—Enfoque Amazônico. Belém, Cejup, IEC, pp. 619–630.

GENERAL MEDICINE

Lilian de Stefani Munao Diniz

Although edentates are usually considered as a single group for veterinary purposes, it is important to consider them separately here, because anteaters, armadillos, and sloths have different characteristics of anatomy, physiology, behavior, and requirements for captive care, and consequently have varying susceptibility to veterinary problems. Although edentates have been studied and important medical and biological information collected, only a few papers on pathological conditions and management of these mammals in zoos have been published.[13-15,19,28] A few reports have been published on clinical problems, etiological agents, treatment protocols, and management problems of anteaters,[12] armadillos,[11, 18] and sloths[9] in Brazilian zoos. Factors influencing the onset of disease in edentates are listed in Table 24.2.

Surgical Conditions

- Rectal prolapse—Rectal prolapse may occur after severe intestinal problems causing diarrhea in armadillos and anteaters. Cystic calculi were responsible for recurrent rectal prolapse in a sloth.
- Fractures—Internal fixation of limb fractures is recommended in combination with pressure bandages for maximum support, because the stout, short limbs do not lend themselves to external fixation. Femoral head osteotomy has been reported in the giant anteater.[28]
- Onychectomy—Surgery may be required to remove the remaining part of a broken claw and to suture the surrounding tissue in giant anteaters.

ZOONOSIS

ARMADILLOS The common habit of maintaining armadillos in homes as pets increases the incidence of zoonosis in Latin American people.[4] Nine-banded

TABLE 24.2. The prevalence factors that may influence the onset of disease in edentates in captivity

	Diseases (%)		
Factors	Armadillos	Anteaters	Sloths
Origin			
Zooborn	8.8	11.2	—
Donation	91.1	88.7	100.0
Climate			
Winter	20.7	27.5	32.5
Spring	33.1	18.8	24.0
Summer	26.0	26.0	22.9
Autumn	19.8	27.5	20.5
Enclosure			
Quarantine	84.0	64.7	96.3
Exhibition	15.0	35.2	3.6
Captive period			
0–6 mo	92.2	78.0	96.3
6 mo–2 yr	0.9	12.7	2.4
2–5 yr	2.3	5.1	1.2
> 5 yr	4.6	4.1	—

Source: See references 9, 11, and 12.
mo, month; y, year.

armadillos are susceptible to a variety of human diseases including leprosy, Chagas' disease, relapsing fever, trypanosomiasis, exanthematous and murine typhus, schistosomiasis, and infection with *Nocardia brasiliensis*. Deep mycosis caused by *Paracoccidioides brasiliensis* is the most important human systemic mycosis in South America. It was demonstrated recently that armadillos are a sylvatic host to this fungus because it is isolated in a high prevalence of this disease in free-living animals.[2] Sporothrichosis (*Sporothrix schenckii*) is also an important deep mycoses, and cutaneous lesions with nodular lymphangitis and multiple portals of infections have been reported in humans.

The development of commercial wildlife farming and increased consumption of meat from wildlife may increase the health risk to human consumers from *Salmonella* spp. and *Campylobacter* spp.[1]

SLOTHS Sloths are natural reservoir hosts for some internal parasites that may affect humans and other mammals. Such blood parasites as *Babesia choloepi, Chabfilaria jonathani, Trypanosoma leeuwenhoeki,* and *Trypanosoma rangeli* have been reported. The two *Trypanosoma* species are the most widely distributed hematozoa in the Amazon region.[24]

Histoplasmosis was isolated from sloths in the Amazon region of Brazil. Latin American wild mammals are exposed and susceptible to this important deep mycosis in human beings.[7]

IMMUNIZATION

Although edentates are susceptible to infectious diseases, there is no vaccination program for this group. It has been reported that tuberculosis, with frequent losses of anteaters, ceased after BCG vaccination.[22]

ARMADILLOS

Behavior

Constant rubbing against the wire or the wall of the enclosure has been observed in enclosures too small to meet the animals' space requirements. Such repetitive patterns may cause severe injuries on the carapace, nostrils, and toes.

Ammonia burns may occur if a large litter is housed inadequately on a wooden floor. Ammonia is produced in urine, and excessive exposure to urine results in erythemic conjunctivitis, lacrimation, and raw ulcers of the contact surfaces of the feet and/or abdomen. Treatment is correction of the environment plus bathing with surgical soap, followed by topical application of antiseptic and/or antibiotic ointments containing vitamin A. Ophthalmic solution is indicated topically for eye problems.

Impaction of the alimentary canal is an important internal problem encountered from consumption of inappropriate items, such as bedding material. Armadillo litters may suffer this kind of distress if sawdust, shavings, and corncobs are used in the enclosure. Clinical signs are vague, but include anorexia, prostration, abdominal distension, and a reduced amount or complete absence of feces. The presumptive diagnosis is confirmed by radiographic examination of the abdomen. Treatment includes oral administration of vegetable oil in food if they are eating, otherwise 20–50 mL of mineral oil may be administered through a stomach tube. If oil does not produce the desired effect in 6–12 hours, a gastrotomy should be considered to manually remove the obstructing mass.

Injuries

Superficial wounds and fractures have been observed in captive armadillos. They usually occur as a result of contact with the wires of enclosures, attempts to burrow in inappropriate surfaces, or during mechanical immobilization for clinical examination, capture, or transportation. The most frequently injured body sites are the mouth, nostrils, nails, digits, interdigital membranes, and scales of the tail and integument. Open wounds or fresh blood may tempt cage mates to cannibalism.

TABLE 24.3. Major clinical disorders in armadillos in captivity

Clinical Problems	*n* (%)[a]	Associated Factors
Injuries	62 (28.5)	Wounds (59), fractures (3)
Digestive	39 (17.9)	Enteroparasites (12), diarrhea (17), hepatic processes (4), tongue inflamation (2), vomiting (1), intestinal impactation (1), rectal prolapse (1), foreign body (1)
Respiratory	33 (15.2)	Pneumonias (32), nose bleeding (1)
Nutritional	29 (13.3)	Nutritional deficiency (25), anemia (4)
Skin	8 (3.6)	Ectoparasites (7), dermatitis (1)
Septicemia	4 (1.8)	Infectious sign (4)
Nervous	3 (1.3)	Convulsions (3)
Urinary	2 (0.9)	Blood in urine
Problems related to extreme climate	2 (0.9)	Winter (1), summer (1)
Circulatory	1 (0.4)	Heart insufficiency
Ophthalmic	1 (0.4)	Conjunctivitis
Behavior	1 (0.4)	Cannibalism
Inconclusive	32 (14.7)	—

Source: See reverence 11.
[a]*n* = 217 (total number of clinical disorders).

TABLE 24.4. The prevalence of etiological agents and respective treatments in captive armadillos

Pathogens	Agents	Treatments [a]
Enteric pathogens		
Protozoan 13%	*Entamoeba* 5%, *Coccidia*	Metronidazole, 50 mg/kg/2 × daily 5–7 days or tinidazole, 50 mg/kg/2 × daily, 5 days
Nematodes 66.6 %	*Ancylostoma, Strongyloides* and *Trichuris*	Thiabendazole, 50–100 mg, 2 × daily, 5 days; mebendazole, 15 mg/kg, 1 × daily, 5 days
	Ascaris	Levamisole, 10 mg/kg, or piperazine, 80–100 mg/kg, 1 × daily, 2 days, repeated after 7 days
Cestodes 1.8%	(Not classified)	Praziquantel, 5 mg/kg, 1 day
Bacteria 18.5%	*Salmonella, Escherichia coli, Enterobacter aerogenes, Acinetobacter hinshawii*	Chloramphenicol, 25–75 mg/kg/2 × daily, 10 days; trimethoprim + sulfamethoxazole 0.5 mL/ kg/ 2 × daily, 5–7 days
Respiratory pathogens		
Bacteria 90%	*Staphylococcus and Streptococcus*	Chloramphenicol, 50–100 mg/kg/2 × daily, 10 days; ampicillin, 50 mg/kg/2 × daily, 5–10 days; oxytetracyline, 20 mg/kg, 5 days; kanamycin, 10–20 mg/kg/ 2 × daily, 10 days
Skin pathogens		
Arthropoda 5%	*Amblyomma*	Benzil benzoate, DDVP [b] (topical)
Bacteria 4%	*Staphylococcus* and *Streptococcus*	Chloramphenicol, 50–100 mg/kg/2 × daily, 10 days; oxytetracyline, 20 mg/ kg, 5 days

Source: See reference 11.
[a] Most commonly employed drugs repeated as necessary.
[b] Dimethyldichorovinyl phosphate.

Infectious Diseases

A study of 113 armadillos in captivity included 55 nine-banded armadillos (*D. novemcinctus*), 48 yellow armadillos (*Eufractes sexcinctus*), 5 *Cabassous* spp., 4 *Tolypeutis* spp., and 1 giant armadillo (*P. maximus*). The most common health problems resulted from injury (28.5%), digestive system upsets (17.9%), respiratory system infections (15.2%), and nutritional deficiencies (13.3%).[11] Clinical problems are summarized in Table 24.3. The prevalence of the primary etiological agents and the recommended treatments are listed in Table 24.4.

A histopathological study on five species of Dasypodidae reported the occurrence of toxoplasmosis, sarcosporidiosis, coccidiosis, thromboembolism, and granuloma.[18]

PERIONYCHITIS Inflammation of the digital cuticle is a serious and common problem in armadillos. Good results may be obtained by spraying with an antiseptic solution and applying an antibiotic solution topically three times a day. Treatment should include debridement and amputation of the necrotic tissue. Amputation of a digit or tail is necessary if osteomyelitis occurs. Surgical wounds should be protected with antibiotic ointment and a pressure bandage. Supportive parenteral antibiotics should be administered to prevent secondary systemic infection. Armadillos are often victims of cat or dog bites. Lacerations of the carapace may be sutured with artificial suture material.[14,28]

LEPROSY *Mycobacterium leprae* is the most important contagious infection transmitted from armadillos to humans. It has been found in 10% of the armadillos of the southern states of the United States, but no infected wild armadillos have been found in South America.[19] Some authors explain that this infection became established in certain populations after a few armadillos initially became infected through contact with fomites derived from people with leprosy. Armadillos are naturally susceptible to leprosy, and for this reason it is used as a laboratory animal model for this infection in humans.

After inoculation, armadillos develop leprous lesions in the skin, and the most conspicuous lesions are observed in the liver and spleen. In addition to these sites, lesions develop in the lung, stomach, and kidney, which are not seen in human cases. These changes may be seen at necropsy.

Rifabutin and rifampin may be administered daily at doses of 6 mg/kg body weight per day, for 8 weeks. These drugs have proved to have bactericidal action on *M. leprae*.[23] The differential diagnosis of any skin ulceration or granuloma should include consideration of leprosy.

PARACOCCIDIOIDOMYCOSIS *Paracoccidioides brasiliensis* is the most common deep mycosis in human beings in Brazil. It was first isolated from armadillos from the Amazon region where this mycosis rarely occurs. Recently this disease was reported as a high-prevalence infection in free-living armadillos from Botucatu, São Paulo State, a hyperendemic area of Brazil. Abscesses in the lungs, spleen, liver, and mesenteric lymph nodes characterize the disease.[2]

Because the soil is considered the natural reservoir of this fungus in South America, terrestrial wild mammals are more likely to contract this infection than arboreal species, borne out by rates of 82.9% and 22.5%, respectively.[8] Armadillo habits of coprography and soil burrowing behavior make them a possible potential reservoir of this infection in South America. The prevalence of paracoccidioidomycosis in an endemic area of Colombia was higher in persons with close contact with armadillos than in persons who had no contact.[4]

Noninfectious Diseases

Traumas, malnutrition, granulomas, neoplasms, and impacted colon are reported frequently. Anesthetic shock requires special attention because it is widely reported in large numbers of armadillos, indicating a high susceptibility.[15]

Adult specimens of such endangered species as the giant armadillo (*P. maximus*), greater naked-tail armadillo (*Cabassous* spp.) and three-banded armadillo (*Tolypeutes* spp.) captured from the wild usually live only a few months in zoos. They are unable to adapt to captivity, suffering from depression, progressive loss of appetite, loss of weight, and finally death.[11]

Dietary diarrhea is seen in young or newly shipped armadillos, but once they have adjusted to a captive diet, the problem clears up. Changing the management is usually all that is necessary.

Parasitic Diseases

INTERNAL PARASITES Armadillos harbor many parasites, but parasitic disease is rare. In 113 armadillos the author found 33.3% harbored *Ancylostoma* spp., 30.5% *Strongyloides* spp., 25% *Ascaris* spp., 11.1% *Trichuris* spp., and 1.8% cestodes. Protozoa (coccidia and entamoeba) represented 13%.[11]

EXTERNAL PARASITES External parasites include flies, ticks, and fleas. See Figure 24.1.

ANTEATERS

The major health problems found in 103 anteaters, lesser anteater or tamandua (*Tamandua tetradactyla*), and giant anteater (*M. tridactyla*) kept in zoos were studied. The most common clinical disorders observed involved digestive system upsets (26%), nutritional deficiencies (20%), injuries (15.5%), respiratory system infections (10%), skin problems (7%), and problems of the circulatory system (4.5%),[12] see Table 24.5. The prevalence of the etiological agents identified and the respective treatment are listed in Table 24.6.

Infectious Diseases

PERIONYCHITIS Infections in the digital cuticles may occur as a consequence of trauma. This kind of lesion prevents the animal from opening anthills to eat

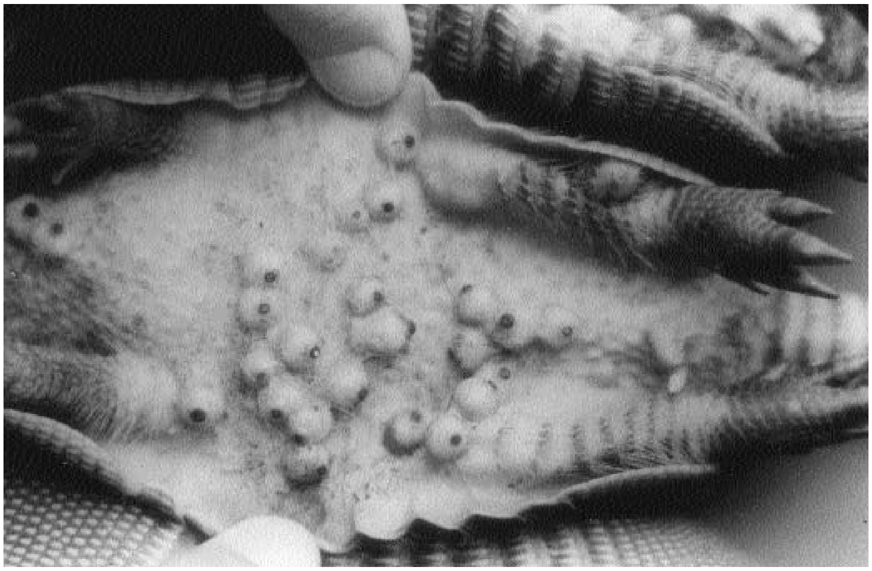

FIGURE 24.1. *Tunga penetrans* (flea) in the ventral region of nine-banded armadillos. The female burrows into the skin and becomes swollen with eggs and the blood of the host until she reaches the size of a small pea. (Courtesy of L. Diniz.)

TABLE 24.5. Major clinical disorders in anteaters in captivity

Clinical Problems	*n* (%) [a]	Clinical Problems	*n* (%) [a]
Digestive	52 (26.0)	Circulatory	9 (4.5)
Enteroparasites	27 (13.5)	Insufficiency	3 (1.5)
Enteritis	18 (9.0)	Myocarditis	3 (1.5)
Nonspecific diarrheas	4 (2.0)	Internal hemorrhage	3 (1.5)
Liver problems	2 (1.0)		
Foreign body	1 (0.5)	Reproductive	2 (1.0)
		Abortion	1 (0.5)
Nutritional	40 (20.0)	Mastitis	1 (0.5)
Poor absorption	23 (11.5)		
Deficiency	17 (8.5)	Septicemia	2 (1.0)
Injuries	31 (15.5)	Urinary diseases	2 (1.0)
Respiratory	20 (10.0)	Puerperal diseases	1 (0.5)
Pneumonia	20 (10.0)		
		Ophthalmologic	1 (0.5)
Skin	14 (7.0)		
Ectoparasites	5 (2.5)	Inconclusive	26 (13.0)
Alopecia or pruritus	5 (2.5)		
Dermatitis	3 (1.5)		
Abscess	1 (0.5)		

Source: See reference 12.
[a]*n* = 200 (total number of clinical disorders in 103 anteaters).

the insects. Local and supportive parenteral antibiotics should be administered.

Parasitic Diseases

INTERNAL PARASITES Nematodes represent the main intestinal parasites found (40%): *Trichuris* 28%, *Strongyloides* 11%, and *Ascaris* 1%.[12]

SLOTHS

A 20-year retrospective study of disease prevalence was carried out with 51 sloths (34 three-toed sloths and 17 two-toed sloths) in captivity. A total of 81 clinical disorders were detected, including nutritional (45.7%), digestive (12.3%), and respiratory (12.3%) problems and

TABLE 24.6. The prevalence of etiological agents and respective treatments in captive anteaters

Pathogen	Agents	Treatments [a]
Enteric pathogens		
Protozoa (16%)	*Eimeria* 10%, *Entamoeba* 5%, *Giardia* 1%	Tinidazole, 20–50 mg/1 ×/5–7days
Nematoda (40%)	*Trichuris* (28%), *Strongyloides 11%, Ascaris 1%*	Mebendazole, 100 mg/2 × daily, 7 days Levamisole, 100 mg/2 × daily, 10 days
Cestoda (8%)	(Not identified)	Mebendazole, 200 mg/2 × daily, 10 days
Acanthocephala (1%)	(Not identified)	—
Bacteria (9%)	*Salmonella enteritidis, Salmonella cholerasuis, Escherichia coli, Enterobacter aerogenes*	Chloramphenicol, 100 mg kg/2 × /7 days or Trimethoprim + sulfamethoxazole, 1 mL/kg, 2 × daily, 7days
Respiratory pathogens		
Bacteria	*Pneumococcus, Staphylococcus, Streptococcus*	Ampicillin, 20 mg/ kg/2 × daily, 10 days; Kanamycin, 20 mg/2 × daily, 10 days
Skin pathogens		
Arthropoda (5%)	*Otodectis, Sarcoptes, Amblyomma*	Coumafos, Diazinon Benzyl benzoate , DDVP [b] (topical solution)
Bacteria (4%)	*Staphylococcus, Streptococcus*	Ampicillin, 10 mg/kg/2 × daily, 7days, Chloramphenicol, 20 mg/kg/2 × daily, 7 days

Source: See reference 12.
[a] Most employed drugs repeated as necessary.
[b] Dimethyldichlorovinyl phosphate.

injuries (6.1%) (see Table 24.7). Diarrhea, pneumonia, and nutritional deficiency may occur concurrently, affecting most animals during the quarantine period.[9]

Three-toed sloths and two-toed sloths may suffer when temperatures drop below 12°C. The average annual temperature range in the sloth's tropical forest habitat is 21–32°C, and most successful exhibits maintain a temperature of 28°C with approximately 60% humidity.[13]

Infectious Diseases

Sloths have no unique infectious diseases.

Parasitic Diseases

EXTERNAL PARASITES Algae is found on the hair coat of wild-caught sloths, giving a greenish aspect to the hair. This fact suggests that sloth fur provides a good substrate for algae growth in rain forest habitat. Bathing with copper sulfate solution is recommended, but the individuals will require chemical restraint to permit such treatment.[28]

REFERENCES

1. Adesiyun, A.A.; Seepersadsingh, N.; Inder, L.; and Caesar, K. 1998. Some bacterial enteropathogens in wildlife and racing pigeons from Trinidad. Journal of Wildlife Diseases 34(1):73–80.
2. Bagali, E.; Sano, A.; Coelho, K. I.; Alquati, S.; Miyaji, M.; De Pires, C.Z.; Gomes, G.M.; Franco, M.; and Montenegro, M.R. 1998. Isolation of *Paracoccidioides brasiliensis* from armadillos (*Dasypus novemcinctus*) captured in an endemic area of paracoccidioidomycosis. American Journal of Tropical Medicine and Hygiene 4:505–512.
3. Botelho, J.R.; Linardi, P.M.; and Encarnação, C.D. 1989. Interrelationships between Ixodidae (Acari) and Edentata hosts from Serra da Canastra, Minas Gerais State, Brazil. Memorias do Instituto Oswaldo Cruz 84(1):61–64.
4. Cavid, D.; and Restrepo, A. 1993. Factors associated with *Paracoccidioides brasiliensis* infection among permanent residents of three endemic area in Colombia. Epidemiology and Infections 111(1):121–133.
5. Correa, A.R.; Vahia, L.A.M.; Siqueira, B.R.; and Huggings, D.W. 1998. List of reservoir hosts of Chagas' disease. Revista Brasileira de Medicina 55(6):414–415, 418, 420.
6. Costa, E.O.; and Diniz, L.M.D. 1999. Evidence of *Nocardia asteroides* infection in Latin American wild mammals. Journal of Zoo and Wildlife Medicine (in press).
7. Costa, E.O.; Diniz, L.M.D.; Fava Netto, C.; Arruda, C.; and Dagli, M.L.Z. 1994. Epidemiological study of sporotrichosis and histoplasmosis in captive Latin American wild mammals. Mycopathologia 125:19–22.
8. Costa, E.O.; Diniz, L.M.D.; Fava Netto, C.; Arruda, C.; and Dagli, M.L.Z. 1995. Delayed hypersensitive test—paracoccidioidin in captive Latin American wild mammals. Journal of Medical and Veterinary Mycology (Sabouraudia) 33:39–42.
9. Diniz, L.S.M.; and Oliveira, P.M.A. 1999. Clinical problems of sloths (*Bradypus* sp and *Choloepus* sp) in captivity. Journal of Zoo and Wildlife Medicine 30(1):76–80.

TABLE 24.7. Major clinical disorders in sloths in captivity

Clinical Problems	*n* (%)	Disorders
Nutritional	37 (45.7)	Weakness, dehydration, thickness, emaciation
Respiratory	10 (12.3)	Pneumonia
Digestive	10 (12.3)	Enteroparasites (3), enteritis (3), stomach impaction (2), gastric dilatation (1), hepatic dysfunction (1)
Injury	5 (6.1)	Wounds (4), fracture (1)
Problems related to extreme climate	4 (4.9)	Hypothermia 4 (4.9)
Skin	4 (4.9)	Ectoparasites
Septicemia	1 (1.2)	General infection
Urinary	1 (1.2)	Urine changes
Circulatory	1 (1.2)	Endocarditis

Source: See reference 9.
[a]*n* = 81 (total number of clinical disorders in 51 slots).

10. Diniz, L.S.M. 1996. *Tunga penetrans* (Siphonaptera, Arthropoda) em tatú [*Tunga penetrans* (Siphonaptera, Arthropoda) in armadillos]. In 25° Anais do Congresso Panamericano de Ciências Veterinárias. Campo Grande, MS, Brasil, Pan American Association of Veterinary Sciences, p. 86.
11. Diniz, L.S.M.; Costa, E.O.; and Oliveira, P.M.A. 1997. Clinical disorders in armadillos (Dasypus, Edentata) in captivity. Journal of Veterinary Medicine B 44(10):577–582.
12. Diniz, L.S.M., Costa, E.O.; and Oliveira, P.M.A. 1995. Clinical disorders observed in anteaters (Myrmecophagidae, Edentata) in captivity. Veterinary Research Communication 19(5):409–415.
13. Divers, B.J. 1986. Edentata. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 622–630.
14. Gillespie, D.S. 1993. Edentata: Disease. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, Vol. 3. Current Therapy. Philadelphia, W.B. Saunders, pp. 304–309.
15. Griner, L.A. 1983. Pathology of Zoo Animals. San Diego, California, Zoological Society of San Diego, pp. 382–387.
16. Lainson, R.; and Shaw, J.J. 1991. Coccidia of Brazilian mammals: *Eimeria marajoensis* n. sp. (Apicomplexa: Eimeriidae) from the anteater, *Tamandua tetradactyla* (Xenarthra: Myrmecophagidae). Journal of Protozoology 38(1):28–30.
17. Lindsay, D.S.; McKown, R.; Upton, S.J.; McAllister, C.T.; Toivio Kinnucan, M.A.; Veatch, J.K.; and Blagburn, B.L. 1996. Prevalence and identity of *Sarcocystis* infections in armadillos (*Dasypus novemcintus*). Parasitology 82(3):518–520.
18. Maccio, O.A.; Rott, M.I.O.; Resoagli, E.H.; Millan, S.G.; and Gallardo, M.E.C. 1988. Patologias natural y adquirida en cautividad del armadillos (Edentata: Dasypodidae) [Pathology in free-living and captive armadillos]. Analisis Anatomopatologicas Veterinaria Argentina 5(43):218–227.
19. Montgomery, G.G. 1985. The Evolution and Ecology of Armadillos, Sloths and Vermilinguas. Washington D.C., Smithsonian Institution Press.
20. Naiff, R.D.; Barrett, T.V.; Naiff, M.F.; Ferreira, L.C.L.; and Arias, J.R. 1996. New records of *Histoplasma capsulatum* from wild animals in the Brazilian Amazon. Revista do Instituto de Medicina Tropical de São Paulo 38(4):273–277.
21. Roth, E.E. 1970. Leptospirosis. In J.W. Davis, ed., Infectious Diseases of Wild Mammals. Ames, Iowa, Iowa State University Press, pp. 125.
22. Ruempler, G. 1982. Edentata. In H.G. Klös and E.M. Lang, eds., Handbook of Zoo Medicine. New York, Van Nostrand Reinhold, pp. 307–310.
23. Sasaki, N; Kawatsu, K.; Tsutsumi, S.; Gidoh, M.; Nakagawa, H.; Kashiwabara, Y.; Matsuki, G.; and Endo, H. 1997. Pathological investigation of armadillos infected with *Mycobacterium leprae*. Japanese Journal of Leprosy 66(3):227–235.
24. Shaw, J. J. 1985. The hemoflagellates of sloths, vermilinguas (anteater), and armadillos. In G.G. Montgomery, ed., Ecology of Armadillos, Sloths, and Vermilinguas. Washington D.C., Smithsonian Institution Press, pp. 279–292.
25. Spinosa, H.S.; Górniak, S.L.; and Bernardi, M.M. 1999. Farmacologia Aplicada à Medicina Veterinária, 2nd Ed. Rio de Janeiro, Guanabara Koogan, pp. 396–404, 444–452.
26. Valente, V.; Valente, A.S.; Noireau, F.; Carrasco, H.J.; and Miles, M.A. 1998. Chagas disease in the Amazon basin: Association of *Panstrongylus geniculatus* (Hemiptera: Reduviidae) with domestic pigs. Journal of Medical Entomology 35(2):99–103.
27. Vasconcelos, P.F.C.; Travassos da Rosa, J.F.S.; Travassos da Rosa, A.P.A.; Dagallier, N.; Pinheiro, E.P.; and Sá Filho, G.C. 1991. Epidemiology of arbovirus encephalitis in Brazilian Amazonia. Revista do Instituto de Medicina Tropical de São Paulo 33(6):465–476.
28. Wallach, J.D.; and Boever, W.J. 1983. Edentates. In J.D. Wallach and W.J. Boever, ed., Diseases of Exotic Animals—Medical and Surgical Management. pp. 613–629.

25 Order Primates (Primates)

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BIOLOGY OF THE CEBIDAE

Anthony B. Rylands

New World primates belong to the infraorder Platyrrhini, a name that relates to their flat, outward-facing nostrils. Their taxonomy has undergone considerable change over the last two decades, stimulated in large part by the extensive revision of the callitrichids by Hershkovitz[4] and his subsequent reviews of the genera *Aotus, Saimiri, Chiropotes, Pithecia, Cacajao,* and *Callicebus.*[5-10]

Considerable attention has also been given to the phylogeny and taxonomy at family and subfamily levels, principally through morphological studies,[16,17] but also more recently using chromosome and molecular genetics.[3,21,22] Cytotaxonomy is also becoming increasingly important for systematics at the species and subspecies level (see, for example, the revision of *Aotus* by Hershkovitz[5]).

Over the last decade, a phylogenetic approach has identified three major groups or clades considered distinct at the family level: Cebidae (the marmosets and tamarins, along with the squirrel monkeys and capuchin monkeys), Atelidae (the howling monkeys, spider monkeys, woolly monkeys, and muriquis), and the Pitheciidae (the uakaris, sakis, and titi monkeys).[20] Both morphological and molecular genetic studies have agreed with this classification except in the case of the night monkey, *Aotus,* which is increasingly seen as a very early platyrrhine offshoot and perhaps even worthy of a separate family, the Aotidae. For convenience, however, the traditional division of these genera in two families is maintained: Callitrichidae (marmosets and tamarins) and Cebidae (the remaining genera).

Physically and taxonomically, the monkeys of the family Cebidae are a diverse group (see Table 25.1). There are 11 genera, 63 species, and 149 taxa ranging in size from 1 kg (*Saimiri*) to 15 kg (*Brachyteles*), and extending in their geographical distribution south from southern Mexico through Central America, the Amazon and the cerrado or bush savanna in Brazil, to the Atlantic forest in eastern Brazil and northeastern Argentina, and Paraguay, and to the chaco in Bolivia, Paraguay, and northwestern Argentina.

The small- to medium-sized cebids include five species (12 taxa) of squirrel monkeys (*Saimiri,* Central America and Amazonia), six species (33 taxa) of capuchins (*Cebus,* widespread), 14 species (26 taxa) of titi monkeys (*Callicebus,* Amazonia, cerrado, chaco, and the Brazilian Atlantic forest), 10 species (12 taxa) of night monkeys (*Aotus,* widespread excepting the Atlantic forest), five species (seven taxa) of saki monkeys (*Pithecia,*

TABLE 25.1. A listing of the species of the family Cebidae

Family Cebidae	Common Name
Aotus (Illiger 1811)	Night monkeys, owl monkeys, douroucouli
Gray-Neck Species Group	
A. lemurinus (I. Geoffroy 1846)	Colombian or lemurine night monkey
A. vociferans (Spix 1823)	
A. trivirgatus (Humboldt 1812)	Douroucouli, owl monkey, night monkey
A. brumbacki (Hershkovitz 1983)	Brumback's night monkey
A. hershkovitzi (Ramirez-Cerquera 1983)	Hershkovitz's night monkey
Red-Neck Species Group	
A. miconax (Thomas 1927)	Andean night monkey
A. nigriceps (Dollman 1909)	Black-headed or Peruvian night monkey
A. infulatus (Kuhl 1820)	Feline night monkey
A. azarai (Humboldt 1812)	Azara's night monkey
A. nancymaae (Hershkovitz 1983)	Ma's night monkey
Callicebus (Thomas 1903)	Titi monkeys
***Callicebus modestus* Group**	
C. modestus (Lönnberg 1939)	
***Callicebus donacophilus* Group**	
C. donacophilus (D'Orbigny 1836)	
C. olallae (Lönnberg 1939)	Beni titi monkey
C. oenanthe (Thomas 1924)	Andean titi monkey
***Callicebus moloch* Group**	
C. cinerascens (Spix 1823)	
C. hoffmannsi (Thomas 1908)	Hoffmann's titi monkey
C. moloch (Hoffmansegg 1807)	
C. brunneus (Wagner 1842)	
C. cupreus (Spix 1823)	
C. caligatus (Wagner 1842)	
C. dubius (Hershkovitz 1988)	
C. personatus (É. Geoffroy 1812)	Masked titi
Callicebus sp. (Kobayashi and Langguth 1999)[a]	
***Callicebus torquatus* Group**	
C. torquatus (Hoffmansegg 1807)	Collared titi, widow monkey
Saimiri (Voigt 1831)	Squirrel monkeys
***Saimiri boliviensis* Group**	
S. boliviensis (I. Geoffroy & de Blainville 1834)	Bolivian squirrel monkey
S. vanzolinii (Ayres 1981)	Black-headed squirrel monkey
***Saimiri sciureus* Group**	
S. sciureus (Linnaeus 1758)	Common squirrel monkey
S. oerstedi (Reinhardt 1872)	Central American squirrel monkey
S. ustus (I. Geoffroy 1843)	Golden-backed squirrel monkey
Cebus (Erxleben 1777)	Capuchin monkeys
Tufted Group	
C. apella (Linnaeus 1758)	Brown capuchin
C. xanthosternos (Wied 1820)	Buffy-headed capucuhin
Untufted Group	
C. albifrons (Humboldt 1812)	White-fronted capuchin
C. capucinus (Linnaeus 1758)	White-throated capuchin, white-faced capuchin
C. olivaceus (Schomburgk 1848)	Wedge-capped capuchin
C. kaapori (Queiroz 1992)	Ka'apor capuchin
Pithecia (Desmarest 1804)	Saki monkeys
P. pithecia (Linnaeus 1766)	White-faced saki
P. monachus (É. Geoffroy 1812)	Monk saki
P. irrorata (Gray 1842)	Gray's bald-faced saki
P. albicans (Gray 1860)	White saki, buffy saki
P. aequatorialis (Hershkovitz 1987)	Equatorial saki
Chiropotes (Lesson 1840)	Bearded sakis
C. albinasus (I. Geoffroy & Deville 1848)	White-nosed bearded saki
C. satanas (Hoffmannsegg 1807)	Bearded saki

(continued)

TABLE 25.1. A listing of the species of the family Cebidae—*continued*

Family Cebidae	Common Name
Cacajao (Lesson 1840)	Uakaris
C. calvus (I. Geoffroy 1847)	Bald uakari
C. melanocephalus (Humboldt 1811)	Black-headed uakari
Alouatta (Lacépède 1799)	Howling monkeys
A. seniculus (Linnaeus 1766)	Red howling monkey
A. arctoidea (Cabrera 1940)	Ursine howling monkey
A. sara (Elliot 1910)	Bolivian red howling monkey
A. belzebul (Linnaeus 1766)	Red-handed howling monkey
A. guariba (Humboldt, 1812)	Brown howling monkey
A. palliata (Gray 1848)	Golden-mantled howling monkey
A. coibensis (Thomas 1902)	Coiba Island howling monkey
A. caraya (Humboldt 1812)	Black-and-gold howling monkey
A. pigra (Lawrence 1933)	Black howling monkey
Ateles (É. Geoffroy 1806)	Spider monkeys
A. geoffroyi (Kuhl 1820)	Geoffroy's spider monkey
A. fusciceps (Gray 1866)	Brown-headed spider monkey
A. chamek (Humboldt 1812)	Black-faced black spider monkey
A. paniscus (Linnaeus, 1758)	Red-faced black spider monkey, Guiana spider monkey
A. marginatus (É. Geoffroy 1809)	White-whiskered spider monkey
A. belzebuth (É. Geoffroy 1806)	White-bellied spider monkey
A. hybridus (I. Geoffroy 1829)	Variegated spider monkey
Lagothrix (É. Geoffroy 1812)	Woolly monkeys
L. lagothricha (Humboldt 1812)	Humboldt's woolly monkey
L. flavicauda (Humboldt 1812)	Yellow-tailed woolly monkey
Brachyteles (Spix 1823)	Muriqui
B. arachnoides (É. Geoffroy 1806)	Southern muriqui
B. hypoxanthus (Kuhl 1820)	Northern muriqui

Source: Data from reference 18.
[a]Kobayashi and Langguth recorded the existence of a new *Callicebus* from the coast of the state of Sergipe, Brazil.[12]

Amazonia), two species (four taxa) of bearded sakis (*Chiropotes,* eastern Amazonia), and two species (six taxa) of uakaris (*Cacajao,* western Amazonia).[18] Some distinctive features of these monkeys include the large troops of insectivorous-frugivorous squirrel monkeys (100 or more), the monogamous family units of the titis and night monkeys, the prehensile tail of the omnivorous capuchins, and the short tails and specialized incisors and canines of the seed-eating sakis, bearded sakis, and uakaris, which allow them to eat hard fruits and nuts.[1] The four remaining genera of New World primates are the large and prehensile-tailed monkeys of the subfamily Atelinae.[17] Nine species and about 23 taxa of the folivorous howling monkeys (*Alouatta*) range widely throughout Central and South America and are easily recognized by their relatively slow, quadrupedal locomotion, sexual dimorphism (males larger than females), and loud territorial vocalizations, amplified by a large, hollow resonating chamber formed by the hyoid bone. The highly frugivorous spider monkeys (*Ateles*), with a distribution similar to that of howlers, but not extending south of the Amazon forests, include seven species and 17 taxa. They lack thumbs entirely and are named for their long limbs and brachiating locomotion. The Amazonian woolly monkeys (*Lagothrix*), are considered folivore-frugivores and are similar in size, but bulkier. One species, *Lagothrix lagothricha* (with three subspecies), is widespread throughout western Amazonia, whereas the other, *Lagothrix flavicauda,* is restricted to a small region of the Peruvian Andes.[14] The largest of the Neotropical primates are the muriquis (*Brachyteles*), folivore-frugivores that are restricted to Brazil's Atlantic forest.[23] The ecology, behavior, distribution, and conservation status of these primates have been extensively reviewed.[2,11,14,15,18,19]

REFERENCES

1. Ayres, J.M. 1989. Comparative feeding ecology of the uakari and bearded saki, *Cacajao* and *Chiropotes*. Journal of Human Evolution 18:697–716.
2. Coimbra-Filho, A.F.; and Mittermeier, R.A. Eds. 1981. Ecology and Behavior of Neotropical Primates, Vol. 1. Rio de Janeiro, Academia Brasileira de Ciências.
3. Dutrillaux, B. 1988. New interpretation of the presumed common ancestral karyotype of platyrrhine monkeys. Folia Primatologica 50:226–229.

4. Hershkovitz, P. 1977. Living New World Monkeys (Platyrrhini) with an Introduction to Primates, Vol. 1. Chicago, Chicago University Press.
5. Hershkovitz, P. 1983. Two new species of night monkeys, genus *Aotus* (Cebidae, Platyrrhini): A preliminary report on *Aotus* taxonomy. American Journal of Primatology 4(3):209–243.
6. Hershkovitz, P. 1984. Taxonomy of squirrel monkeys, genus *Saimiri,* (Cebidae, Platyrrhini): A preliminary report with description of a hitherto unnamed form. American Journal of Primatology 4:209–243.
7. Hershkovitz, P. 1985. A preliminary taxonomic review of the South American bearded saki monkeys genus *Chiropotes* (Cebidae, Platyrrhini), with the description of a new subspecies. Fieldiana, Zoology, New Ser. (27):iii, 46.
8. Hershkovitz, P. 1987. The taxonomy of South American sakis, genus *Pithecia* (Cebidae, Platyrrhini): A preliminary report and critical review with the description of a new species and new subspecies. American Journal of Primatology 12:387–468.
9. Hershkovitz, P. 1987. Uacaries, New World monkeys of the genus *Cacajao* (Cebidae, Platyrrhini): a preliminary taxonomic review with the description of a new subspecies. American Journal of Primatology 12:1–53.
10. Hershkovitz, P. 1990. Titis, New World monkeys of the genus *Callicebus* (Cebidae, Platyrrhini): a preliminary taxonomic review. Fieldiana, Zoology, New Ser. (55):1–109.
11. Kinzey, W.G. Ed. 1997. New World Primates: Ecology, Evolution, and Behavior. New York, Aldine de Gruyter.
12. Kobayashi, S. and Langguth, A. 1999. A new species of titi monkey, *Callicebus* Thomas, from north-eastern Brazil (Primates, Cebidae). Revista Brasileira de Zollogia 16(2):531–551.
13. Mittermeier, R.A.; de Macedo-Ruiz, H.; Luscombe, A.; and Cassidy, J. 1977. Rediscovery and conservation of the Peruvian yellow-tailed woolly monkey (*Lagothrix flavicauda*). In H.S.H. Prince Rainier III of Monaco and G.H. Bourne, eds., Primate Conservation. New York, Academic Press, pp. 95–115.
14. Mittermeier, R.A.; Rylands, A.B.; Coimbra-Filho, A.F.; and da Fonseca, G.A.B. Eds. 1988. Ecology and Behavior of Neotropical Primates,Vol. 2. Washington, D.C., World Wildlife Fund.
15. Norconk, M.A..; Rosenberger A.L.; and Garber, P.A. Eds. 1996. Adaptive Radiations of the Neotropical Primates. New York, Plenum Press.
16. Rosenberger, A. L. 1981. Systematics: The higher taxa. In A.F. Coimbra-Filho and R.A. Mittermeier, eds., Ecology and Behavior of Neotropical Primates, Vol. 1. Rio de Janeiro, Academia Brasileira de Ciências, pp. 9–27.
17. Rosenberger, A.L.; and Strier, K. B. 1989. Adaptive radiation of the ateline primates. Journal of Human Evolution 18:717–750.
18. Rylands, A.B.; Mittermeier, R.A.; and Rodríguez-Luna, E. 1994. A species list for the New World primates (Platyrrhini): Distribution by country, endemism, and conservation status according to the Mace-Lande system. Neotropical Primates 3(suppl.):113–160.
19. Rylands, A. B.; Rodríguez-Luna, E.; and Cortés-Ortiz, L. 1996–1997. Neotropical primate conservation—The species and the IUCN/SSC Primate Specialist Group Network. Primate Conservation (17):46–69.
20. Schneider, H.; and Rosenberger, A.L. 1996. Molecules, morphology, and platyrrhine systematics. In M.A. Norconk, A.L. Rosenberger, and P.A. Garber, eds., Adaptive Radiations of Neotropical Primates. New York, Plenum Press, pp. 1–19.
21. Schneider, H.; Schneider, M.P.C.; Sampaio, I.; Harada, M.L.; Stanhope, M.; Czelusniak, J.; and Goodman, M. 1993. Molecular phylogeny of the New World monkeys (Platyrrhini, Primates). Molecular Phylogenetics and Evolution 2(3):225–242.
22. Schneider, H.; Sampaio, I.; Harada, M.L.; Barroso, C.M.L.; Schneider, M.P.C.; Czelusniak, J.; and Goodman, M. 1996. Molecular phylogeny of the New World monkeys (Platyrrhini, Primates) based on two unlinked nuclear genes: IRBP Intron 1 and epsilon-globin sequences. American Journal of Physical Anthropology 100:153–179.
23. Strier, K.B. 1992. Faces in the Forest: The Endangered Muriqui Monkeys of Brazil. New York, Oxford University Press.

BIOLOGY AND CONSERVATION: FAMILY CALLITRICHIDAE

Cláudio Valladares-Pádua

TAXONOMY

It is widely accepted that the Callitrichidae family of New World primates encompasses four genera of marmosets and tamarins: *Cebuella, Callithrix, Saguinus,* and *Leontopithecus*[12]. Organization of the lower levels within this family, however, has still not been unanimously agreed upon (even after the broad revision conducted by Hershkovitz in 1977).[7] Two of the disputed areas within Callitrichidae taxonomy relate to the status of the *Callithrix* of southeastern Brazil and of the entire *Leontopithecus* genus. Whereas Hershkovitz[7] merged all southeastern *Callithrix* into the species *Callithrix jacchus* and resolved the existence of only one *Leontopithecus* species, many authors disagree. Of these, the most prominent are: Coimbra-Filho, Mittermeier, and Rylands, as well as Natori.[4,8,10] These researchers argue that all southeastern *Callithrix,* including the disputed *Callithrix kuhlii*[8,13] are separate species. Hershkovitz concluded that this latter was a hybrid and Vivo[17] included it as a subspecies of *Callithrix penicillata.* Additionally, Hershkovitz organized the *Leontopithecus* genus with *Leontopithecus* *chrysopygus* and *Leontopithecus chrysomelas* as subspecies of *Leontopithecus rosalia. Leontopithecus caissara* was excluded from this classification because its discovery was not made until 1990.

The historic disputes concerning Callitrichidae were aggravated by recent discoveries of many new species. These include *Callithrix mauesi,*[9] *Callithrix saterei,*[15] *Callithrix nigriceps,*[5] *Callithrix marcai,*[1] *Callithrix huimilis,*[17] and two other species, yet to be named, by van Roosmalen.[16]

Including these new species, the total number of basic taxonomic divisions in the Callitrichidae family has risen to 57. Of these, 36 may be considered species and 21 subspecies.

DISTRIBUTION

The recent discovery of "new" species suggests that, although these primates are among the best-studied mammals in the New World, their distribution is still not well understood. It is known that the family is primarily distributed in Brazil, with some northern species reaching no farther than the Costa Rica/Panama frontier and southern species no farther than Paraguay. Marmosets and tamarins are primarily arboreal and are not usually found in areas with higher elevations.

HABITAT

The marmosets and tamarins occupy a variety of habitats in tropical forests, including primary and secondary forests, forest edges, tree fall gaps, dry forests, and wet forests.[6] The habitat preference, however, varies between genera. Pygmy marmosets, for example, are specialized to seasonally inundated mature floodplain forests. They occupy the border as well as interior of these areas, as long as the flood level does not surpass 2 to 3 m.[16]

Individuals of the *Callithrix* genus, a widely distributed and generalist taxon, inhabit a varied range of habitats, including: hard-substrate forest, evergreen and seasonal semideciduous forests (Atlantic Forest region), highly dry thorn scrub forests (northeast Brazil), gallery and savanna forests (Central Brazil), white sand forest patches (Amazonia), gallery forest patches, and other forest patches in the pantanal (wetlands in central Brazil).

Saguinus also displays a variety of habitat preferences, including primary and secondary forest; a mixture of forest types, however, seems to be a requirement for this genus. The *Leontopithecus* species occur exclusively in the Atlantic forests. Three of these, *L. rosalia, L. chrysomelas,* and *L. caissara,* inhabit primarily the coastal lowlands forest whereas *L. chrysopygus* is found uniquely in the semideciduous forest of the interior.

EXPLOITATION

Several members of the Callitrichidae are exploited, mainly as pet animals. This is particularly common with *C. jacchus* and *C. penicillata*. Another, although today a less-common practice, is the capture of animals in the wild for biomedical research.

RESEARCH IN THE WILD

Despite a recent increase in the literature regarding Callitrichidae, there are still substantial gaps in this area. In Amazônia, some species are relatively well understood. Such is the case for the *Cebuella* genus, which has been studied for many years, primarily in Peru.[16] In this region, long-term work has also been conducted with various species of the *Saguinus* genus such as *Saguinus fuscicollis* and *Saguinus mistax*. In Panama several field studies have focused on *Saguinus oedipus*. Some species of the *Callithrix* genus in the Atlantic Forest and the cerrado (savanna from central Brazil), have also received increased attention during recent years (e.g., *C. jacchus, C. penicillata, Callithrix kuhlii, Callithrix aurita,* and *Callithrix flaviceps*). The *Leontopithecus* species, however, are the callitrichids most represented in the literature, with innumerable publications on each of the four lion tamarins.

CONSERVATION PROGRAMS

Among the conservation programs for callitrichids are those focusing on the four species of lion tamarins. These are well characterized in two population and habitat viability analyses (1990 and 1997).[2,14] The programs have succeeded in the creation of new conservation areas, animal reintroductions, and community involvement for the species' conservation.

REFERENCES

1. Alperin, R. 1993. *Callithrix argentata* (Linnaeus, 1771): Consideraçáes taxonômicas e descriçáo de subespécie nova. Boletim do Mususen Paraense Emílio Goeldi, Serié Zoologia 9(2):317–328.
2. Ballou, J.D.; Lacy, R.C.; Kleiman, D.G.; Rylands, A.B.; and Ellis, S. 1997. *Leontopithecus* II: The Second Popu-

lation and Habitat Viability Analysis.Apple Valley, Minnesota, IUCN/SSC/CBSG.
3. Coimbra-Filho, A.F. 1984. Situaçáo atual dos calitriquídeos que ocorrem no Brasil (Callitrichidae-Primates). In M.T. de Mello, ed., A Primatologia no Brasil. Brasília, Sociedade Brasileira de Primatologia, pp. 15–33.
4. Coimbra-Filho, A.F. 1990. Sistemática, distribuição geográfica e situaçáo atual dos símios brasileiros (Platyrrhini-Primates). Revista Brasileira De Biologia 50:1063–1079.
5. Ferrari, S.F.; and Lopes, M.A. 1992. A new species of marmoset, genus *Callithrix* Erxleben, 1977 (Callitrichidae, Primates), from western Brazilian Amazonia. Goeldiana Zoologia 12:17–41.
6. Garber, P.A. 1993. Feeding ecology and behaviour of the genus *Saguinus*. In A.B. Rylands, ed., Marmosets and Tamarins. Oxford, Oxford University Press, pp. 273–295.
7. Hershkovitz, P. 1977. Living New World Monkeys, Pt. 1. (Platyrrhini) with an introduction to Primates. Chicago, Chicago University Press.
8. Mittermeier, R.A.; Rylands, A.B.; and Coimbra-Filho, A.F. 1988. Systematics: Species and subspecies—an update. In R.A. Mittermeier, A.B. Rylands, A.F. Coimbra-Filho, and G.A.B. da Fonseca, eds., Ecology and Behavior of Neotropical Primates, Vol. 2. Washington, D.C., World Wildlife Fund, pp. 13–75.
9. Mittermeier, R.A.; Schwarz, M.; and Ayres, J.M. 1992. A new species of marmoset genus *Callithrix* Erxleben, 1977 (Callitrichidae, Primates) from the Rio Maués region, state of Amazonas, central Brazilian Amazonia. Goeldiana Zoologia 14:1–17.
10. Natori, M. 1986. Interspecific relationship of *Callithrix* based on the dental characters. Primates 27(3):321–326.
11. Rylands, A.B.; Coimbra-Filho, A.F.; and Mittermeier, R.A. 1993. Systematics, geographic distribution and some notes on the conservation status of the Callitrichidae. In A.B. Rylands, ed., Marmosets and Tamarins: Systematics, Behaviour and Ecology. Oxford, Oxford University Press, pp. 11–77.
12. Rylands, A.B. 1989. Sympatric Brazilian callitrichids: The black tufted-ear marmoset, *Callithrix kuhli* and the golden-headed lion tamarin, *Leontopithecus chrysomelas*. Journal of Human Evolution, 18:679–695.
13. Seal, U.; Ballou, J.; and Valladares-Padua, C. 1990. Leontopithecus Population Viability Analysis.Apple Valley, Minnesota, IUCN/SSC/CBSG.
14. Silva, J.S., Jr.; and Noronha, M. de A.2000. On a new species of bare-eared marmoset, genus *Callithrix* Erxleben, 1977, from Central Amazonia Brazil (Primates: Callitrichidae). Goeldiana Zoologia 18:(in press).
15. Soini, P. 1993. The ecology of the pygmy marmoset, *Cebuella pygmaea:* Some comparisons with two sympatric tamarins. In A.B. Rylands, ed., Marmosets and Tamarins. Oxford, Oxford University Press, pp. 257–261.
16. Van Roosmalen, M.G.M.; Van Roosmalen, T.; Mittermeier, R.A.; and Fonseca, G.A.B. 1998. A new distinctive species of marmoset (Callitrichidae, Primates) from the lower Rio Aripuaná, State of Amazonas, Central Brazilian Amazonia. Goeldiana Zoologia 22:1–27.
17. de Vivo, M. 1991. Taxonomia de Callithrix Erxleben, 1977(Callitrichidae, Primates). Belo Horizonte, Fundaçáo Biodiversitas.

NUTRITION

Roberto da Rocha e Silva

Studies in the 1970s attempted to understand primate nutrition[7,12] because primates were used for biomedical investigations and models for research in human nutrition.[8] The growing importance of primatology has brought new knowledge, which is used in improving the husbandry of these species in captivity, including caging, feeding, and veterinary medical assistance.[6,10]

The total amount of food to be fed must be divided into two or more portions. Although a feeder may contain enough food for all, hierarchical ranking may keep a low-ranking individual from access. Primates distribute themselves in the available space, keeping a certain distance from one another, according to a hierarchy not always obvious to humans. Izar and Sato,[9] studying the space organization of *C. apella* feeding in the wild, verified that feeding may be critical and competitive. It is not always possible to observe animals exactly at the moment food is deposited in the feeder, therefore, who ate cannot be verified.

Some physiologically normal conditions may demand a larger amount of food. Pregnant or lactating females need more energy, otherwise the infant will die. Lactating females may be offered calcium caseinate (1 g per adult per day, mixed into the food for tamarins), with excellent acceptance and success. The caseinate, derived from milk, is of high biological value, containing 10 essential amino acids.

Nogueira[13] studied the diet of females of *Brachytheles arachnoides* in various reproductive conditions. He observed that nonpregnant females spent less time than pregnant females looking for food and saved energy by remaining at rest, whereas pregnant females searched to diversify their diet. In the lactation stage energy requirements increased drastically, indicating that it is necessary to consider this aspect in captivity. During periods of circumstantial physiological stress, such as when there is excessive control of the feeder by the alpha individuals, it is best to place feeders in different locations in the enclosure, out of sight of the alpha individuals, to enable subordinates to eat without being harassed.

Each species may need different eating and drinking places. Feeders and waterers must be durable and should have no sharp edges that may cause injury.[16,17]

SELECTING FOODS AND FORMULATING DIETS

Although most species of primates are omnivorous, that does not mean that the same type of formulas should be offered to all of them. Commercial foods for dogs were initially used to supplement diets of nonhuman primates in captivity, and many zoos still make use of such feeds. Some primates are fed products of animal origin, such as beef, heart, or poultry, in addition to powdered milk, cheese, yogurt, honey, eggs, and meal worms. Currently, most zoos and primate centers feed a commercial primate pellet, supplemented with the items mentioned previously. Several North American manufacturers offer specific feeds, which are distributed in countries of South America. Some commercial diets for Neotropical primates are:

- Purina—Mazuri—New World primate diet (dry for small species, may be soaked in a fruit juice), Mazuri marmoset jelly (high-protein level), Mazuri primate high-fiber sticks (when additional fiber is valuable in the diet), leaf-eater primate diet (biscuit with a high-fiber diet). From Purina Mills, P.O. Box 66812, St. Louis, Missouri, USA 63166-6812 (*mazuri@purina-mills.com*).
- Zu/Preem Diets—Zu/Preem primate dry (biscuit form with 20% protein), Zu/Preem primate diet (canned, with 20% protein), and Zu/Preem Marmoset Diet (canned).
- Premier Laboratory Diets—Wayne Primate Diet (20% protein), New World Primate Diet (for small New World species). From Premium Nutritional Products, Inc., 7401 S. Adams St., Bartonville, Illinois, USA 61607 (*mail@zupreem.com*).
- Bio-Serv—Prima-Treats Juniors (for smaller primates such as squirrel monkeys and marmosets), Marmo-Jelly (jellylike substance to spread on bread), Primilac Infant Formula (powder formula) for New and Old World infant primates and others. From: Bio-Serve, Frenchtown, New Jersey, USA 08825.
- Ziegler—New World maintenance (17% protein), New World starter and reproduction (23% protein) and marmoset diet (20% protein). From: Ziegler Bros., Inc., P.O. Box 95, Gardners, Pennsylvania, USA 17324-0095.
- Animal Spectrum Primate Diets—biscuit form, with 18% protein. From Animal Spectrum, Lab Diet Sales Manager, Country Foods Division of Agway, Inc., Box 4933, Syracuse, New York, USA 13221.
- Nuvilab—In Brazil, there is currently only one supplementary diet for Neotropical primates, known as Nuvilab Primates 6030. From Nuvital, Estrada da Ribeira, 3001, CEP, Colombo, Paraná, Colombo, Puerto Rico(*nuvital@nuvital.com.br*).

When cytogenetic studies were analyzed by Schneider et al,[14] 16 genera of Neotropical primates were grouped according to their phylogeny. It was verified that they had similar feeding regimens. Ecological and behavioral studies of primates in the wild also corroborated that they have different feeding preferences in relation to their feeding habits.[2,11] These facts led to the possibility of establishing five different types of diets, which should be considered as husbandry-oriented programs.

- Diet A—For *Ateles-Brachyteles-Lagothrix/Allouatta,* species that are primarily leaf and fruit eating. Excessive offering of products of animal origin to these animals in captivity may produce feed overload and organic malfunctions. Fiber is indispensable for the digestive process of these species and should be permanently available to them. Leaf and fruit eating are more common among middle- and large-sized animals, and these species require a diet rich in vegetable fibers. These animals also ingest a great amount of flowers.
- Diet B—For *Aotus/Cebus-Saimiri,* omnivorous species, generalists. The diet of this group should be varied.
- Diet C—For *Chiropotes-Cacajao/Pithecia/Callicebus,* species that are primarily fruit eaters, especially seeds.
- Diet D—For *Callimico/Leontopithecus-Saguinus,* omnivorous species, specialized in the ingestion of invertebrates and small vertebrates.
- Diet E—For *Callithrix-Cebuella,* omnivorous species specialized in the ingestion of invertebrates and small vertebrates, but that also consume tree sap extracted by digging holes in the bark of trees with their specialized tooth. Experiences of Coimbra-Filho et al[4] show clearly the several degrees of concern regarding the callitrichids when they are offered artificial gums.

Diets for Neotropical primates should become more and more specific, thus providing a variety of nutrients to satisfy the requirements for special anatomical/physiological adaptations brought about by evolution.[3]

REFERENCES

1. Coimbra-Filho, A.F. 1981. Animais predados ou rejeitados pelo sauí-piranga, *Leontopithecus r. rosalia* (L., 1766) na sua área de ocorrência primitiva (Callitrichidae-Primates). Revista Brasileira de Biologia 41(4):717–731.
2. Coimbra-Filho, A.F.; and Mittermeier, R.A. 1981. Ecology and Behaviour of Neotropical Primates, Vol. 1. Rio de Janeiro, Academia Brasileira de Ciências.

3. Coimbra-Filho, A.F.; Rocha, N. da C.; and Pissinatti, A. 1980. Morfologia do ceco e sua correlaçáo com o tipo odontológico em Callitrichidae (Platyrrhini, Primates). Revista Brasileira de Biologia 40(1):117–185.
4. Coimbra-Filho, A.F.; Silva, R. da Rocha; and Aleksitch, S. 1988. Gomas enriquecidas na alimentaçáo de saguis em cativeiro. In M.T. Mello, ed., A Primatologia no Brasil, Brasilia, Sociedade Brasileira de Primatologia, pp. 133–136.
5. Coimbra-Filho, A.F.; Silva, R. da Rocha; and Pissinatti, A. 1981. Sobre a dieta de Callitrichidae em cativeiro. Biotérios 1:83–93.
6. Diniz, Lilian de Stefani Munaó. 1997. Primatas em Cativeiro: Manejo e Problemas Veterinários, Enfoque para Espécies Neotropicais. Sáo Paulo, ícone.
7. Harris, R.S. Ed. 1970. Feeding and Nutrition of Nonhuman Primates. New York, Academic Press.
8. Hayes, K.C. Ed. 1979. Primates in Nutritional Research. New York, Academic Press.
9. Izar, P.; and Sato, T. 1997. Influência de abundância alimentar sobre a estrutura de espaçamento interindividual e relaçáes de dominância num grupo de macacos-prego (*Cebus apella*). In S.F. Ferrari and H. Schneider, orgs., A Primatologia no Brasil, No. 5. Belém, Sociedade Brasileira de Primatologia, pp. 249–267.
10. Martin, D.P. 1986. Feeding and nutrition. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 661–663.
11. Mittermeier, R.A.; Coimbra-Filho, A.F.; and Fonseca, G.A.B. 1988. Ecology and Behavior of Neotropical Primates, Vol. 2. Washington D.C., World Wildlife Fund.
12. National Research Council (NRC). 1978. Nutrient Requirements of Nonhuman Primates. Washington D.C., National Academy of Sciences.
13. Nogueira, C.P. 1996. Comparaçáo entre as Dietas de Fêmeas de Muriquis (Brachyteles arachnoides, Primates, Cebidae) em Diferentes Estágios Reprodutivos. Masters thesis, University of Guarulhos, Sáo Paulo.
14. Schneider, H.; Sampaio, I.; and Schneider, M.P.C. 1997. Systematics of the platyrrhines. In S. Ferrari and H. Schneider, orgs., A Primatologia no Brasil, No. 5. Rio de Janeiro, Sociedade Brasileira de Primatologia, pp. 315–324.
15. Silva, R. da Rocha e. 1984. Elaboraçáo e distribuiçáo de dietas para calitriquídeos em cativeiro. In M.T. de Mello, ed., A Primatologia no Brasil. Brasília, Sociedade Brasileira de Primatologia, pp. 137–142.
16. Silva, R. da Rocha e. 1986. Novo modelo de comedouro/bebedouro para primatas calitriquídeos em cativeiro. In M.T. de Mello, ed., A Primatologia no Brasil, No. 2. Brasília, Sociedade Brasileira de Primatologia, pp. 419–422.
17. Silva, R. da Rocha e; Coimbra-Filho, A.F.; and Pissinatti, A. 1991. Modelos de comedouros experimentais para espécies das famílias Cebidae e Atelidae (Primates). In A.B. Rylands and A.T. Bernardes, eds., A Primatologia no Brasil, No. 3. Belo Horizonte, Sociedade Brasileira de Primatologia, pp. 311–315.

BEHAVIOR AND ENVIRONMENTAL ENRICHMENT

Vanner Boere

Environmental enrichment consists of a series of measures that modify the physical or social environment, improving the quality of life of captive animals by meeting their ethological needs. Environmental enrichment, well-being, and stress levels are intimately associated concepts regarding techniques, physiology, and behavior. Enrichment may reduce stress while increasing animal well-being in captivity. Prevalence of behavioral disorders, minimal clinical interventions, suitable ethical conditions, cost/benefit relationships, low mortality, and higher reproduction rates reflect directly the quality of captive breeding colonies.

Optimal conditions in natural environments are not usually found. Wild animals develop adaptive physiological and behavioral responses to harsh and varying environments. Individuals do not merely respond automatically to stimuli, but interact selectively and are permanently alert to significant novelties affecting their lives. Neotropical primates have complex and sophisticated central nervous systems. Therefore, compared with other vertebrate orders, primates are predisposed to continuously select and respond to novel stimuli in the environment.

Discrepancies in these processes as a result of captive environments have profound effects on physiological and psychological states. Captivity is an incomparably more stationary environment than a wild habitat, reducing attention and propensity to seek and relate to novelty. Prolonged periods of low stimulation lead to gradual loss of attention and search capabilities of new stimuli. "Environmental poverty," defined as inappropriate social and physical surroundings in captivity related to primates' ideal needs, may trigger a series of extreme nonadaptive responses.[1,2,3,4] Insufficient space, inadequate substrates, and isolation are only a few examples of impoverished environments. Ultimately, extreme nonadaptive responses result from chronic stress.

Complete neurological development is enhanced by enriched conditions, potentially influencing behavioral complexity.[5] Adaptive physiological and behavioral responses when facing stressful events depend on neural integrity. The final result of chronic stress is secretion of high levels of glucocorticoids (cortisol) by the adrenal cortex. Cortisol not only mobilizes stored energy, but acts as a catabolic agent, and may predispose nervous tissues to damage and impair an adequate immune response.[4] Other neural pathways, employing different neurotransmitters, are also involved, implicating serious motor disorders and consumptive behaviors.[4,6]

Simple structural modifications, changes in daily routines, and proper socialization are sufficient measures to stimulate and improve the psychological state and welfare of a colony (Table 25.2). By implementing a few basic measures of captivity maintenance and enrichment, mortality rates have dropped in some colonies.[7] Stimuli may not necessarily be protective or pleasurable. Mild stress stimuli, such as brief exposures to simulated predators, may induce behaviors similar to those occurring in the wild.[8] Some behaviors will only be expressed in captivity. Nonetheless, environmental enrichment is mainly aimed at introducing changes in the surroundings that will elicit behavioral patterns normally found in the wild.

Some experimental protocols require impoverished environments. However, adequate and enriched surroundings reflect research quality and influence colony maintenance costs.[9] Maintaining enriched home cages is not only justified by technical aspects. Moral and ethical issues are implicitly involved, especially because of the growing concept that human beings are more and more similar to other species from a functional and evolutionary perspective. When minimal enrichment measures are established, chronic stress is reduced, thus increasing colony success, reducing clinical interventions, minimizing costs, and creating a feeling of satisfaction among personnel involved. Before any change is introduced, good background knowledge of the species is needed. In the absence of available data, the closest taxon may be considered.

Ideally, an adequate diagnosis of the environment requires evaluation of both physiological and behavioral indicators. A simple health parameter consists of periodically evaluating the weight or growth curves in infants. Clinical history indicating a frequent occurrence of diseases or lacerations points to inadequate captive environments. Other auxiliary laboratory parameters may be used to evaluate environmental inadequacy, such as cortisol levels and lymphopenia.[4]

Ethological indicators are more easily accessible measures to detect inadequate individual needs in captivity. Some behaviors are especially disturbed by environmental inadequacy, such as increased aggressiveness, abnormal behaviors, lower body contacts, decrease of grooming and play.[10,11] Other indicators of anxiety include frequent scratching and reduced exploratory behavior.[12] Primates maintained in impoverished environments seem to have lower expectations of stimuli in their surroundings, decreasing motor performance, motivation, and consumptive behaviors.[13] This unresponsive scenario constitutes a state of boredom that, under the impact of intense stimuli, may lead to sudden death. Under these same conditions, some individuals develop alternative strategies in the hope of increasing environmental stimulation, such as coprophagy, lethargy, stereotyped behaviors, automutilation, inappropriate sexual mounts, hypersexuality, abnormal postures, and overt expression of some normal behaviors out of context.

At least two of the following categories are necessary to qualify captivity conditions as satisfactory:[9] good physical health, complete behavioral repertoire, absence of chronic stress, and problem-solving abilities. The main factors considered in enrichment programs include health, hygiene, space dimension and complexity (substrate use, toys, games, and challenges), diet, climate, social composition, communication, and personnel involved. During some procedures, for example, quarantine, isolation becomes justifiable, while also maintaining strict aseptic control of these enclosures. Ideally, the goal is to obtain a clean, pathogen-free, enriched environment.

Complex home cage designs may reduce emotional reactions.[11] More than just physical space, environmental complexity or novelty have been considered a basic element for enrichment.[14] Primates are intensely curious about new or strange objects, more because of novelty than object's characteristics in itself. Response to novel objects lasts longer when contingencies with reinforcement are made.[15] Primates are sensitively responsive to change and social demands. Presence of another compatible conspecific in the enclosure is considered the greatest enrichment measure possible for primates.[8] The most crucial social contact occurs during infancy, through mother-infant bonding.[2,16]

Taking advantage of primates' strong propensity for learning, colony management and quality of life may be improved by implementing routines. Training, when followed by carefully intervened contacts and reinforcements, is enriching in itself. Cooperative conditioned training tasks have demonstrated decreases in stereotyped behaviors and overall individual improvement.[17] Primates may be actively trained to cooperate in such management routines as venipuncture, weighing, restraint, clinical exams, and home cage cleaning, always employing positive reinforcement criteria.[17] Besides enrichment advantages, routines derived from learned experiences create bonds between primates and their caretakers, increase security aspects for all involved, reduce risk of accidents, lower the incidence of stress, and save time. Conditioning presents the advantage of attenuating autonomic responses by avoiding maximal stimulation. Individuals must always be trained considering probable behavioral patterns to avoid physiological and psychological overcharge. Primates seem to be capable of recognizing

TABLE 25.2. Environmental enrichment for primates

Taxon	Problems and Goals	Enrichment	Results
Callithrix jacchus[20, 21]	Inactivity	Alternated access to an exercise cage	Increased locomotor activity
Callithrix jacchus[22]	Enrich the environment	Hollow wooden dowel containing Arabic gum (artificial "gum tree")	Active exploration of the apparatus
Callithrix jacchus[23]	Establishing a standard cage for the species	Increase home cage size and complexity (more than one feeding device, nest box, perches, refuges, and wood shavings)	Positive behaviors (increased exploratory activities) were mainly due to environmental complexity, rather than increased home cage size.
Callithrix jacchus[24]	Evaluate enrichment related to social group composition.	Add new support substrates (ropes and branches) and increase foraging difficulty (whole fruits under suspension increase foraging difficulty and grains on the ground)	Decreased rest activities, while increasing foraging among adults and juveniles; for infants, increased foraging, play, and social grooming was observed
Callithrix[25]	Enrichment; protection against the cold; educational purposes for the public in general	Trunks and branches; ground covered with plants; infra-red lamp	Stress decreased and successful mating occurred; effective protection against the cold
Callithrix and *Saguinus*[19]	Improve captive environment	Various branches and tree trunks, periodically rearranged in the home cage	Increased exploration and provided appropriate substrates
Saguinus mystax and *Saguinus labiatus*[26]	Compare performance in platyrrhini and catarrhini while undertaking a manipulative task contingent with reinforcement and increased task difficulty	Foraging tray and dispenser with an increasing access difficulty device	Successful acceptance of foraging tray; however unsuccessful with the dispenser
Saguinus oedipus[27]	Reproductive inhibition; stereotyped behaviors; evaluation of critical home cage size	Increase dimensions of the home cage	In larger home cages stereotyped behaviors were not observed
Saguinus oedipus[28]	Sedentary postures and exacerbated aggressiveness toward humans	Increase number of feeding trays, shifts in food location, food "search" apparatus, and introduction of new items	Increased exploratory activity; lower exacerbated aggressive behaviors
Saimiri sciureus[29]	Enrichment	Larger home cage, containing different environments (perches, substrates, and plants); feeding device	Increased locomotor in the beginning, followed by a decrease in movement; species-specific behaviors
Saimiri sciureus[30]	Stereotyped and idiosyncratic specific behaviors (normal behaviors exhibited out of context or more frequently than normally expressed)	Disperse food within the home cage; introduction of novel objects and apparatuses with food rewards	Increased manipulation, while decreasing stereotyped behaviors; food dispersion was not very successful
Cebus capucinus[31]	Nonspecific	Different food dispersers, made of plexiglass, with varying degrees of difficulty to obtain food items	Foraging, rest, and movement closely resembled naturally occurring behavioral patterns
Cebus apella[32]	Stereotyped behaviors and single housing	Larger home cage and socialization with conspecifics	Reduced stereotyped behaviors and calmer behaviors

and categorizing humans.[18] For the most part, humans represent potential threats to other primates. Growing evidence indicates, however, that interactions between captive primates and maintenance personnel may lead to improvements in daily routines, constituting an important enrichment measure.[19] Relative individual control over environmental conditions, and over themselves, must also be provided to captive animals.

Response to enrichment is usually fast. However, adequacy and results must be evaluated in the long run. Enriched environments not inducing response point to inadequate measures employed. Sometimes individuals are so "behaviorally disorganized" that they are unable to respond to usually employed enrichment techniques.[1]

REFERENCES

1. Meyer-Holzapfel, M. 1968. Abnormal behavior in zoo animals. In M.W. Fox, ed., Abnormal Behavior in Animals. Philadelphia, W.B. Saunders, pp. 476–503.
2. Harlow, H.F.; Harlow, M.K.; Schiltz, K.A.; and Mohr, D.J. 1971. The effect of early adverse and enriched environments on the learning ability of Rhesus monkeys. In L.E. Jarrard, ed., Cognitive Processes of Nonhuman Primates. New York, Academic Press, pp. 121–148.
3. Mason, G.J. 1991. Stereotypes: A critical review. Animal Behavior 41:1015–1037.
4. Sapolsky, R.M. 1993. Neuroendocrinology of the stress-response. In J.B. Becker, S.M. Breedlove, and D. Crews, eds., Behavioral Endocrinology. Cambridge, MIT Press, pp. 287–324.
5. Rosenzweig, M.R. 1996. Aspects of the search for neural mechanisms of memory. Annual Review of Psychology 47:1–32.
6. Kraemer, G.W.; and Clarke, A.S. 1990. The behavioral neurobiology of self-injurious behavior in rhesus monkeys. Progress Neuro-Psychopharmacology and Biological Psychiatry 14:141–168.
7. Roberts, J.A. 1989. Environmental enrichment, providing psychological well-being for people and primates. American Journal of Primatology 1(Suppl.):25–30.
8. Chamove, A.S.; and Moodie, E.M. 1990. Are alarming events good for captive monkeys? Applied Animal Behaviour Science 27:169–176.
9. Novak, M.A.; and Suomi, S.J. 1988. Psychological well-being of primates in captivity. American Psychologist 43(10):765–773.
10. Schoenfeld, D. 1989. Effects of environmental impoverishment on the social behavior of marmosets (*Callithrix jacchus*). American Journal of Primatology 1(Suppl.):45–51.
11. Thompson, K.V. 1996. Behavioral development and play. In D.G Kleiman, M.E. Allen, K.V. Thompson, and S. Lumpkin, eds., Wild Mammals in Captivity. Chicago, The University Of Chicago Press, pp. 352–371.
12. Barros, M.; Boere, V.; Huston, J.; and Tomaz, C. 1999. The stuffed predator: A new model to evaluate anxiety in primates. Behavioural Brain Research 108(2):205–211.
13. Carlstead, K. 1996. Effects of captivity on the behavior of wild mammals. In D.G Kleiman, M.E. Allen, K.V. Thompson, and S. Lumpkin, eds., Wild Mammals in Captivity. Chicago, University of Chicago Press, pp. 317–333.
14. Woolverton, W.L.; Ator, N.A.; Beardsley, P.M.; and Carrol, M.E. 1989. Effects of environmental conditions on the psychological well-being of primates: a review of the literature. Life Sciences 44:901–917.
15. Poole, T. 1990. Environmental enrichment for marmosets. Animal Technology 41(2):81–86.
16. Mason, W.A. 1991. Effects of social interaction on well-being: Development aspects. Laboratory Animal Science 41(4):323–328.
17. Reinhardt, V. and Reinhardt, A. 2000. Social enhancement for adult nonhuman primates in research laboratories: A review. Laboratory Animal 29(1):34–41.
18. Mitchell, G.; Steiner, S.; Dowd, B.; Tromborg, C.; and Hering, F. 1991. Male and female observers evoke different responses from monkeys. Bulletin of the Psychonomic Society 29(4):358–360.
19. Snowdon, C.T.; and Savage, A S. 1989. Psychological well-being of captive primates: General considerations and examples from callithrichids. In E.F. Segal, ed., Housing, Care, and Psychological Well-Being of Captive and Laboratory Primates. Park Ridge, New Jersey, Noyes, pp. 75–88.
20. Hearn, J.P.; Abbot, D.H.; Chambers, P.C.; Hodges, J.K.; and Lunn, S.F. 1978. Use of the common marmoset, *Callithrix jacchus,* in reproductive research. In Proceedings of the Conference on Marmosets in Experimental Medicine. Oak Ridge, S. Koager pp. 40–49.
21. Price, E.C.; and Mcgrew, W.C. 1990. Cotton-top tamarins (*Saguinus o. oedipus*) in a semi-naturalistic captive colony. American Journal of Primatology 20:1–12.
22. Mcgrew, W.C.; Brennan, J.A.; and Russel, J. 1986. An artificial "gum-tree" for marmosets (*Callithrix jacchus*). Zoo Biology 5:45–50.
23. Kerl, J.; and Rothe, H. 1996. Influence of cage size and cage equipment on physiology and behavior of common marmosets (*Callithrix jacchus*). Laboratory Primate Newsletter 35(3):10–15.
24. Peregrino, H.A.S.; Yamamoto, M.E.; and Sousa, M.B.C. 1997. Efeito do enriquecimento ambiental sobre a freqüência e distribuiçáo do comportamento em *Callithrix jacchus* (Enrichment effects on frequency and distribution behavior of *Callithrix jacchus*). In Proceedings of the 8° Congresso Brasileiro de Primatologia, Joáo Pessoa, Sociedade Brasilieria de Primatologia, p. 150..
25. Carrol, J.B. 1991. The captive breeding of the genus *Callithrix* at the Jersey Wildlife Preservation Trust. In A.B. Rylands and A.T. Bernardes, eds., A Primatologia no Brasil, No. 3. Belo Horizonte, Fundaçáo Biodiversitas, pp. 17–23.

26. Evans, H.L.; Taylor, J.D.; Ernst, J.; and Graefe, J.F. 1989. Methods to evaluate the well-being of laboratory primates: Comparisons of macaques and tamarins. Laboratory Animal Science 39(4):318–323.
27. Baldwin, J.D.; and Baldwin, J.I. 1977. The role of learning phenomena in the ontogeny of exploration and play. In S. Chevalier-Skolnikoff and F.E. Poirier, eds., Primate Bio-Social Development: Biological, Social, and Ecological Determinants. New York, Garland, pp. 343–406.
28. Glick-Bauer, M. 1997. Behavioral enrichment for captive cotton-top tamarins (*Saguinus oedipus*) through novel presentation of diet. Laboratory Primate Newsletter 36(1):1–4.
29. Fragaszy, D.M. 1979. Titi and squirrel monkeys in a novel environment. In J. Erwin, T.L. Maple, and G. Mitchell, eds., Captivity And Behavior. New York, Van Nostrand Reinhold, pp. 172–216.
30. Boinski, S.; Noon, C.; Stans, S.; Samudio, R.; Sammarco, P.; and Hayes, A. 1994. The Behavioral profile and environmental enrichment of a squirrel monkey colony. Laboratory Primate Newsletter 33(4):1–5.
31. Hayes, S.L. 1990. Increasing foraging opportunities for a group of captive capuchin monkeys (*Cebus capucinus*). Laboratory Animal Science 40(5):515–519.
32. Bayne, K.; Dexter, S.; and Suomi, S. 1991. Social housing ameliorates behavioral pathology in *Cebus apella*. Laboratory Primate Newsletter 30(2):7–11.

MEDICINE

José Luiz Catão-Dias

ECTOPARASITES

Publications on ectoparasitic diseases in Neotropical primates are relatively scarce. This may be explained, to some extent, by the lack of medical importance given to these parasites in platyrrhini, because ectoparasitic diseases are not listed among the main diseases.[1,2,7] An updated review presents, in a detailed and precise text, the main parasites described in nonhuman primates,[9] which include flies, lice, ticks, and acari.

Fly larvae of the family Cuterebridae, genera *Cuterebra, Dermatobia,*and *Alouattamyia,* are responsible for the occurrence of myiasis in howlers and squirrel monkeys.[6,9] These processes are expressed as dermal cysts, with a risk of secondary bacterial infection. Surgery is suitable for removing the larvae. Antiseptics should be applied to the sites. Pediculosis in Neotropical primates is caused predominantly by lice of the order Anoplura, genus *Pediculus,* affecting howlers, spider monkeys, tamarins, and marmosets.[3,9]

Ticks are observed in free-ranging Neotropical primates, as well as those kept in captivity. These ectoparasites are potential vectors for several diseases, including zoonoses.[9] *Ixodes* is described as the most frequently identified genus among the platyrrhini. However, *Amblyomma oblonguttatum* was recently observed in *Alouatta caraya* (Dr. R. Teixeira, personal communication, 1999). Tick infestation is often asymptomatic, even though serious infection may lead to distress, weakness, and emaciation.[3,9]

Acari of major importance to New World primates belong to the suborder Sarcoptiformes. These arthropods parasitize the skin and hair follicles of multiple species of platyrrhini, including the capuchins (*Cebus* sp.), marmosets (*Callithrix* sp.), and night monkeys (*Aotus* sp.). The major genera of sarcoptiformes described are *Prosarcoptes, Dunnalges,* and *Rosalialges.*[9] Recently, *Fonsecalges saimirii,* originally reported in *S. sciureus,*[4] was observed inside the ear pinna of a capuchin (*Cebus apella*) (Dr. L.R.M. Sá, personal communication, 1998). Sarcoptic mange was described in *S. mystax.*[5] Clinically, acariasis is characterized by intense pruritus and distress, with self-mutilation as a common occurrence in severely infested animals. Coexisting symptoms are anorexia, emaciation, and alopecia. Microscopic lesions include exuberant hyperkeratosis and parakeratosis. Demonstration of parasites upon microscopic examination of skin scrapings is diagnostic.

Control of the various ectoparasitic diseases that harm primates can be accomplished by strict sanitation management and treatment of affected individuals.[9] For this purpose, products used for domestic cats and humans are usually efficient.[8]

REFERENCES

1. Belluomini, H.E.; Diniz, L.S.M.; Saliba, A.M. 1980. Findings of pathological anatomy in mammals from the "Fundação Parque Zoológico de Sáo Paulo" 1971. Memórias do Instituto Butantan 44/46: 133–152.
2. Diniz, L.S.M.; da-Costa, E.O. 1995. Health problems of *Callithrix jacchus* in captivity. Brazilian Journal of Medical and Biological Research 28: 61–64.
3. Fiennes, R.N.T.-W. 1972. Ectoparasites and vectors. In R.N.T.-W. Fiennes, ed. Pathology of Simian Primates. New York, Karger, Part 2, pp. 158–176.
4. Flatt, R.E.;Patton, N.M. 1969. A mite infection in squirrel monkeys. Journal of the American Veterinary Medicine Association 155: 1233–1235.
5. Gozalo, A.; Montoya, E. 1991. Mortality causes of the moustached tamarin (*Saguinus mystax*) in captivity. Journal of Medical Primatology 21: 35–38.
6. Middleton, C.C.; Clarkson, T.B.; Garner, F.M. 1964. Parasites of the squirrel monkeys (*Saimiri sciureus*). Laboratory Animal Care 14: 335.
7. Montali, R.J.; Bush, M. 1999. Diseases of the Callithrichidae. In M.E. Fowler and E. Miller, eds. Zoo and

Wildlife Medicine. Current Therapy 4. Philadelphia, W.B. Saunders, pp. 369–376.
8. Renquist, D.M.; Whitney, R.A., Jr. 1987. Zoonoses acquired from pet primates. Veterinary Clinics of North America: Small Animal Practice 17(1): 219–240.
9. Toft II, J.D.; Eberhard, M.L. 1998. Parasitic diseases. In B.T. Bennett; C.R. Abee; R. Henrickson, eds. Nonhuman Primates in Biomedical Research. Diseases. San Diego, Academic Press, pp. 111–206.

TOXOPLASMOSIS

Toxoplasmosis is a cosmopolitan disease that affects a great variety of vertebrates, including Neotropical primates. It is caused by an intestinal coccidia, *Toxoplasma gondii*. Known definitive hosts are domestic cats and other species of the family Felidae. The infection of intermediate hosts occurs by ingestion of food or water contaminated by sporulated oocysts or cysts, or through the placenta. The physiopathological basis of this disease is a multifocal necrosis produced by the intracellular proliferation of tachyzoites.[7]

Neotropical primates are apparently more susceptible to toxoplasmosis than catarrhini, and the clinical manifestation of the disease among the platyrrhini is usually acute and fatal.[7] The main morphologic changes observed are severe lung edema and congestion, hepatomegaly associated with multifocal areas of necrosis, splenomegaly, and remarkable mesenteric fibrinohemorrhagic lymphadenitis.[4,5,7] Necrotizing ulcerative or segmental enteritis is a relatively common finding, being associated with the agent's portal of entry.

Reports on toxoplasmosis in Neotropical primates have considerably increased in recent years.[1,2,3,6] In a similar fashion, the occurrence of this protozoonosis among the platyrrhini collections kept captive in Brazil has been particularly high, affecting both callitrichids and cebids. This author is acquainted with five major outbreaks, involving four zoos, three of which are located in the state of São Paulo and one in Paraná (Dr. Z. Cubas, personal communication, 1999), causing loss of at least 38 individuals. In at least two of these outbreaks, the source of infection was believed to be feral cats in the surroundings of the animal facilities.

The diagnosis of toxoplasmosis can be accomplished by finding the agent upon microscopic examination, using routine staining, as well as immunohistochemistry techniques, or by isolation of *T. gondii* after inoculation in laboratory animals.[5,7] The differential diagnosis includes microsporidiosis and *Neospora* ssp.[5]

The treatment of toxoplasmosis in platyrrhini holds questionable efficiency. As a general procedure, the same therapy strategy adopted in human pediatrics is recommended, that is, association of pyrimethamine and sulfonamides, along with daily supplement with folic acid.[7] This treatment is effective only against the tachyzoite forms.[5] In experimental therapy, the substance hydroxynaphtoquinone has been effective against the bradyzoite forms.[5]

Prophylaxis of toxoplasmosis in Neotropical primates encompasses a reduction in concentration of infectious forms in the environment. To do this, it is necessary to follow rigid sanitation guidelines and strictly control cat access to facilities occupied by primates, as well as to the kitchens. Other aspects that also should be regarded are control of potential horizontal transmissions by keepers who keep cats at home and the feeding of raw fresh meat to platyrrhini.[7]

REFERENCES

1. Epiphanio, S.; Catáo-Dias, J.L.; Guimaráes, M.A.B.V. 1999. Toxoplasmosis in emperor tamarin (*Saguinus imperator*). Brazilian Journal of Veterinary Research and Animal Science 36 (in press).
2. Epiphanio, S.; Guimaráes, M.A.B.V,; Fedullo, D.L.; Correa, S.H.R.; Catáo-Dias, J.L. 2000. Toxoplasmosis in *Leontopithecus chrysomelas* and *Saguinus imperator*. Journal of Zoo and Wildlife Medicine 31(2)(in press).
3. Juan-Sallés, C.; Prats, N.; Marco, A.J.; Ramos-Vara, J.A.; Borrás, D.; Fernandes, J. 1998. Fatal acute toxoplasmosis in three golden lion tamarins (*Leontopithecus rosalia*). Journal of Zoo and Wildlife Medicine 29(1): 55–60.
4. Montali, R.J.; Bush, M. 1999. Diseases of the Callithrichidae. In M.E. Fowler and E. Miller, eds. Zoo and Wildlife Medicine. Current Therapy 4. Philadelphia, W.B. Saunders, pp. 369–376.
5. Osborn, K.G.; Lowenstine, L.J. 1998. Respiratory diseases. In B.T. Bennett, C.R. Abee, R. Henrickson, eds. Nonhuman Primates in Biomedical Research. Diseases. San Diego, Academic Press, pp. 263–310.
6. Pertz, C.; Dubielzig, R.R.; Lindsay, D.S. 1997. Fatal *Toxoplama gondii* infection in golden lion tamarin (*Leonopithecus rosalia rosalia*). Journal of Zoo and Wildlife Medicine 28(4): 491–493.
7. Toft II, J.D.; Eberhard, M.L. 1998. Parasitic diseases. In B.T. Bennett, C.R. Abee, R. Henrickson, eds. Nonhuman Primates in Biomedical Research. Diseases. San Diego, Academic Press, pp. 111–206.

NEOPLASIA

The first studies describing proliferative processes in nonhuman primates suggested that the prevalence of neoplasic disorders was relatively low. As an example of this situation, as few as 122 reports of spontaneous tumors were described until 1968.[1] However this situation has dramatically changed over the past two decades as the number of neoplasia cases reported in Neotropical primates has considerably increased.[6,8] The reasons for this increase are not clear, however it is suggested that an improvement of husbandry conditions of animals in zoological parks and research centers has led to a greater life span of individuals, and longevity, in conjunction with the adoption of more detailed pathology protocols, may have contributed to this picture.[2]

Most cases reported reflect an incidental occurrence of isolated tumors, including a melanocitic ependymoma in *C. goeldi,* a seminoma in *Aotus* sp., and a primary kidney hemangioma in *S. mystax*.[3,4,7] Those readers interested in more detailed and precise discussion of neoplasias described in platyrrhini should read available reviews.[1,6,8]

At least two types of neoplasia seem to occur frequently in platyrrhini, pheochromocytomas and colon carcinomas. In a retrospective pathological survey of Neotropical primates recently conducted at the National Zoo, Washington, DC, 27 neoplasias were identified. Five of these tumors were benign pheochromocytomas, three of which occurred in golden lion tamarins (*L. rosalia*), one in a howler monkey (*Alouatta villosa*), and one in a spider monkey (*Ateles fusciceps*). The causes, as well as the possible predisposing factors to this incidence, have not been identified.[2]

The second type, colon carcinoma, particularly affects *S. oedipus*. The etiology of this process has not been clearly identified, however the occurrence of cancer was correlated with the presence of acute colitis and chronic changes of the mucosa. Dietary and other environmental factors seem to be involved.[5]

REFERENCES

1. Bieniashvili, D. Sh. 1989. An overview of the world literature on spontaneous tumors in nonhuman primates. Journal of Medical Primatology 18: 423–437.
2. Catão-Dias, J.L.; Montali, R.J.; Strandberg, J.D.; Johnson, L.K.; Wolff, M.J. 1996. Endocrine neoplasia in New World primates. Journal of Medical Primatology 25: 34–41.
3. Gozalo, A.; Chavera, A.; Dagle, G.; Montoya, E.; Weller, R. 1993. Primary renal hemangiosarcoma in a moustached tamarin. Journal of Medical Primatology 22: 431–432.
4. Gozalo, A.; Nolan, T.; Montoya, E. 1992. Spontaneous seminoma in an owl monkey in captivity. Journal of Medical Primatology 21: 39–41.
5. Lorna, D.J.; Ausman, L.M.; Prabhat, K.S.; King, N.W. Jr. 1996. A prospective study of the epidemiology of colitis and colon cancer in cotton-top tamarins (*Saguinus oedipus*). Gastroenterology 110: 102–115.
6. Lowenstine, L.J. 1986. Neoplasms and proliferative disorders in nonhuman primates. In K. Benirschke, ed. Primates: The Road to Self-Sustaining Populations. New York, Springer-Verlag, pp. 781–814.
7. Nichols, D.K.; Catão-Dias, J.L. 1995. Melanotic ependymoma in a Goeldi's marmoset (*Callimico goeldii*). Journal of Medical Primatology 24: 49–51.
8. Weller, R.E. 1998. Neoplasia/Proliferative disorders. In B.T. Bennett; C.R. Abee; R. Henrickson, eds. Nonhuman Primates in Biomedical Research. Diseases. San Diego, Academic Press, pp. 207–232.

INFECTIOUS DISEASES

Viral, bacterial and fungal diseases figure as important causes of morbidity, affecting both captive and free-ranging Neotropical primates.[4,18] Yet another relevant point is the zoonotic aspect of some of these diseases.[7] It is necessary that the practitioner in charge of the health management of platyrrhini take particular precautions and institute routine surveillance to protect employees. The objective of this section is to introduce some of the most important infectious diseases affecting captive platyrrhini in South America. To readers interested in more details, some excellent recently published reviews are recommended.[8,17,18,22]

The platyrrhini are susceptible to a wide variety of viral agents, among them alpha-herpesvirus (*Herpesvirus hominis, Herpesvirus tamarinus*), gamma-herpesvirus (*Herpesvirus saimiri, Herpesvirus ateles,* and Epstein-Barr virus), cytomegalovirus, *Papillomavirus, Morbillivirus, Paramyxovirus, Flavivirus,*and the lymphocytic choriomeningitis virus.[17,18,22] However, reported cases of viral infections in Neotropical primates kept in South America are scarce, which probably reflects the need for more accurate diagnostic methods.

Among the available studies are those related to the occurrence of herpesviruses caused by *Herpesvirus hominis* in callitrichids,[21] in which clinical changes and anatomo-pathological findings are described in two individuals of *C. jacchus*. Signs exhibited were oral ulcerations, located predominantly in the gingiva, tongue, and oral cavity, and neurological deficits, including motor incoordination, ataxia, anisocoria, nystagmus, and tonic-clonic seizures. The anatomo-pathological examination revealed hyperkeratosis of the tongue epithelium. The brain showed focal areas of hemorrhage, edema, and microcavitation, associated with severe meningoencephalic inflammatory infiltration, composed of mononuclear and polymorphonuclear cells. The cortex was the most affected region, with development of perivascular inflammatory cuffing and presence of intranuclear inclusions in glial cells.

Recently, a probable new papillomavirus was described in a male adult brown howler monkey, *Alouatta fusca,* causing a process compatible with a condition known as focal epithelial hyperplasia (FEH).[23] FEH is a rare disease that primarily affects human beings. In addition to humans, other affected primates are the chimpanzee, *Pan troglodytes,*and the pygmy chimpanzee, *Pan paniscus.* FEH is grossly characterized by papules located inside the oral cavity, particularly in the mucous membranes of lower lip, gingiva, and tongue. Microscopically, the main characteristics of FEH are acanthosis, elongation and fusion of the rete ridges, and koilocytosis.[1]

In the brown howler monkey, immunohistochemistry tests for generic papillomavirus (PV) antigen yielded a strong positive in koilocyte nuclei. However, the specific reactions for detection of human papillomaviruses (HPV) types 6, 11, and 18 were negative, as well as the reactions of "in situ" hybridization for detection of HPV DNA using a broad-range biotinylated probe. These results indicated that the described process contained no HPV DNA, suggesting that the PV identified in this case is probably a mucosotropic virus specific to the oral cavity of howler monkeys.[23]

Finally, callitrichid hepatitis (CH), caused by an arenavirus termed lymphocytic choriomeningitis virus (LCMV), is a zoonosis and an important emerging viral infection, originally described in the mid-1980s at North American zoos.[20] This is a highly lethal disease, clinically identified by acute onset associated with variable degrees of jaundice, lethargy, anorexia, and an increase in serum enzyme levels. The disease is directly related to ingestion of infected mice, through providing the animals with neonate rodents or through capture of wild mice by callitrichids. Grossly, CH is characterized by hepatosplenomegaly and by subcutaneous and intramuscular hemorrhages. Histologically, the most characteristic finding is a mild multifocal liver necrosis, associated with the presence of Councilman bodies, representing hepatocellular apoptosis.[19] The importance of HC has gained more dimension with the program for reintroduction of golden lion tamarins, *L. rosalia,* into natural reserves in the state of Rio de Janeiro, Brazil. This author is not aware of any reports of CH affecting populations of callitrichids kept in South America, and wide serological surveys haven't revealed the existence of cases or previous exposure of wild golden lion tamarins in their natural habitats.[25]

Bacterial diseases represent important causes of morbidity and mortality involving platyrrhini collections. Several bacterial genera have been described as responsible, and a recent review gave a detailed description of the main processes.[8] Available reports on bacterial infections in Neotropical primates kept in South American collections are relatively numerous, when compared with reports of viral and mycotic diseases.

In a 20-year retrospective study, bacterial diseases were responsible for 7% of the health problems that occurred in the *C. jacchus* collection of a zoological park in Sáo Paulo, Brazil.[5] The most frequently isolated bacteria were *Shigella* spp., *Salmonella* spp., *Arizona* sp., enteropathogenic *Campylobacter* spp., and *Escherichia coli* strains. In addition, *Staphylococcus* spp., *Streptococcus* spp., *Haemophilus* spp., and *Pneumococcus* spp., were isolated from respiratory tract secretions, frequently involved with pneumonia. A 5-year retrospective analysis of necropsies performed in Neotropical primates at the Laboratory of Comparative Pathology of Wild Animals of the Department of Pathology of the School of Veterinary Medicine of the University of São Paulo (LAPCOM-USP), revealed the presence of several bacteria, including *Streptococcus zooepidemicus, Campylobacter jejuni, Staphylococcus aureus, Staphylococcus xylosus, Proteus mirabilis, Citrobacter freundii, Klebsiella pneumoniae,* and *Salmonella* spp. Similar results were obtained in retrospective surveys on the causes of death of *Saguinus mystax, Aotus nancymae,* and *A. vociferans.*[12,14]

Pasteurella haemolytica was responsible for the sudden death of an adult male *Callimico goeldi.* Grossly, the animal showed multifocal pale areas over the hepatic surface. Histologically, the foci comprised necrotic centers surrounded by polymorphonuclear cells.[15] An infectious outbreak of *K. pneumoniae,* affecting 17 individuals, the majority of which were *Saguinus* and *Aotus* spp., was reported by a primatology center in Peru.[13] Suppurative peritonitis, pyothorax, lobar pneumonia, and abscessed hepatitis were the most common findings. Therapy based on tetracycline dissolved in drinking water and administered for 5 days was effective in overcoming the outbreak.

The involvementof *Campylobacter* spp. in the development of enteropathogenic processes has been widely discussed. *C. jejuni* has been isolated from healthy golden lion tamarins at the National Zoo, Washington, DC, and this bacterium has been associated with bowel diseases in other Callitrichidae colonies.[18] In a study conducted in Peru, the prevalence of *Campylobacter* spp. in fecal samples of 16 *Saguinus labiatus* and 9 *S. mystax* that had been recently captured from the wild, was higher than that observed in callitrichids kept captive for more than 1 year. Nevertheless, there was no correlation between infected animals and the occurrence of diarrhea.[9] However, the isolation of *Campylobacter* spp. was correlated with the development of atrophic lymphoplasmocytic enteritis with fatal outcome, in a private callitrichid breeding colony (Dr. L.R.M. Sá, personal communication, 1999).

Recently, an outbreak of leptospirosis was detected, affecting one *Saguinus midas niger,* two *Saguinus midas*

midas, and one *Pithecia monachus* kept in a zoological park in the state of São Paulo.[24] Leptospirosis is considered to be a rare disease among the platyrrhini.

Finally, mycobacterioses are considered unusual diseases among the platyrrhini.[18] Case reports of individuals kept in South American collections are rare. Recently, one case of tuberculosis caused by *Mycobacterium tuberculosis* was described in a group of four captive *C. apella.*[26] Spontaneous mycobacteriosis was reported in one *Aotus trivirgatus* that was captured in the wild and kept in captivity for 7 years in a Peruvian primate center.[10] In research on the efficacy of tuberculin testing in Neotropical primates kept in a zoo in the state of São Paulo, Brazil, all *C. jacchus* tested negative, whereas four individuals, representing 7% of the *C. apella,* showed positive reactions.[6] In a similar survey performed in Peru, all the animals kept in a primate center were subjected to tuberculin testing. The results were contradictory, because five *Saimiri* spp. that were strongly positive to the test were euthanized, but at necropsy no lesions of tuberculosis were found.[16]

Case reports of mycotic diseases in Neotropical primates are relatively scarce. Among the superficial mycotic infections found, those caused by *Microsporum* spp. and *Trichophyton* spp. are considered infrequent findings in platyrrhini. The infection is acquired by direct contact with infected animals or fomites. Diagnosis is based on cutaneous lesions associated with the cytopathological/histopathological findings of compatible structures and fungal culture.[8]

Fungal organisms include *Candida* spp., *Coccidioides immitis, Histoplasma capsulatum, Cryptococcus neoformans, Paracoccoidioides braziliensis,* and *Sporothrix schenkii.*[8,11,22] As a general rule, with the exception of candidiasis, the lesions induced by these agents are pyogranulomatous to granulomatous, with the involvement of multinucleated giant cells. More often, candidiasis brings about predominantly neutrophilic responses.[8]

Recent epidemiological surveys of the wild mammal collection kept in a zoological park in São Paulo, Brazil, revealed positive reactions to intradermal tests for sporotrichin in 6% of the *C. apella* individuals and in 6.25% and 15% of *C. jacchus* and *C. apella,* respectively, when tested for histoplasmin.[2,3]

Finally, descriptions of deep fungal infections caused by filamentous fungi, especially aspergillosis and mucormycosis, are particularly scarce. Aspergillosis is caused by saprophytic fungi of the genus *Aspergillus* (*Aspergillus fumigatus*). Mucormycoses are processes generated by cosmopolitan opportunistic pathogens, belonging to the genera *Absidia, Mucor, Rhizopus, Rhizomucor,* and *Mortierella.*[8] The author is not acquainted with any publication on deep filamentous mycosis in platyrrhini collections kept in South America, but microscopic lesions compatible with mucormycosis were recently found in *C. jacchus* (Dr. L.R.M. Sá,personal communication, 1999).

Aspergillosis and mucormycosis produce granulomatous lesions, with involvement of multinucleated giant cells. Vascular invasion and thrombosis are characteristic findings of mucormycosis.[8] Presumptive diagnosis of these mycoses may be made through morphological characteristics of hyphae upon histopathological examination, associated with the type of lesion and culture.

REFERENCES

1. Anderson, D.C.; and McClure, H. 1993. Focal epithelial hyperplasia, chimpanzees. In T.C. Jones, U. Mohr, and R.D. Hunt, eds., Monographs on Pathology of Laboratory Animals: Nonhuman Primates I. New York, Springer-Verlag, pp. 233–237.
2. Costa, E.O.; Diniz, L.S.M.; Dagli, M.L.Z.; and Arruda, C. 1991. Delayed hypersensitivity test: Paracoccioidioidin in Latin America wild mammals considering habits terrestrial X tree-dwellings. In Proceedings of the World Veterinary Congress. Rio de Janeiro, Brazil, World Veterinary Association, p. 320.
3. Costa, E.O.; Diniz, L.S.M.; Netto, C.F.; Arruda, C.; and Dagli, M.L.Z. 1994. Epidemiological study of sporotrichosis and histoplasmosis in captive Latin America wild mammals, São Paulo, Brazil. Mycopathologia 125:19–22.
4. Diniz, L.S.M. 1997. Primatas em Cativeiro, Manejo e Problemas Veterinários: Enfoque para Primatas Neotropicais. São Paulo, Ícone.
5. Diniz, L.S.M.; and da-Costa, E.O. 1995. Health problems of *Callithrix jacchus* in captivity. Brazilian Journal of Medical and Biological Research 28:61–64.
6. Diniz, L.S.M.; da-Costa, E.O.; and Fava Netto, C. 1994. Importância e avaliação do teste de hipersensibilidade do tipo tardio—tuberculina—em mamíferos selvagens mantidos em cativeiro, São Paulo, Brasil. A Hora Veterinária 82:52–54.
7. Dubois, R. 1996. Zoonoses transmissíveis por primatas no Brasil. [Transmissible zoonosis from primates in Brazil]. A Hora Veterinária 90:21–24.
8. Gibson, S.V. 1998. Bacterial and mycotic diseases. In B.T. Bennett, C.R. Abee, and R. Henrickson, eds., Nonhuman Primates in Biomedical Research: Diseases. San Diego, California, Academic Press, pp. 59–110.
9. Gozalo, A.; Block, K.; Montoya, E.; Moro, J.; and Escamilla, J. 1991. A survey for *Campylobacter* in feral and captive tamarins. In A. Ehara; T. Kimura; O. Takenaka; and M. Iwamoto, eds., Primatology Today. Amsterdam, Elsevier Science, pp. 675–676.
10. Gozalo, A.; King, N.; and Montoya, E. 1993. Possible spontaneous mycobacteriosis in an owl monkey. Journal of Medical Primatology 23:58–59.

11. Gozalo, A.; King, N.W.; Chandler, F.W.; and Montoya, E. 1994. Paracoccidioidomicosis natural en un mono *Aotus* (Primates: Cebidae) [Spontaneous paracoccidioidomycosis in an *Aotus* monkey (Primates: Cebidae)]. Revista de Investigationes Pecuarias (IVITA). 7:54–56.
12. Gozalo, A.; and Montoya, E. 1990. Mortality causes of owl monkeys (*Aotus nancymae* and *A. vociferans*) in captivity. Journal of Medical Primatology 19:69–72.
13. Gozalo, A.; and Montoya, E. 1991. *Klebsiella pneumoniae* infection in a New World nonhuman primate center. Laboratory Primate Newsletter 30(2):13–15.
14. Gozalo, A.; and Montoya, E. 1992. Mortality causes of the moustached tamarin (*Saguinus mystax*) in captivity. Journal of Medical Primatology 21:35–38.
15. Gozalo, A.; Montoya, E.; and Revolledo, L. 1992. *Pasteurella haemolytica* infection in a Goeldie's monkey. Journal of Medical Primatology 21:387–388.
16. Gozalo, A.; Montoya, E.; Southers, J.; and Revolledo, L. 1992. Non-specific tuberculin test reactions in New World monkeys. Laboratory Primate Newsletter 31(4):8–10.
17. Mansfield, K.; and King, N. 1998. Viral diseases. In B.T. Bennett, C.R. Abee, and R. Henrickson, eds., Nonhuman Primates in Biomedical Research: Diseases. San Diego, California, Academic Press, pp. 1–57.
18. Montali, R.J.; and Bush, M. 1999. Diseases of the Callithrichidae. In M.E. Fowler and E. Miller, eds., Zoo and Wildlife Medicine: Current Therapy, Vol. 4. Philadelphia, W.B. Saunders, pp. 369–376.
19. Montali, R.J.; Connolly, B.M.; Armstrong, D.L.; Scanga, D.A.; and Holmes, K.V. 1995. Pathology and immunohistochemistry of callitrichid hepatitis, an emerging disease of captive New World primates caused by lymphocytic choriomeningitis virus. American Journal of Pathology 148:1441–1449.
20. Montali, R.J.; Ramsay, E.C.; Stephensen, C.B.; Worley, M.; Davis, J.A.; and Holmes, K.V. 1989. A new transmissible viral hepatitis of marmosets and tamarins. Journal of Infectious Diseases 160:759–765.
21. Pachaly, J.R.; Werner, P.R.; and Diniz, J.M.F. 1991. Infecção natural por *Herpesvirus hominis* em *Callithrix jacchus jacchus* Callithrichidae (Thomas, 1903)—Primates [Natural infection due to *Herpesvirus hominis* in *Callithrix jacchus jacchus* Callithrichidae (Thomas, 1903)—Primates]. A Hora Veterinária 61:11–12.
22. Potkay, S. 1992. Diseases of the Callitrichidae: A review. Journal of Medical Primatology 21:189–236.
23. Sá, L.R.M.; DiLoreto, C.; Leite, M.C.P.; Wakamatsu, A.; Santos, R.T.M.; and Catão-Dias, J.L. 1999. Oral focal epithelial hyperplasia in howler monkey (*Alouatta fusca*). Verhandlungsbericht des Internationalen Symposiums über die Erkrankungen der Zoo- und Wildtiere 39:463.
24. Sá, L.R.M.; Teixeira, R.; DiLoreto, C.; and Catão-Dias, J.L. 1999. Leptospirosis in neotropical primates. In Resumos do 3° Congresso da Associação Brasileira de Veterinários de Animais Selvagens. Sao Pedro, Brazil, Associação Brasileira de Veterinários de Animais Selvagens, p. 7.
25. Scanga, C.A.; Holmes, K.W.; and Montali, R.J. 1993. Serological evidence of infection with lymphocytic choriomeningitis virus, the agent of callitrichid hepatitis, in primates in zoos, primate research centers, and a natural reserve. Journal of Zoo and Wildlife Medicine 24:469–474.
26. Tury, E.; Muniz, J.A.P.C.; Brigídio, M.C.O.; Freitas, J.A.; and Souza, J.S. 1998. Tuberculose causada pela *Mycobacterium tuberculosis* entre macacos (*Cebus apella*) mantidos em cativeiro: Observações clínico-anatômicas e histopatológicas [Tuberculosis due to *Mycobacterium tuberculosis* in *Cebus apella* in captivity: Clinical and pathological considerations]. A Hora Veterinária 104:54–57.

MEDICINE, SELECTED DISORDERS

Alcides Pissinatti

TOXICITIES

Accidental poisoning may occur during reintroduction projects. Two accidents were reported during the reintroduction of golden-lion tamarins, *L. rosalia,* in Poço das Antas National Reserve in Brazil. Gastric dilation resulting from fermentation and poisoning after ingestion of large amounts of wild fruits was suspected in one case. Another accident was a fatal case of snake bite.

PARASITIC DISEASES

Parasitic diseases are recognized as the most common necropsy findings in New World monkeys. The following parasitic diseases or conditions have been diagnosed at the Rio de Janeiro Primate Center (CPRJ).

For amebiasis and giardiasis, water and food may be the source of contamination. Measures of control include food hygiene, sanitary education, and periodic medical examination of animals and caretakers.

Cestodiasis is an uncommon finding, probably because of periodic antiparasitic treatments, implemented two to three times a year. However, severe massive infection by *Hymenolepis* sp. was diagnosed in a *C. apella nigritus* that belonging to a private owner. Death resulted from duodenal perforation, enteritis, and peritonitis. Cestodes belonging to the Anoplocephalidae and Hymenolepididae families were found in free-living *Callicebus personatus nigrifons.*[3]

Cases of filariasis occur, mainly in animals originating from northern Brazil. Pleuritis and peritonitis may occur.

Strongyloides stercoralis infection was diagnosed in a *Brachyteles arachnoides* kept as a pet. Clinical signs included weakness, chronic diarrhea, lethargy, and dehydration. Necropsy findings include diffuse peritonitis, acute, severe, diffuse, hemorrhagic, necrotizing enterocolitis, intestinal perforation, esophagitis, and pulmonary hemorrhage and emphysema.

Pancreatic nematodiasis causing death has been described in *Leontopithecus chrysopygus, C. jacchus, S. fuscicollis,* and *Saguinus nigricollis*. Pulmonary nematodiasis caused by *Filariopsis barretoi* have been described by the author in marmosets and capuchin monkeys. Capillariasis is a less common endoparasite of Neotropical primates, but it has been found in marmosets. Pentastomid cysts were reported in the lungs, liver, and serosal surfaces of *L. rosalia*[1] and also in *Saguinus fuscicollis, S. nigricollis*, and *Saimiri sciureus*.

Infections caused by the Acantocephala parasite *Prosthernorchis elegans* have occurred in a few instances in certain species of New World primates at the CPRJ. This parasite adheres firmly to the intestinal wall, causing severe enteritis, formation of fibrous nodules, and intestinal perforation, leading to hemorrhage, peritonitis, and death.

The use of anthelmintics and good hygiene is essential to control endoparasites in captive New World primates.

NONINFECTIOUS DISEASES

Problems Associated with Capture and Shipping

Traumatic lesions may occur as a consequence of illegal hunting using firearms, capturing of newborns, shipping, inadequate management in captivity, and during either confiscation or acquisition of simians sold in illegal markets. Synechiae between internal organs may occur in animals tied by the waist and suspended in a pendular way at the time they are offered for sale in the black market. This also affects parturition, as synechiae between the uterus and urinary bladder will prevent normal uterine contractions, leading to death of the fetus and even of the mother.

Some strongly territorial animals, although sociable, may fight if they are put together in the same shipping box, or even in larger cages while waiting for transportation. Rectal prolapse may occur after traumatism, mainly in young animals, in association with hypoproteinemia or parasitism in clinically unattended nonhuman primates.

Care should be taken to avoid fractures, joint luxation, teeth fracture, and internal wounds during restraint.

Stress

Several problems observed in nonhuman primates kept in captivity are caused by stress, usually associated with inadequate management during illegal possession of these animals. The following conditions have been seen in Neotropical simians sold in illegal markets or as a result of mishandling by private owners.

Gastric dilatation (bloat syndrome) leading to death has been observed by the author in *Saguinus midas niger.*[9] This seems to be a frequent condition that has been described in *S. midas, C. jacchus, Saimiri* sp., and in *C. apella nigrittus*.[6,8] It occurs when the animal's routine is suddenly changed. The mechanism involved appears to be high psychological pressure under abnormal conditions in the group, leading to exaggerated food consumption in a short period of time. The condition may advance to severe gastric dilation, gas formation and hemorrhage, hyperemia of several organs, and pulmonary edema and emphysema.[4,6,8]

Alopecia and self-mutilation may be related to stress.

Reproductive Problems

In platyrrhini and catarrhini, the parturition period begins in August and is concentrated from September to November in the Southern Hemisphere, and from March to May in the Northern Hemisphere. During this period, dystocia, abandonment of newborns, agalactia, and killing of newborns by the mother may occur. In *Leontopithecus* spp. maintained under the identical handling and feeding conditions, dystocia associated with large fetal size has been reported, most frequently in *L. chrysomelas*.[7] A study on sexual dimorphism of the pelvic bones in three species of this genera found short pelvic bones in *L. chrysomelas* that, associated with large fetal size, explains the frequent obstetric problems observed in this species when maintained in captivity. Only a few cases of vaginal prolapse have been observed by the author. The prevalence of abortion is also low in Neotropical species. Dystocia and posterior presentation are problems more frequently seen.

Metabolic Disorders

The presence of gallstones associated with gallblader septation has been reported in callitrichids. High levels of cystine and calcium oxalate were found in these animals.[5]

Diabetes mellitus characterized by severe diffuse degeneration of the islets of Langerhans has been diagnosed in *L. chrysomelas* and *L. chrysopygus*. Fatty changes of the liver, atherosclerosis of the medial and inner layers of the aorta, chronic bilateral interstitial nephritis, and hydronephrosis were also observed in these tamarins.

Fibrous osteodystrophy has been reported in marmosets (*Callithrix* spp.).[2] Animals were weak, with osseous demineralization, multiple fractures, and bowed bones. Microscopically, high osteoclastic activity leading to osseous demineralization, parathyroid hypertrophy, and membranous glomerulonephritis and chronic interstitial nephritis were present. Rickets is a commonly observed disorder of nonhuman primates maintained as pets.

REFERENCES

1. Cosgrove, G.E.; Nelson, B.M.; and Self J.F. 1970. The pathology of pentastomid infection in primates. Laboratory Animal Care 20(2):354–360.
2. Cruz, J.B.; Pissinatti, A.; Nascimento, M.D.; and Campello Costa, C.H. 1993. Osteodistrofia fibrosa em híbridos de *Callithrix* (Erxleben, 1777). Primates: Callitrichidae(Relato de 2 casos [Case report—Fibrous osteodystrophy in hybrid marmosets]. In M.B.C de Souza and A.L.L. Menezes, eds., A Primatologia no Brasil, No. 6. Natal, Yamamoto Universidade Federal do Rio Grande do Norte, pp. 241–248.
3. Melo, A.L.; Neri, F.M.; and Ferreira; M.B. 1997: Helmintos de sauás, *Callicebus personatus nigrifrons,* coletados em resgate faunístico durante a construção da usina hidrelétrica Nova Ponte [Helmints of free-living *Callicebus personatus nigrifrons* from the Nova Ponte Hydroelectric Dam Rescue Project, Brazil]. In M.B.C Souza and A.L.L. Menezes, eds., A Primatologia no Brasil, No. 6.,Universidade Federal do Rio Grande do Norte, pp. 193–198.
4. Pissinatti, A.; and Tortelly, R. 1984. Alterações produzidas por *Porocephalus crotali* (Humboldt, 1811) em *Leontopithecus rosalia* [Lesions caused by *Porocephalus crotali* in golden-lion tamarins, *Leontopithecus rosalia*]. In M.T. de Mello, ed., A Primatologia no Brasil, No. 1, Proceedings of the 1st Congresso Brasileiro de Primatologia. Belo Horizonte, Sociedade Brasileira de Primatologia, pp. 253–257.
5. Pissinati, A.; Cruz, J.B.; Nascimento, M.D.; Silva, R.R.; and Coimbra-Filho, A.F. 1992. Spontaneous gallstones in marmosets and tamarins (Callithrichidae—Primates). Folia Primatologica 59(1):44–50.
6. Pissinatti, A.; Silva, R.R.; Coimbra-Filho, A.F.; and Cruz, J.B. 1986. Dilatação gástrica aguda em *Saguinus midas niger* (Geoffroy, 1803), Callitrichidae, Primates [Acute gastric dilatation in *Saguinus midas niger*]. Revista Brasileira de Medicina Veterinária 8(5):154–157.
7. Pissinatti, A.; Silveira, A.K.; Coimbra-Filho A.F.; and Silva, R.R. 1984. Acerca de cesáreas em símios do gênero *Leontopithecus* genera *Leontopithecus*). Revista Brasileira de Medicina Veterinária 1(1):9–19.
8. Potkay, Y.S. 1980. Transportation, non-infection diseases and nutritional management of neotropical nonhuman primates. Paper presented at the Workshop on the Management and Production of Nonhuman Primates, Iquitos, Peru, National Institute of Health, Maryland.
9. Soave, O.A. 1978. Observations on acute gastric dilatation in nonhuman primates. Laboratory Animal Science 28:331–334.

REPRODUCTION

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INTRODUCTION

Neotropical nonhuman primates have variable endocrine and reproductive strategies of interest to clinicians and scientists. Some of these species are the object of systematic and consistent studies by researchers in such areas as reproductive endocrinology, biotechnology, pharmacology, physiology, immunology, and animal behavior. As a result, some of these species, such as *C. jacchus*[14] and *C. apella,*[25] have even been proposed as biological models for studies related to reproduction endocrinology.

Research on the reproduction of New World primates in captivity is important for several reasons. One is to provide data to increase the reproductive performance of certain species that may be used in biomedical research, preventing exploitation from the wild. Another reason is to maintain genetic diversity of endangered species. Thus, it is vitally important to develop technologies that can be applied to preservation of germ plasma, as well as adapted to the modern artificial breeding techniques originally developed in domestic animals, such as semen and oocyte collection and freezing, artificial insemination, in vitro fertilization, embryo transfer, and even cloning.

This section will present only general aspects related to the reproduction of New World primates, such as anatomy of the reproductive system, reproductive endocrinology, artificial breeding techniques, the use of noninvasive methods for endocrine and behavioral studies, and contraceptive methods. This is an attempt to provide general, basic information without trying to exhaust the subject or even to present a complete review.

ANATOMICAL ASPECTS

Males

All New World primate males have similar reproductive anatomy features, with the most significant variation found in the shape of the glans and proportional penis size. They have a pair of large seminal vesicles, one bilobed prostate, and a pair of bulbourethral glands. Both testicles are in the scrotum. Some species, such as *Cebus sp.* and *Ateles sp.*, have a vestigial penile bone, the os penis, a primitive evolutional characteristic found in some mammals.

Females

All female New World primates have a pair of ovaries, two uterine tubes, and a single cavity uterus. The vaginal canal is short and straight, with most of the differences being found in the size and shape of the clitoris. In some species, as in *Ateles* sp., the clitoris is large and pendulous, and from a certain distance may be confused with the male penis. In some species, there are bone remnants in the clitoris, the os clitoris, as in *Ateles sp.* and *Cebus sp.* The discoid placenta of these species is hemochoriol or deciduate, which is the most efficient model of maternal-fetal exchanges. This characteristic is probably related to the chimerism sometimes seen between twin siblings of different sexes (*C. jacchus* and *L. rosalia),* assumed to be a consequence of placental anastomosis.[2]

REPRODUCTIVE ENDOCRINOLOGY

Most males do not have a seasonal variation in sperm production and often copulate with several females at any time during the day. The males of some species of New World primates have higher levels of circulating testosterone than males of Old World primates. *C. apella* has seven times higher levels.[25] The social hierarchy within the group and dominance relationships influence reproductive performance. As in other primates, semen coagulates after ejaculation due to the presence of secretions from the anterior lobe of the prostate. Several authors have proposed different semen solubilization techniques, because a large number of spermatozoa remain trapped in the coagulum.[5,25,31]

Female New World primates present a large variation in physiological reproductive parameters, but some characteristics are common. They all have a menstrual ovarian cycle. For a long time it was thought that only the Old World primates menstruate, because an abundant vaginal bloody discharge was easily seen, whereas no discharge was observed in New World females. This phenomenon is due to the fact that Neotropical females do not have spiral arterioles in the endometrium, as Old World females do. In Old World primates, menstruation is preceded by vasoconstriction of the spiral arterioles, resulting in an endometrial necrosis and an endothelial lesion in the vessels. When the arterioles relax, there is obvious bleeding. It is presently known that this hemorrhage is stimulated by the local release of vasodilators.[35] Menstrual bleeding of New World primates may be detected by using vaginal swabs to collect material for cytology under light microscope, where an increase in the number of red blood cells may be seen.

The onset of puberty and ovarian cycling are largely influenced by factors related to the social environment and individual behavior in the group. The presence of an adult male to trigger but not to maintain the cycling activity of the ovaries has been described in *S. oedipus.*[39] In the same way, circumgenital odors of dominant *C. jacchus* females have been described as interfering with the reproductive behavior of other females and males in the group.[36] There may be natural cycle synchronization among females maintained in the same environment, although physically separated, which was described in *L. rosalia.*[23]

As was mentioned for the plasma testosterone levels in males, circulating progesterone levels found in *C. apella,*[25] *Aotus trivirgatus,*[4] *Saimiri*sp.,[40] and *Saguinus* sp.[27] females are approximately 8 to 12 times higher than those found in females of Old World primates and 6 to 10 times higher than described for humans.

For most species, only one oocyte is released in each cycle, alternating between the two ovaries, twins being rare. Important exceptions are *Callithrix* sp.,which release two or three oocytes in most cycles; consequently birth of twins or even triplets is not uncommon.

Artificial Breeding

As already mentioned, one of the reasons for studying reproduction in different primate species is to find ways to adapt the techniques used in artificial breeding of domestic animals to the anatomical, physiological, and management conditions of each primate species. Most studies using artificial reproduction methods in primates have involved Old World primates, and only a few have used New World primates.

The methods most often studied in New World primates are semen collection by electroejaculation in *Saimiri sciureus,*[3] *C. apella,*[12,25] *Callicebus moloch,* and *Saimiri sciureus,*[11]and semen freezing.[9,34] Embryo transfer after in vivo and in vitro fertilization has been studied in *C. jacchus.*[21]

Noninvasive Techniques for Reproduction Studies

Extraction and measuring of sexual steroids or their metabolites from feces or urine are among the noninvasive techniques more widely used in reproductive endocrinology studies related to behavioral aspects. Samples may be collected every day or several times per day without physical or chemical restraint, and these techniques may be used in captive and free-living animals.

Most studies using these methods were developed with Old World primates. Only the more recent involved New World primates such as *Cebus sp,*[7,13,16] *Saguinus oedipus oedipus,*[42] *Leontopithecus rosalia rosalia,*[10,30] *Leontopithecus chrysomelas, C. jacchus,*[44] *Cebuella pygmaea,*[43] *C. goeldii,*[17,28,29] *Saguinus labiatus, L. rosalia,*[29] *Saguinus fuscicollis,*[15] *Pithecia pithecia,*[33] *Saguinus labiatus,*[19] *S. oedipus,*[44] and *Brachyteles arachnoides.*[37,45] Estrogens, progestogens, androgens, and gonadotrophins (LH) are the more frequently studied hormones.

The most used methods for fecal extraction in the laboratory are chemical extraction with ethanol and petroleum ether (progestogens), followed by hydrolysis and solvolysis (estrogens), and later separation and purification by high-performance liquid chromatography (HPLC). Then analysis is made by radioimmunoassay or immunoenzymatic methods. The study of hormones in urine uses similar procedures; that is, samples undergo solvolysis to remove steroid conjugates (glucoronides and sulphates), followed by purification by chromatography, and measurement by radioimmunoassay or immunoenzymatic methods. In general, the study of gonadotrophins (LH) involves in vitro assay in rat interstitial cell culture (RICT) to measure testosterone production as an indirect way of separating the bioactive and inactive LH isoforms.[16]

More recently, immunoassay using monoclonal antibodies for measuring gonadotrophins in urine was proposed.[43] In all cases it is important to remember that values obtained for any hormone in urine should be corrected to correlate with creatinine levels in every sample. The use of ultrasound is another recent noninvasive method to study ovarian function in New World primates.[26]

Contraception

Contraceptive methods have been used in primates to control the population of certain species for a determined period or to prevent reproduction of certain individuals with excessive genetic representation in a given population. Many studies have been conducted and results analyzed in order to establish safe and efficient protocols for the use of contraceptive methods. Based on the recommendations and considerations of the Contraceptive Committee of the American Association of Zoos, Parks and Aquariums (AAZPA) for New World primates, the methods can be divided into two major classes:

1. Permanent or nonreversible contraception–In general, nonreversible methods involve surgical sterilization, which includes ovariosalpingohysterectomy and vasectomy. Gonadectomy is not recommended in males, because it may lead to ruptures in the social hierarchy structure and some secondary sexual characteristics may be lost. In females, gonadectomy without uterus removal is not recommended, because there is a potential for infections in a postpuberty uterus with atony. The risks involved are small, as in any minor surgical procedure.

   Vas deferens plugs. These plugs are being studied, involving the surgical placement of silicone plugs in the lumen of the deferens, preventing the passage of spermatozoa. Preliminary studies indicate that this may be a safe option for permanent or even temporary contraception.

   Immunocontraception is being studied in females, using vaccines against zona pellucida, and it is hoped that in the near future it will be safe to use for contraception in New World primates, either as a permanent or a temporary method.
2. Temporary or reversible contraception—Other than physical separation of males and females, hormone treatments may also be used, such as progestogens such as melengestrol acetate and medroxyprogesterone. Progestogens have been used in injectable form or as silastic implants. However, it is now known that long-term use of hormones may increase the risk of breast or uterine neoplasia. Little information is available on the effects of contraceptive pills formulated for humans, with progestogens associated with estrogens, in nonhuman primates.

   For the New World primate Cebidae, there is no information on the safety of implants of melengestrol acetate or ethinyl-estradiol plus melengestrol acetate. Their use is still experimental, thus they cannot be recommended.

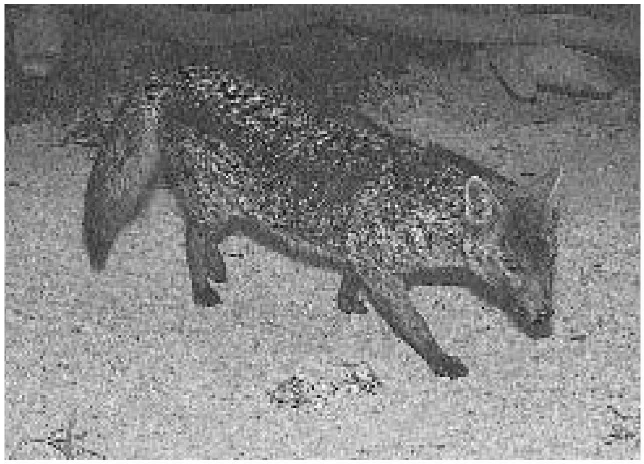
   For Callithricidae, as in the previous group, little information is available on melengestrol acetate implants. At least one retained placenta has been reported, as well as endometritis, in a Callithricidae that became pregnant during treatment with melengestrol acetate.[24] There are also some reports on the return to fertility after the implant was removed in Callithricidae.[1]

It is evident that the use of hormones for reversible contraception in New World primates is still experimental. More information is needed in order to establish a safe and efficient protocol.

REFERENCES

1. Asa, C.S.; and Porton, I. 1990. Primate contraception methods in use and in development. In Proceedings of the American Association Zoo Veterinarians. South Padre Island, Richard Cambre, pp. 263–264.
2. Benirschke, K.; Anderson, J.M.; and Brownhill, L.E. 1962. Marrow chimerism in marmosets. Science 104:513–515.
3. Bennet, J.P. 1967. Semen collection in the squirrel monkey. Journal of Reproduction and Fertility 13:353–355.
4. Bonney, R.C.; Dixson, A.F.; and Fleming D. 1979. Cycle changes in the circulating and urinary levels of ovarian steroids in the adult female owl monkey (*Aotus trivirgatus*). Journal of Reproduction and Fertility 56:271–280.
5. Bush, D.E.; Russel, L.H.; Flowers, A.I.; and Sorensen, A.M. 1975. Semen evaluation in capuchin monkeys (*Cebus apella*). Laboratory Animal Science 25:588–593.
6. Carlstead, K.; Brown, J.L.; Monfort, S.L.; Killens, R.; and Wildt, D.E. 1992. Urinary monitoring of adrenal responses to psychological stressors in domestic and nondomestic felids. Zoo Biology 11:165–176.
7. Czekala, N.M.; Hodges, J.K.; and Lasley, B.L. 1981. Pregnancy monitoring in diverse primate species by estrogen and bioactive luteinizing hormone determinations in small volumes of urine. Journal of Medical Primatology 10(10):1–15.
8. Dettmer, E.L.; Phillips, K.A; Rager, D.R.; Bernstein, I.S.; and Fragaszy, D.M. 1996. Behavioral and cortisol responses to repeated capture and venipuncture in *Cebus apella*. American Journal of Primatology 38:357–362.
9. Durrant, B.S. 1990. Semen collection, evaluation, and cryopreservation in exotic animal species: maximizing reproductive potential. Ilar News 32(1):2–10.
10. French, J.A; Degraw, W.A; Hendricks, S.E.; Wegner, F.; and Bridson, W.E. 1992. Urinary and plasma gonadotropin concentrations in golden lion tamarins (*Leontopithecus rosalia rosalia*). American Journal of Primatology 26:53–59.
11. Fussell, E.N.; Roussel, J.D.; and Austin, C.R. 1967. Use of the rectal probe method for electrical ejaculation of apes, monkeys and a prosimian. Laboratory Animal Care 17(5):528–530.
12. Guimarães, M.A. B.V.; Barnabe, R.C.; and Barnabe, A H. 1995. Contribuição para o estudo da colheita e avaliação do semem de macaco-prego (*Cebus apella*) [Contribution to the study of semen collection and evaluation in capuchin monkeys, *Cebus apella*]. In Proceedings of the 1° Simpósio Brasileiro de Pesquisas em Medicina Veterinária. São Paulo, Brazil, Valquiria Hyppolito Barnabe, p. 103.
13. Guimarães, M.A. B.V. 1999. Ovarian cycle of the capuchin monkey (*Cebus apella*): Techniques of extraction and dosage of fecal progestins and urinary luteinizing hormone. Masters thesis, Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo.
14. Hearn, J.P. 1994. New world primates for research in human reproductive health. American Journal of Primatology 34:11–17.
15. Heistermann, M.; and Hodges, J.K. 1995. Endocrine monitoring of the ovarian cycle and pregnancy in the saddle-back tamarin (*Saguinus fuscicollis*) by measurement of steroid conjugates in urine. American Journal of Primatology 35:117–127.
16. Hodges, J.K.; Czekala, N.M.; and Lasley, B.L. 1979. Estrogen and luteinizing hormone secretion in diverse primate species from simplified urinary analysis. Journal of Medical Primatology 8(6):349–364.
17. Jurke, M.H.; Pryce, C.R.; Döbeli, M.; and Martin, R.D. 1994. Non-invasive detection and monitoring of pregnancy and the post-partum period in Goeldi's monkey (*Callimico goeldii*) using urinary pregnanediol-3α-glucuronide. American Journal of Primatology 34:319–331.
18. Jurke, M.H.; Czekala, N.M.; Lindburg, D.G.; and Milliard, S.E. 1997. Fecal corticoid metabolite measurement in the cheetah (*Acinonix jubatus*). Zoo Biology 16:133–147.
19. Kuederling, I.; Evans, C.S.; Abbott, D.H.; Pryce, C.R.; and Epple, G. 1995. Differential excretion of urinary oestrogen by breeding females and daughters in the red-bellied tamarin (*Saguinus labiatus*). Folia Primatologica 64:140–145.
20. Line, S.W.; Clarke, A S.; and Markowitz, H. 1987. Plasma cortisol of female rhesus monkeys in response to acute restraint. Laboratory Primate Newsletter 26(4):1–4.
21. Lopata, A.; Summers, P.M.; and Hearn, J.P. 1988. Births following the transfer of cultured embryos obtained by in vitro and in vivo fertilization in the marmoset monkey (*Callithrix jacchus*). Fertilty and Sterility 50:503.
22. Mendoza, S.P.; Capitanio, J.P.; McChesney, M.B.; and Lerche, N.W. 1994. Stress, hypothalamic-pituitary-adrenal activity and leucocyte levels. American Journal of Primatology 33(3):228–229.
23. Monfort, S.L.; Bush, M.; and Wildt, D.E. 1996. Natural and induced ovarian synchrony in golden lion tamarins (*Leontopithecus rosalia*). Biology of Reproduction 55:875–882.
24. Munson, L. 1993. Adverse effects of contraceptives in carnivores, primates and ungulates. In Proceedings of the American Association Zoo Veterinarians.St. Louis, Randall E. Junge, pp. 284–288.
25. Nagle, C.; and Denari, J.H. 1982. The cebus monkey (*Cebus apella*). In J. Hearn, ed., Reproduction in New World Primates. Lancaster, Pennsylvania, MTP Press, pp. 39–69.
26. Oerke, A.K.; Einspanier, A.; and Hodges, J.K. 1996. Noninvasive monitoring of follicle development, ovulation, and corpus luteum formation in the marmoset

monkey (*Callithrix jacchus*) by ultrasonography. American Journal of Primatology 39:99–113.
27. Preslock, J.P.; Hampton, S.H.; and Hampton, J.A. 1973. Cyclic variations of serum progestins and immunoreactive estrogens in marmosets. Endocrinology 92:1096–1101.
28. Pryce, C.R.; Schwarzenberger, F.; and Döbeli, M. 1994. Monitoring fecal samples for estrogen excretion across the ovarian cycle in Goeldi's monkey (*Callimico goeldii*). Zoo Biology 13:219–230.
29. Pryce, C.R.; Schwarzenberger, F.; Döbeli, M.; and Etter, K. 1995. Comparative study of oestrogen excretion in female new world monkeys: An overview of non-invasive ovarian monitoring and a new application in evolutionary biology. Folia Primatologica 64:107–123.
30. Ribeiro, E.A.A. 1994. Uma análise da relação entre o comportamento reprodutivo e os níveis de progestinas fecais em um grupo silvestre do mico-leão-dourado, *Leontopithecus rosalia* [A comparison of the reproductive behavior and levels of fecal progestins in a wild group of golden-lion-tamarins, *Leontopithecus rosalia*]. Masters thesis, Instituto de Biociências da Universidade de São Paulo.
31. Roussel, J.D.; and Austin, C.R. 1968. Improved electroejaculation of primates. Journal of the Institute of Animal Technicians 19(1):22–32.
32. Sapolsky, R.M.; and Krey, L.C. 1988. Stress induced suppression of luteinizing hormone concentrations in wild baboons: Role of opiates. Journal of Clinical Endocrinology and Metabolism 66(4):722–726.
33. Savage, A; Lasley, B.L.; Vecchio, A.J.; Miller, A.E.; and Shideler, S.E. 1995. Selected aspects of female white-faced saki (*Pithecia pithecia*) reproductive biology in captivity. Zoo Biology 14:441–452.
34. Seymour, J. 1994. Freezing time at the zoo. New Scientist 1(1910):21–23.
35. Short, R.V. 1987. Oestrous and menstrual cycles. In C.R. Austin and R.V. Short, eds., Reproduction in Mammals: Hormonal Control of Reproduction. Cambridge, Cambridge University Press, pp. 115–152.
36. Smith, T.E.; and Abbott, D.H. 1998. Behavioral discrimination between circumgenital odor from periovulatory dominant and anovulatory female common marmosets (*Callithrix jacchus*). American Journal of Primatology 46:265–284.
37. Strier, K.B.; and Ziegler, T.E. 1997. Behavioral and endocrine characteristics of the reproductive cycle in wild muriqui monkeys, (*Brachyteles arachnoides).* American Journal of Primatology 42:299–310.
38. Whitten, P.L.; Stavisky, R.; Aureli, F.; and Russel, E. 1998. Response of fecal cortisol to stress in captive chimpanzees (*Pan troglodytes*). American Journal of Primatology 44:57–69.
39. Widowski, T.M.; Porter, T.A ; Ziegler, T.E.; and Snowdon, C.T. 1992. The stimulatory effect of males on the initiation but not the maintenance of ovarian cycling in cotton-top tamarins (*Saguinus oedipus*). American Journal of Primatology 26:97–108.
40. Wolf, R.C.; O'Connor, R.F.; and Robinson, J.A. 1977. Cyclic changes in plasma progestines and estrogens in squirrel monkeys. Biology of Reproduction 17:228–231.
41. Wolfensohn, S.; and Lloyd, M. 1998. Handbook of Laboratory Animal Managment and Welfare. Oxford, Blackwell Science, pp. 16–19.
42. Ziegler, T.E.; Sholl, S.A.; Scheffler, G.; Haggerty, M.A.; and Lasley, B.L. 1989. Excretion of estrone, estradiol, and progesterone in the urine and feces of the female cotton-top tamarin(*Saguinus oedipus oedipus*). American Journal of Primatology 17:185–195.
43. Ziegler, T.E.; Matteri, R.L.; and Wegner, F.H. 1993. Detection of urinary gonadotropins in callitrichid monkeys with a sensitive immunoassay based upon a unique monoclonal antibody. American Journal of Primatology 31:181–188.
44. Ziegler, T.E.; Scheffler, G.; Wittwer, D.J.; Schultz-Darken, N.; Snowdon, C.T.; and Abbot, D.H. 1996. Metabolism of reproductive steroids during the ovarian cycle in two species of callitrichids, *Saguinus oedipus* and *Callithrix jacchus,* and estimation of the ovulatory period from fecal steroids. Biology of Reproduction 54:91–99.
45. Ziegler, T.E.; Santos, C.V.; Pissinati, A.; and Strier, K.B. 1997. Steroid excretion during the ovarian cycle in captive and wild muriquis, *Brachyteles aracnoides*. American Journal of Primatology 42:311–321.



26

Order Carnivora, Family Canidae (Dogs, Foxes, Maned Wolves)

Cecília Pessutti
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BIOLOGY

Cecília Pessutti

INTRODUCTION

The first true carnivore to enter South America appeared in the Miocene as an early raccoonlike form. The terrestrial order Carnivora has long been subdivided into two infraorders, the Aeluroidea and the Arctoidea, and two superfamilies, Canoidea and Feloidea. The family Canidae is placed in the Arctoidea (Canoidea). Canids appeared in the Eocene. There are 16 genera today comprised of some 36 natural species, distributed in all land areas of the world except the West Indies, Madagascar, Taiwan, the Philippines, Borneo and islands to the east, New Guinea, Australia, New Zealand, Antarctica, and most oceanic islands. The dog (*Canis familiaris*) was introduced to Australia by aboriginal humans about 4000 to 7000 B.P., possibly later than its introduction into the Western Hemisphere. The living Canidae traditionally have been divided, mainly on the basis of dentition, into three subfamilies: Caninae, with the genera *Canis, Alopex, Vulpes, Fennecus, Urocyon, Nyctereutes, Dusicyon, Cerdocyon, Atelocynus,* and *Chrysocyon;* Simocyoninae, with *Speothos, Cuon,* and *Lycaon;* and Otocyoninae, with *Otocyon.*[13,26,33]

Members of this order tolerate a variety of habitats from hot deserts to the extreme climatic conditions of the Arctic and alpine zones of high mountains. In general the canids differ from most other arctoids in possessing adaptations for running: semirigid, elongate legs ending in four well-developed toes placed close together and tipped with blunt, nonretractile claws that are nearly straight, with the reduced fifth toe (pollex) forming the dew claw and a locked radius and ulna that prevent rotations of the front leg. Thus, canids are cursorial digitigrades, running on their toes or the small pad under their toes.

The shearing carnassial are well developed, with some adaptation for grinding as well. The basic dental formula for canids is I3/3, C1/1, P4/4, M3/3: a total of 42 teeth. The elongate skull, long nose, and powerful cheek muscles are adaptations for seizing, biting, and holding prey. Other cranial characteristics are large bullae with longitudinal septa, a zygomatic process that projects strongly outward, elevated cheeks, smooth tongue, no entepicondylar foramen of the humerus, a small, cartilaginous clavicle, a grooved and well-developed baculum, a cecum coiled into an S-like form, and a complex brain, indicating that these carnivores have a high level of intelligence.

Since canids are adapted for running, they pursue prey with a seemingly effortless gait, and some species are able to take animals larger than themselves. Their food habits are broad, including fruit, invertebrates, and small and large vertebrates, which are opportunistically taken.

In Brazil there are two endangered species. The maned wolf is classified as endangered by Brazilian Institute of Environment (IBAMA) and vulnerable by International Union for the Conservation of Nature (IUCN), the bush dog is listed as endangered by both IBAMA and IUCN.

BIOLOGY

The species found in South America are presented in Table 26.1. A karyological approach to the taxonomy of the Canidae and the origin of the domestic dog seems especially relevant at this time. In many different animal groups, recently acquired knowledge of chromosome number and morphology has furnished important data necessary for an attempt to reconstruct their phyletic evolution and has provided scientists with one more criterion for taxonomic organization. Knowledge of chromosomes is important to the study of phyletic evolution and taxonomy because these structures are direct carriers of genetic information, which has a strict and stable organization. The genera *Canis, Lycaon, Chrysocyon, Atelocynus, Dusicyon,* and *Speothos* seem to be more closely related among themselves than to others. Also, they appear to share morphologically identical X chromosomes and some of the autosomes present similar morphological peculiarities.[8]

Distribution

South American canids have a wide distribution.[20,23,26] The hoary fox, *Lycalopex vetulus,* occurs in Bahia, Goias, Mato Grosso, Minas Gerais, and São Paulo in south central Brazil. The *Pseudalopex* genera has four species: the pampas fox, *Pseudalopex gymnocercus,* in southern Brazil, Paraguay, northern Argentina, Uruguay, and eastern Bolivia; the culpeo fox, *Pseudalopex culpaeus,* in the Andes region from Equador to Patagonia; the Argentine gray fox, *Pseudalopex griseus,* in northern Chile, western Argentina, and Patagonia; the sechuran fox, *Pseudalopex sechurae,* in southwestern Ecuador and northwestern Peru. The crab-eating fox, *Cerdocyon thous,* is found in Colombia, Venezuela, Guyana, French Guyana, Surinam, eastern Peru, eastern Bolivia, Paraguay, Uruguay, northern Argentina, and most of Brazil outside the lowlands of the Amazon basin. The small-eared dog, *Atelocynus microtis,* is found in the Amazon, upper Orinoco, and upper Parana basins in Brazil, Peru, Equador, Colombia, and probably Venezuela. The bush dog, *Speothos venaticus,* is found in Panama, Colombia, Venezuela, the Guyanas, eastern Peru, Brazil (except in the northeast), eastern Bolivia, Paraguay, and extreme northeastern Argentina. The maned wolf, *Chrysocyon brachyurus,* is found in central and eastern Brazil, eastern Bolivia, Paraguay, northern Argentina, and Uruguay.

Habitat Requirement

Canids use a wide variety of habitats, including grassy savannas, smooth uplands, steppes, hills and mountains as high as 4500 m, woodlands and forests, swamps, dry rough country, deserts, and transitional areas.[10,20,23,26,27]

TABLE 26.1. Natural history and cytogenetic characteristics of South American canids

Species	Common Name	Geographic Range	Habitat	Chromosomes Total	Acrocentric: Metacentric
Chrysocyon brachyurus	Maned wolf	NE S. America	High grass plains	76	74:0
Speothos venaticus	Bush dog	NE S. America	Rain forest	74	72:0
Lycalopex vetulus	Hoary fox	NE S. America	Varied	74	72:0
Cerdocyon thous	Crab-eating fox	NE S. America	Varied	74	38:34
Atelocynus microtis	Small-eared dog	NE S. America	Varied	74	72:0
Pseudalopex griseus	Argentine gray fox	Andes	Varied	74	72:0
Pseudalopex culpaeus	Culpeo fox	Andes	Varied	74	72:0
Pseudalopex gymnocercus	Pampas fox	E S. America	Varied	74	72:0
Pseudalopex sechurae	Sechuran fox	NW S. America	Varied	74	72:0

Source: See reference 36.

Exploitation[15]

The maned wolf is hunted, but the fur is not highly valued for commercial purposes. Some body parts are used for medical purposes in folk culture.[12] The crab-eating fox is hunted for its pelt, although the pelt has little commercial value. The culpeo fox is extensively trapped and hunted for its pelt. There is a considerable trade in the pelts of the Argentine gray fox, most of which originate in Argentina, or skins are shipped to Argentina. The pampas fox has been heavily hunted and trapped for fur in several countries (Uruguay, Paraguay, Argentina).

Studies in Free-Ranging Populations

The major studies of wild canids have focused on the maned wolf: its ecology, dietary habits, and home range. Studies of other species are being carried out, and many more should be instituted to learn to understand and preserve the canids.

Conservation Efforts

In 1989, the Brazilian Zoological Society created a breeding management plan, initially for the maned wolf. The primary objective was to establish a self-sustaining population in captivity.

Research was conducted on immunology, veterinary medicine, and reproduction. Guidelines were offered to all zoos to improve management, promote environmental education, and establish a cooperative effort with field researchers and regional species survival plans for the maned wolf on other continents. In 1995, the Maned Wolf Breeding Plan was changed to the Brazilian Canids Work Group, which includes other Brazilian canid species.

The Zoological Society of São Paulo State supports the Maned Wolf Program, which focuses on collecting biological samples to establish base information on blood biochemistry, biometry, hematology, endo- and ectoparasites, hemoprotozoa, infectious diseases (salmonellosis), and clinical evaluation (M.S. Gomes, personal communication, 1999).

MANAGEMENT IN CAPTIVITY

Housing

Enclosures for canids should be designed to provide privacy and entertainment and to encourage development of natural behavior. The following specifications are for the maned wolf. The minimum total for an exhibit area is 200 m^2, but zoo and private institution managers are encouraged to build enclosures larger than this. The topography should permit animals to move about easily and incorporate hiding places that allow them to avoid the public. The enclosure should have only one side open to public view. For safety, the barriers should be 2 m high; the materials for the barrier may be concrete, fence, or dry or wet moat. Usually the substrate is grass and soil. Two or more nesting boxes should be placed in the enclosure; the material may be wood or concrete.

The maternity area should be 4 m^2, with natural light and a nesting box. Part of the floor should be covered by concrete and part with earth and grass.

For other canids, the basic Brazilian Institute for Environment and Natural Renewable Resources (IBAMA) recommendations are listed in Table 26.2. The rules adopted for the maned wolf may be adapted for them. However, the author suggests twice as much space as IBAMA recommends for these animals.

Indoor Enclosures[38]

In countries with severe winters, an indoor area is necessary. The minimum dimensions recommended for this enclosure are 16-25 square feet per animal. If more than one adult is housed, each animal must have its own resting box. Resting boxes within these enclosures may be made of wood, but care should be taken to prevent destructive chewing of the boxes. Resting boxes should be large enough for the animal to stand and turn around; 4 feet × 4 feet × 4 feet high is adequate.

Concrete, wood or natural substrates may be used for the floors. If natural substrate is used, some type of litter may be needed (shavings, straw, grass) to absorb

TABLE 26.2. Recommendation of minimal space for canids in captivity

Genus	Area	Number of Animals	Shelter	Holding Areas	Barrier	Nursery	Floor
Lycalopex, Pseudalopex, Dusycion, Cerdocyon, Atelocynus	20 m^2	2	2 m^2	2 m^2	Fence, dry or wet moat	2 m^2	Ground (0.5 m) over concrete

Source: See reference 16.

urine. Bedding may also be necessary during colder weather. Whatever substrate is used should be able to withstand disinfectants, urine, and feces. Animals housed in concrete areas should be provided with an elevated resting bench or the floor should be covered with bedding such as hay or straw.

Natural or artificial lighting should be provided for each area. Some type of auxiliary heating should be provided if temperatures in the area will fall below 4.5°C (40°F), for adult animals. For whelping areas, the minimum must be 7°C (45°F), but may vary between individuals. Heat may be in the form of forced air space heaters, radiant heaters, heating pads or panels, or heat lamps.

Nutrition

Canids are primarily carnivorous, but most of the Neotropical species have omnivorous habits, using a large variety of items in an ecosystem with multiple ecological niches of primary, secondary, and tertiary consumers.[9] The bush dog (*S. venaticus)* and the culpeo fox (*P. culpaeus)* are strictly carnivorous. Diverse foods for South American canids include insects, arthropods, crabs, frogs, eggs, reptiles, birds, mammals, carrion, roots, sugarcane, fruits, and grasses.[4,6,7,11,17,18,21,24,25,31,39]

One of the most important ecological issues involving canids is seed dispersion.[22,24] Some species have a particular item in their diet; the fruit called "fruta-do-lobo" (*Solanum lycocarpum)* represents 40% of the total items consumed by the maned wolf.

Precise nutritional requirements for most of the canids are unknown. Many times they are fed with the same food as domestic dogs or solely with commercial carnivore diets. One of the medical problems associated with commercial carnivore diets that have high protein levels is cystinuria, a disorder that compromises renal function.[5] Diets with a low percentage of protein are preferred to avoid cystinuria, a common problem in North American zoos.[2]

The author studied the digestibility of plant material consumed by the maned wolf and crab-eating fox in captivity, and found that these species have a high capacity to digest and absorb plant material from the mixed diets used in zoological parks.[29] The digestibility capacity for basic nutrients by the crab-eating fox was higher than in the maned wolf, with the exception of nitrogen-free extracts. In glycemic metabolism the two species showed differences. The maned wolf has a glycemic metabolism peculiar to carnivorous animals and the crab-eating fox a glycemic metabolism peculiar to omnivorous animals.

It is suggested that diets should have a low to moderate protein content, between 20-25% dry matter basis (DMB), to reduce the amount of cystine that kidneys must excrete. Because low protein dog foods are often also low in fat, the addition of vegetable oil or cooked chicken fat is recommended to increase the energy density of the diet and help to improve palatability.[1]

Young maned wolves (under the age of 15–18 months) should be offered diets formulated for growing puppies. Lactating females should not be offered diets with crude protein levels lower than 22% (DMB); otherwise, the nutrient demands on the female during milk production may not be met.[1]

To promote oral health, whole prey (rats, mice, chicks) may be offered in small amounts. Oxtail or horse bones with some meat attached are also used to stimulate gums and teeth. The extent to which soft diets promote gingivitis and dental problems in maned wolves has not been determined.[1]

For insectivorous-frugivorous species, the diet should be formulated to include a variety of fruits and insects, such as mealworms (*Tenebrio molitor),* crickets, and termites. The items most often used to feed canids in Brazilian zoos are presented in Figure 26.1.

Reproduction

Most South American canids are solitary animals, but a few species, such as the bush dog, form social groups. Solitary animals pair only during the breeding season. *Pseudalopex* and *Lycalopex* are monoestrous animals with a gestation length of 55–60 days, *Chrysocyon* also is monoestrous, with 63–65 days of gestation. *Speothos* is polyestrous, with a 67-day gestation period. *Cerdocyon* females produce two litters annually; the gestation period is 52–59 days. The reproductive status of *Atelocynus* is unknown.[26]

Despite the fact that the maned wolf and some other canids are solitary in the wild, many zoos house pairs together year round. The behavior of males and females changes during the breeding season, and it is possible to observe such typical breeding behavior as increased scent marking, following, smelling, licking, mounting, copulating, and playing.[30]

Breeding may or may not occur in the presence of caretakers. Pregnancy may be difficult to determine externally in maned wolves, but radiograph or ultrasound can be used to determine pregnancy. Ultrasound has been used successfully 30 days after copulation, whereas radiographs should be made during the final 3 weeks of pregnancy when bone formation is occurring.[37]

Another tool to monitor the breeding season is hormonal analysis. Fecal steroid concentrations reflect the reproductive cycle of females; this noninvasive method has important management implications for the captive population.[35] Fecal samples are easy to collect and provide crucial information.

Males and females may or may not remain together after she gives birth. A behavior evaluation should be

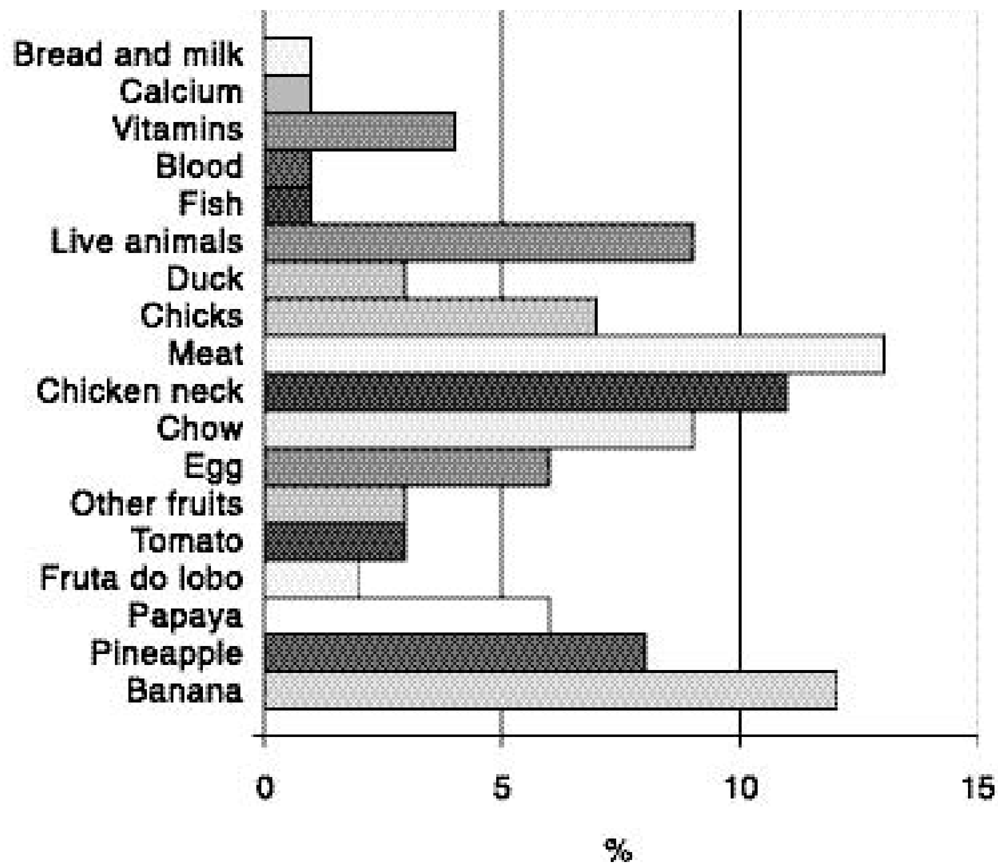

FIGURE 26.1. Percentage of the food items used by Brazilian zoos to feed maned wolves. (Source: Reference 1.)

made before and in the first days after birth, and if the male shows aggressive behavior toward the female and/or the pups, he should be removed, with much care to avoid stressing the female. If the male is calm, he may stay in the same enclosure. Some reports indicate that the male helps care for the pups.[3,34] For the privacy and safety of the female and pups, the enclosure may be isolated from the public by visual barriers and closing the paths around the enclosure. Offspring may remain with the parents until the next breeding season, around 11 months.

The amount of food offered the female during the third trimester and lactation should be increased by half, supplemented with calcium and vitamins in the same proportion as for the domestic dog.

When hand-rearing is necessary, the pups should be fed with a commercial formula, such as Esbilac (Borden Pet-Ag Inc., Norfolk, VA, USA) or a similar goat's milk. A homemade formula may be useful if commercial preparations are not available. The homemade formula contains 250 mL milk, two egg yolks, 50 g powdered baby cereal, and supplements of calcium and vitamin complex in similar amounts as used for domestic canids.[28,30,32] The pups should be fed with a baby bottle or a bottle used for pets. During the first day after birth, an electrolyte solution should be offered to the pups every 2 hours; on the following day they should be fed with a mixture of electrolyte solution and formula 1:1, continuing with the formula only for 30 days. During the first week food is offered every 2 hours, in the second and third weeks, every 3 hours, and in the fourth week every 4 hours. Around this time the pups should begin to eat solid puppy food and the formula may gradually be discontinued. The temperature of the enclosure should be kept around 37°C (98.6°F). Young pups must be stimulated to urinate and defecate. Keepers may gently rub the anogenital area with moistened cotton immediately before and after each feeding.

Most of the recommendations made for maned wolf care may be followed for other canids in breeding management programs, including those for hand-rearing.

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REFERENCES

1. Allen, M.E. 1995. Maned wolf nutritional management. Captive management. In Husbandry Manual for the Maned Wolf, Pt. 3. Front Royal, Maned Wolk SSP, pp. 1–5.
2. Barbosa, P.R.; Allen, M.E.; Rodden, M.; and Pojeta, K. 1994. Feed intake and digestion in the maned wolf (*Chrysocyon brachyurus*). Zoo Biology 13:375–381.
3. Bartmann, W.; and Nordhooff, L. 1984. Paarbindung und elternfamilie beim Mahnenwolf (*Chrysocyon brachyurus,* Illiger, 1811). Zeitschrift des Kölner Zoo. 27(2):63–71.
4. Beccaceci, M.D. 1991. The maned wolf, *Chrysocyon brachyurus.* in Argentina. International Studbook for Maned Wolf. Frankfurt, Frankfurt Zoological Garden, pp. 50–55.
5. Bovée, K.C.; Bush, M.; Dietz, J.; and Segal, S. 1981. Cystinuria in the maned wolf of South America. Science 212:919–920.
6. Brady, C.A. 1978. Observations on the behavior and ecology of the crab-eating fox (C*erdocyon thous*). In J.F. Eisenberg, ed., Vertebrate Ecology in the Northern Neotropics. Washington, D.C., Smithsonian Institution Press, pp. 161–171.
7. Campusano, M.D.L.; and Bacherer, L.S. 1997. Determinacion de la dieta del aguara guazu (*Chrysocyon brachyurus*) en el Parque Nacional Noel Kempff Mercado. In 3° Congresso Internacional sobre Manejo da Fauna Silvestre de la Amazonia. Santa Cruz de La Sierra, p. 21.
8. Chiarelli, A.B. 1975. The chromosomes of the Canidae. In M.W. Fox, ed., The Wild Canids. New York, Van Nostrand Reinhold, pp. 40–53.
9. Crespo, J.A. 1975. Ecology of the pampas gray fox and the large fox (culpeo). In M.W. Fox, ed., The Wild Canids. New York, Van Nostrand Reinhold, pp. 179–191.
10. Dalponte, J.C. 1995. The hoary fox in Brazil. Canid News 3:23–24.
11. Dietz, J.M. 1984. Ecology and social organization of the maned wolf (*Chrysocyon brachyurus*). Smithsonian Contributions to Zoology 392:51.
12. Dietz, J.M. 1987. Grass roots of the maned wolf. Natural History 3:52–59.
13. Eisenberg, J.F. 1989. Order Carnivora (Fissipedia). In J.F. Eisenberg, ed., Mammals of the Neotropics: The Northern Neotropics. Chicago, University of Chicago Press, pp. 262–267.
14. Figueira, C.J.M. 1995. Ocorrência, relações gerais com residentes e comportamento alimentar do lobo guará (*Chrysocyon brachyurus*), em zona rural do sul do estado de Minas Gerais. Monografia em Ecologia. Rio Claro, Universidade Estadual Paulista, p. 39.
15. Ginsberg, J.R.; and Macdonald, D.W. 1992. Foxes, wolves, jackals and dogs: An action plan for the conservation of canids. In Canid, Hyena and Aardwolf. Conservation Assessment and Management Plan (CAMP). Fossil Rim Wildlife Center, pp. 23–32.
16. IBAMA. 1989. Legislação sobre zoológicos. Brasília, IBAMA, p. 28.
17. Jaksic, F.M.; Schlatter, R.P.; and Yáñez, J. 1980. Feeding ecology of central Chilean foxes, *Dusycion culpaeus* and *Dusicyon griseus.* Journal of Mammology 61(2):254–260.
18. Johnson, W.E.; and Franklin, W.L. 1994. Role of body size in the diets of sympatric gray and culpaeo foxes. Journal of Mammology 75(1):163–174.
19. Juarez, K.M. 1997. Dieta, Uso de Habitat e Atividades de Três Espécies de Canídeos Simpátricas do Cerrado. Masters thesis, Universidade de Brasília, p. 59.
20. Langguth, A. 1975. Ecology and evolution in the South American canids. In M.W. Fox, ed., The Wild Canids. New York, Van Nostrand Reinhold, pp. 192–206.
21. Lilienfeld, C.M. 1998. Dieta del Berochi (*Chrysocyon brachyurus*) en el distrito los Fierros del Parque Nacional noel Kempff Mercado. In Anais 13° Jornadas Argentinas de Mastozologia. Porto Iguazu, pp. 100–101.
22. Lombardi, J.A.; and Motta-Junior, J.C. 1993. Seed dispersal of *Solanum lycocarpum* St. Hil. (Solanaceae) by the maned wolf, *Chrysocyon brachyurus* Illiger (Mammalia, Canidae). Ciência e Cultura 45(2):126–127.
23. Mendel, R.G.; and Jaksic, F.M. 1988. Ecología de los cánidos sudamericanos: Una revision. Revista Chilena de Historia Natural 61:67–79.
24. Motta-Junior, J.C.; Lombardi, J.; and Talamoni, S.A. 1994. Notes on crab-eating fox (*Dusicyon thous*) seed dispersal and food habits in southeastern Brazil. Mammalogy 58(1):156–159.
25. Motta-Junior, J.C.; Talamoni, S.A.; Lombardi, J.A.; and Simokomaki, K. 1996. Diet of maned wolf *Chrysocyon brachyurus,* in central Brazil. Journal of Zoology London 240:277–284.
26. Nowak, R.M. 1991. Walker's Mammals of the World, 4th Ed. Baltimore, Johns Hopkins University Press, pp. 1045–1083.
27. Peres, C.A. 1991. Observations on hunting by small-eared (*Atelocynus microtis*) and bush dogs (*Speothos venaticus*) in central-western Amazonia. Mammalogy 55(4):635–639.
28. Pessutti, C.; and Santiago, M.E.B. 1994. Protocolo de Manejo para o Lobo Guará e Resposta, ao 2° Questionário Biológico, Veterinário e Ambiental. Sorocaba, Sociedade de Zoológicos do Brasil, p. 68.
29. Pessutti, C. 1997. Aspectos da fisiologia digestiva de lobo guará *Chrysocyon brachyurus* e cachorro do mato *Cerdocyon thous.* Masters thesis, Universidade Estadual Paulista, Botucatu, p. 65.
30. Pessutti, C. 1991. Respostas ao 1° Questionário Biológico, Veterinário de Manutenção de Lobo Guará e Criação de Filhotes em Cativeiro. Sorocaba, Sociedade de Zoológicos do Brasil, p. 71.
31. Quadros, J.; and Wangler, M.S. 1998. Observações sobre a dieta do lobo guará (*Chrysocyon brachyurus*) em uma área do município de Telemaco Borba, Paraná, Brasil. In Anais 13° Jornadas Argentinas de Mastozologia. Porto Iguazu, p. 107.
32. Rodden, M.; and Rosenthal, M. 1995. Hand hearing and infant development: Captive management. In Husbandry Manual for the Maned Wolf, Pt. 6. Front Royal, Maned Wolf SSP, pp. 1–11.

33. Stains, H.J. 1975. Distribution and taxonomy of the Canidae. In M.W. Fox, ed., The Wild Canids. New York, Van Nostrand Reinhold, pp. 3–26.
34. Veado, B.V. 1997. Parental behaviour in maned wolf at Belo Horizonte Zoo. International Zoo Yearbook 35:279–286.
35. Vellosa, A.L. 1995. The Maned Wolf Reproductive Cycle as Determined by Fecal Steroid Monitoring. Masters thesis, University of Maryland at College Park.
36. Wayne, R.K.; Geffen, E.; Girman, D.J.; Koepfli, K.P.; Lau, L.M.; and Marshall, C.R. 1997. Molecular systematics of the Canidae. Systematic Biology 46(4):622–653.
37. Weinhardt, D.; and Roddewn, M. 1995. Management of reproduction. In Husbandry Manual for the Maned Wolf, Pt. 5. Front Royal, Maned Wolf SSP, pp. 1–10.
38. Werle, L.; Fletchal, N.B.; Weinhardt, D.; and Westbrook, D. 1995. Captive management. In Husbandry Manual for the Maned Wolf, Pt. 2. Front Royal, Maned Wolf SSP, pp. 1–11.
39. Yanes, J.; and Jaksic, F. 1978. Rol ecologico de los zorros (*Dusycion*) en Chile central. Anales del Museo de Historia Natural 11:105–112.

MEDICINE

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RESTRAINT AND HANDLING

Physical Restraint

Physical restraint is recommended for interventions that are simple and of short duration. This practice, if performed inappropriately, may lead to trauma, injury, and even the death of the animal being restrained. Special attention should be paid to problems that may result from stress caused by restraint. Stress may be reflected in the animal's general well-being, behavior, and reproductive success, and may even result in the subsequent death of the animal. South American canids can be restrained with the aid of a net on a pole or a normal net, with its mesh size adapted to the species to be restrained. A catchpole may also be used, but it should be handled by an expert, taking care to include a front limb in the loop that goes around the neck, to avoid asphyxia. The use of a squeeze cage may minimize the danger of physical injury to the animals and the handlers.

Chemical Restraint

Chemical restraint is usually preceded by physical restraint of the animal. Immobilizing agents may be administered by a handheld syringe, pole syringe, or darts from a blowpipe or gun. Ketamine is the drug used most frequently in Brazilian zoos today, because it is easy to obtain and handle. It is frequently administered with xylazine or acepromazine.[28] Other drugs are listed in Table 26.3.

Inhalation Anesthesia

In long surgical procedures or for animals in a weak state of health, the maintenance of anesthesia with gaseous agents is safer than with an injectable anesthetic, although an injectable anesthetic may be necessary for induction. The volatile agents used are halothane and isoflurane, either of which may be administered through a face mask or an endotracheal tube.

TABLE 26.3. Doses of some anesthetics for South American canids

Medication	Species	Dose (mg/kg/ IM)	Observations	References
Ketamine + xylazine	*Chrysocyon brachyurus*	6 – 8 + 0.5 – 1	Small interventions	25
		3 – 5 + 0.6 – 0.8		[a]Oliveira
	Cerdocyon thous	20 + 1		15
		10 + 05 – 1		[b]Santiago
	Dusycion vetulus	3 – 5 + 0.6 – 0.8		[a]Oliveira
Ketamine + acepromazine	*C. brachyurus*	6 – 8 + 0.11		28
	Speothos venaticus	20 + 0.1		15
Tiletamine + zolazepam	*C. brachyurus*	4 – 7		28
		7.5		18
	S. venaticus	10		15

[a]Oliveira L.T.F., unpublished data.
[b]Santiago, M.E.B., unpublished data.

SURGERY

Surgical techniques are similar to those used in companion animals, since the anatomy of wild canids is similar to that of domestic canids. Fractures and dislocations are the most common orthopedic injuries in wild canids.[8] These injuries have been repaired using internal and external fixation. Among the most common fractures are those found in maned wolves (*C. brachyurus*) run over on highways. A congenital abnormality of the acetabular cavity, which also affected the articulation of the femurotibial joint, was reported in a 50-day-old maned wolf specimen. This was corrected surgically through excision of the patella.[14] In crab-eating foxes closed reductions of tibial fractures caused by snares were carried out using splints.

Wild canids rarely suffer dystocia, and few cases have been verified where surgical intervention was necessary. A hysterectomy in a maned wolf was performed using the same technique as described for domestic dogs, substituting subcuticular sutures for skin sutures to close the skin to prevent the animal from pulling out the sutures. Another procedure that may be adopted to prevent sutures from being removed by the animal is the topical application of a repulsive mixture, the nonilic acid of vanilla and beta-nicolitic-butoxitilester acid (Finalgon Linimento, Boehringer de Angeli Química Farmacêutica Ltda, Itapercerica da Serra, São Paulo, Brazil) along the suture line. The taste is unpleasant and discourages licking of the wound (L.T.F Oliveira, personal communication 1995).

Examples of dermatological lesions that have been observed in maned wolves are sebaceous cysts and epithelial hyperplasia resulting from a nonspecific chronic inflammatory process. In these cases surgical excision is recommended.

DIAGNOSIS

Clinical examination is carried out as for the domestic dog.

Special diagnostic procedures include ultrasound, which is recommended for the diagnosis of pathological conditions, pregnancy, and renal parasitism, since the infected kidney appears to be smaller than the unaffected organ.[6]

Radiography not only aids in the diagnosis of fractures, but also in the diagnosis of other pathological conditions, for example, in dirofilariasis, where an increase in size of the right side of the heart may be indicative of the presence of the parasite.[18]

TABLE 26.4. Hematological values for maned wolves

Parameter	Mean	SD
WBC ($\times 10^3/\mu L$)	11.22	4.626
RBC ($\times 10^6/\mu L$)	5.400	0.986
HGB (gm/dL)	14.6	2.6
HCT (%)	43.1	6.9
MCH (pg)	27.3	1.6
MCHC (g/dL)	34.1	1.4
MCV (fl)	80.2	5.9
Segments ($\times 10^3/\mu L$)	12.22	14.82
Bastonetes ($\times 10^3/\mu L$)	1.731	4.921
Lymphocytes ($\times 10^3/\mu L$)	4.381	6.805
Monocytes ($\times 10^3/\mu L$)	0.277	0.289
Eosinophils ($\times 10^3/\mu L$)	0.577	0.537
Basophils ($\times 10^3/\mu L$)	0.005	0.023
NRCB (WBC) /100	0.4	1.0
Platlets ($\times 10^3/\mu L$)	200.0	0.0
Glucose (mg/dL)	121	27
BUN (mg/dL)	25	8
Uric Acid (mg/dL)	0.6	0.2
CA (mg/dL)	9.7	0.7
PHOS (mg/dL)	5.2	2.0
Na (mEq/L)	146	4
K (mEq/L)	4.8	0.4
CL (meq/L)	113	4
MG (meq/L)	2.5	0.0
Cholesterol (mg/dL)	2.5	0.0
Total protein (gm/dL)	6.4	0.7
Albumn (gm/dL)	3.1	0.8
AST (SGOT) (IU/L)	36	13
ALT (SGPT) (IU/L)	42 24	
Total Bilirubin (mg/dL)	0.3	0.1
Alk. Phos. (IU/dL)	50	43
LDH (IU/L)	262	151
CPK (IU/L)	180	0
OSMO (mosmol/L)	293	5

WBC, white blood cells; RBC, red blood cells; HGB, hemoglobin; GCT, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; NRCB, nucleated red cell blood; BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; Alk. Phos., alkaline phosphate; LDH, lactate dehydrogenase; CPK, creatine phosphate kinase; OSMO, osmality.

Laboratory Samples

Laboratory samples may be collected as in the domestic dog. Table 26.4 lists reference hematological values for the maned wolf.

Diseases

INFECTIOUS DISEASES The infectious diseases of wild canids are similar to those of domestic dogs. Conditions that have been reported in South American wild canids include leptospirosis, salmonellosis, otitis externa, enterovirus, and rabies. Only unique diseases are discussed here.

BACTERIAL DISEASES

Clostridium perfringens. Maned wolves suffer from watery hemorrhagic diarrhea, anorexia, and dehydration when infected by this bacterium. At necropsy, the mesenteric lymph nodes are enlarged and the intestines are hemorrhagic, containing a large quantity of digested blood. Anaerobic culture of liver, kidney, and intestinal contents may isolate the organism.[27] Inappropriately stored food may be the source of infection. Treatment of an affected animal with electrolyte solutions and enrofloxacin at an initial dose of 10 mg/kg subcutaneously (SC), followed by 5 mg/kg SC, every 24 hours (SID), was effective (M.E.B Santiago, unpublished data).

VIRAL DISEASES

Canine Distemper. Outbreaks of distemper affecting canids have been reported in Brazilian zoos. Transmission is primarily aerosol, since infected animals, whether symptomatic or not, excrete the virus in all bodily secretions.[2] Clinical signs include diarrhea, purulent ocular discharge, ataxia, convulsions, and myoclonus of the limbs. Death usually ensues rapidly. Histopathological lesions include lymphocytolysis in the spleen, rarefaction of germinal centers and vacuolar degeneration, necrosis of hepatocytes, and diffuse pneumonia with pulmonary edema, congestion, and hemorrhage. Other findings include infiltrative enteritis and necrosis of enterocytes and severe hemorrhagic meningitis/encephalitis with intracytoplasmic inclusion bodies.[26] In Brazil there are no reports of animals that have survived the disease.

Prevention, through vaccination, quarantine of recently arrived animals, and a ban on domestic canines in wild canid facilities, is the best way to combat the disease. Because of the highly infectious and lethal nature of the disease, affected animals should be isolated immediately and given supportive treatment, using electrolyte solutions and antibiotics to avoid secondary infections. Euthanasia may be considered to prevent the spreading of the virus throughout the collection.

From 1989 to 1994, deaths of maned wolf cubs from distemper were recorded in Brazilian zoos (Figure 26.2). Based on these figures the Committee for Management of Maned Wolves in Captivity recommended vaccination with modified live virus vaccine, cultivated in an avian cell. After introducing the vaccination program, the number of deaths from distemper among maned wolf cubs fell[25,28] (Figure 26.3).

PARASITIC DISEASES Wild canids may have fleas, ticks, sarcoptic mange, protozoal infections, and internal parasites common to domestic dogs.

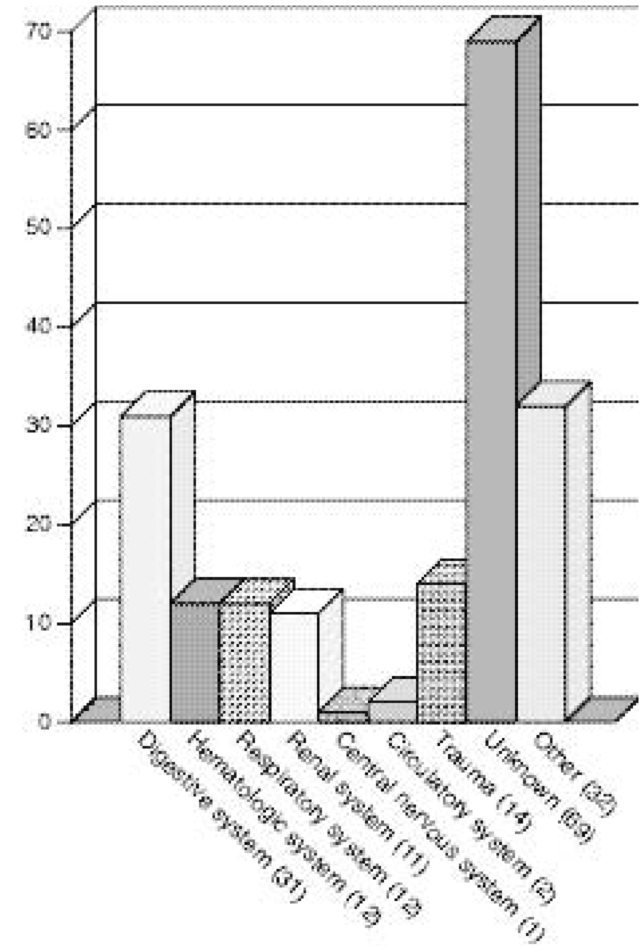

FIGURE 26.2. Causes of mortality in maned wolves in Brazilian zoos from 1994 to 1997. (Source: References 25 and 28.)

Endoparasites. In recent research into the prevalence of parasitic infections in maned wolves in zoos in the state of São Paulo, Brazil, nematodes identified were *Ancylostoma caninum, Strongyloides* spp., *Uncinaria stenocephala,* and *Capillaria* spp.[30] *Toxocara* spp. and *Trichuris* spp., as well as the cestodes *Dipylidium caninum* and *Echinococcus granulosus,* may also infect maned wolves kept in captivity.[18]

Maned wolves are also susceptible to *Dirofilaria immitis*. Diagnosis may be made by microscopic identification of microfilaria in the blood, which has been previously mixed with an isotonic solution of sodium chloride (1 drop of blood to 1 drop of the solution). Ivermectin is the recommended treatment.[18]

Another nematode, *Angiostrongylus vasorum,* which is a parasite of the pulmonary and coronary arteries, was found in a specimen of *Dusicyon vetulus*.[16] Infestation by *Diocthopyme renale* is frequently found in animals captured in the wild, but its importance as a cause of death is not proven.[10]

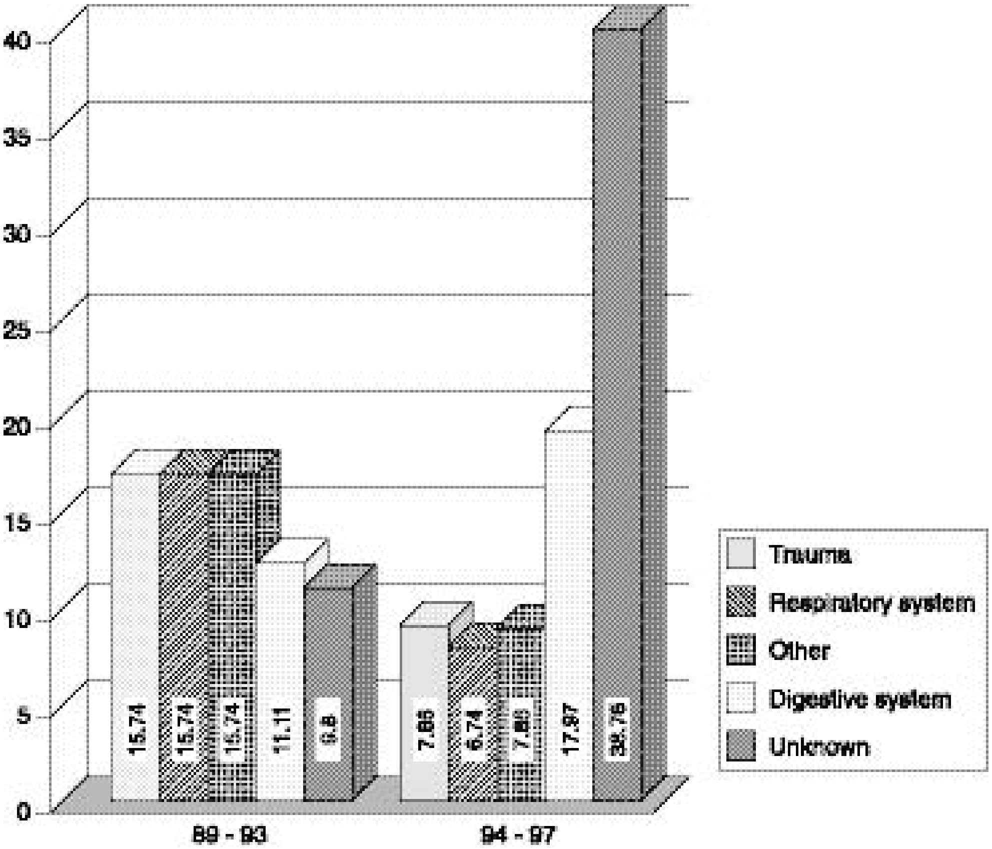

FIGURE 26.3. Comparison of mortality in maned wolves in Brazilian zoos in the periods 1989 to 1993 and 1994 to 1997. (Source: Reference 28.)

In a study carried out on nine free-living maned wolves in the region known as Triângulo Mineiro (Minas Gerais State, Brazil), examination of nematode ova and adult specimens identified *A. caninum* and *U. stenocephala;* this was the first reported occurrence of *U. stenocephala* in maned wolves in Brazil.[21] Infected animals should be treated with the anthelmintic and antiprotozoal drugs listed in Table 26.5.

Noninfectious Diseases

Urolithiasis. This condition involves the formation in the urinary tract of calculi from the precipitation of salts in the urine, usually in association with an organic matrix.[8] One male maned wolf suffering from urolithiasis presented with abdominal distension, recurrent urinary tract infections, urinary incontinence, anorexia, and urinary tenesmus, developing into anuria. On clinical examination, obstruction of the urethra by calculi was observed, and radiological examination revealed distension of the bladder and the presence of numerous calculi. At necropsy, an increase in volume, thickening, and necrosis of the bladder wall were observed, as well as numerous calculi in the lumen and encrusted on the wall of the organ. The urine was hemorrhagic.

The calculi were composed of calcium pyrophosphate, iron phosphate, and magnesium, determined by a diffractometric test. This condition may have been a consequence of previous urinary infections arising from an inappropriate diet during captivity (L.T.F. Oliveira, unpublished data).

Cystinuria. Cystinuria is a metabolic disease associated with excessive excretion of cystine and other amino acids in the urine.[7] This condition has been diagnosed in maned wolf specimens kept in Brazilian zoos, but the majority of animals were asymptomatic. A balanced diet and regular monitoring of the urinary pH and sediments may prevent cystinuria and the formation of calculi.[18]

Miscellaneous Noninfectious Diseases. Other noninfectious diseases include nutritional secondary hyperparathyroidism, neoplasia, and intussusception.

TABLE 26.5. Parasiticides used in maned wolves

Recommended Drugs	Dosage (mg/kg)	Interval/Duration	Reference
Mebendazole	15	24 h/3 days PO	25
Praziquantel	5	single dose PO	25
Pyrantel	5	single dose PO	1
Fenbendazole	50	24 h/3 days PO	25
Piperazine	80 to 100	single dose PO	25
Levamisole	10	single dose PO	25

PO, by mouth.

PREVENTIVE MEDICINE

Vaccination

In Brazil, wild canids are vaccinated against distemper and parvovirus with modified live virus vaccine. To avoid problems arising from the immunization, such as vaccine-induced distemper,[19,20,4] vaccines cultivated in avian cells are recommended.[17] Vaccination against rabies should be carried out using a killed virus vaccine.[28,11] The Canid Working Group suggests the following guidelines:

- Distemper and parvovirus—Three doses at 3-week intervals for cubs, beginning at 45 days of age or after weaning (modified live virus vaccine, cultivated in avian cell), and three doses at 3-week intervals for unvaccinated adults. Annual boosters should be given.
- Leptospirosis—One dose, every 6 months, for animals in zoos that have already had an outbreak of the disease or in cases of outbreaks in areas close to the zoo.
- Rabies—An annual dose for animals in zoos located in endemic areas (killed virus).
- Deworming—Feces should be examined for parasite ova at least twice a year, as well as giving anthelmintics to adult animals. For cubs, dosing with anthelmintics can be started at 45 days old, preferably before the first dose of the vaccine. A dose for cubs every 3 months is recommended.

REFERENCES

1. Allen, D.G.; Pringle, J.K.; and Smith, D.A. 1998. Handbook of Veterinary Drugs, 2nd Ed. Philadelphia, Lippincott Raven, pp. 6, 8, 23, 36, 45, 47.
2. Appel, M.J.G.; and Montali, R.J. 1994. Canine distemper and emerging morbilivirus diseases in exotic species. In Proceedings American Association of Zoo Veterinarians. pp. 336–339.
3. Barbies, R.; and Bush, M. 1995. Medical management of maned wolves. In N.B. Fletchal, M. Rodden, and S. Taylor, eds., Husbandry Manual for the Maned wolf *Chrysocyon brachyurus*.
4. Bittle, J.L. 1993. Use of vaccines in exotic animals. Journal of Wildlife Medicine 24(3):352–356.
5. Buschinelli, M.C. 1992. 1° Workshop sobre Conservação e Manejo de Cachorro Vinagre *Speothos venaticus*. Americana, Sociedade de Zoologicos do Brasil, p. 8.
6. Buschinelli, M.C.P. 1991. Diagnóstico e tratamento de dioctofimose em *Chrysocyon brachyurus*. Arquivos da Sociedade de Zoológicos do Brasil 10/11:20.
7. Bush, M.; and Bovee, K. 1978. Cystinuria in a maned wolf. Journal of the Veterinary Medical Association 173(9):1159–1162.
8. Carlton, W.W.; and Mcgavin, M.D. 1998. Patologia Veterinária Especial de Thonson, 2nd Ed. Porto Alegre, Artes Médicas Sul, p. 261.
9. Cavaliero, T.C.; Santana, A.E.; Malheiros, E.B.; Costa, A.M.; Machado, R.Z.; and Machado, C.R. 1989. Valores hematológicos, bioquímicos e temperatura interna de lobos(guarás—*Chrysocyon brachyurus*). ARS Veterinaria 5(1):25–32.
10. Cubas, Z.S. 1996. Special challenges of maintaining wild animals in captivity in South America. In M.E. Fowler, ed., Wildlife Husbandry and Diseases. Paris, Office International des Epizooties. pp. 267–268. (Scientific and Technical Review 15(1):267–288.)
11. Gandras, R.; and Steeger, H. 1982. Wild dogs and hyenas. In H. Georg-Klös and F.M. Lang, eds., Handbook of Zoo Medicine. Diseases and Treatment of Wild Animals in Zoos, Game Parks, Circuses and Private Collections. New York, Van Nostrand Reinhold, p. 93.
12. Gomes, N.B.N.; Aguiar, P.H.P.; and Costa, M. e L.T. 1992. Adenocarcinoma pulmonar em Lobo guará (*Chrysocyon brachyurus*). Arquivo Brasileiro de Medicina Veterinária e Zootecnia Rio de Janeiro 43(4):247–253.
13. Hoskins, J.D. 1997. Update on canine parvoviral enteritis. Veterinary Medicine Companion Animal Practice. pp. 694–709.
14. Iamaguti, P.; Teixeira, C.R.; Sampaio, G.R; Cruz, M.L.; Mamprim, M.J.; Nunes, A.V.; and Teixeira, R.H.F. 1995. Anomalia da articulação fêmur-tibio-patelar em *Chrysocyon brachyurus:* correção cirúrgica. Arquivos da Sociedade de Zoológicos do Brasil 14/15/16:3.
15. Kreeger, T. J. 1999. Chemical restraint and immobilization of wild canids. In M.E. Fowler, ed., Zoo and Wild

Animal Medicine, Vol. 4. Current Therapy. Philadelphia, W.B. Saunders, pp. 429–435.

16. Lima, W.S.; Lemos, I.S.; and Guimaraes, M.P. 1993. *Angiostrongylus vasorum* (Baillet, 1866) in the fox *Dusicyon vetulus* from Belo Horizonte zoo in Minas Gerais, Brazil. Arquivos da Sociedade de Zoológicos do Brasil 14/15/16:99.
17. Maia, O.B. 1998. Perfil Sorológico e Avaliação Pós Vacinal de Lobo Guará, *Chrysocyon brachyurus,* (Illiger, 1811) para o Vírus da Cinomose e Parvovirose Caninas. Masters thesis, Universidade Federal de Minas Gerais.
18. Matern, B. Guidelines for Keeping the Maned Wolf, *Chrysocyon brachyurus* (Illiger, 1811). Frankfurt, Zoologischer Garten Frankfurt.
19. Mcinnes, E.F.; Burroughs, R.E.; and Duncan, N.M. 1992. Possible vaccine induced canine distemper in South American bush dog (*Speothos venaticus*). Journal of Wildlife Diseases 28(4):614–617.
20. Montali, R.J.; Tell, L.; Bush, M.; Cambre, R.C.; Kenny, D.; Sutherland-Smith, M.; and Appel, M.J. 1994. Vaccination against canine distemper in exotic carnivores: Successes and failures. In Proceedings of the American Association of Zoo Veterinarians. pp. 340–344.
21. Mundin, M.J.S.; Machado, M.J.; Bevilaqua, E.; Mundin, A.V.; Maywald, P.G.; and Oliveira, M.G. 1995. Ocorrência e identificção de ancilostomídeos em lobo guará (*Chrysocyon brachyurus,* Illiger, 1811) da região do triângulo mineiro, Minas Gerais, Brasil. Braz. J. Vet. Res. An. Sel. :39–43.
22. Munson, L.; and Montali, R.J. 1991. High prevalence of ovarian tumors in maned wolves (*Chrysocyon brachyurus*) at the National Zoological Park. Journal of Zoo and Wildlife Medicine 22(1):125–129.
23. Nunes, A.L.V.; and Puglia, L.R.R. 1983. Ocorrência de babesiose em lobo guara (*Chrysocyon brachyurus*) no zoo de Sorocaba. Arquivos da Sociedade de Zoológicos do Brasil 4:7– 8.
24. Nunes, A.L.V. 1991. Babesiose em lobo guará *Chrysocyon brachyurus:* Ocorrência , tratamento e recuperação em dois casos clínicos. Arquivos da Sociedade de Zoológicos do Brasil 10/11:4.
25. Pessutti, C.; and Santiago, M.E.B. 1994. Protocolo de Manejo para o Lobo Guará (*Chrysocyon brachyurus*) e Resposta ao Segundo Questionário Biológico, Veterinário e Ambiental. Sorocaba, Sociedade de Zoológicos do Brasil.
26. Rego, A.A. M. da S.; Matushima, E.R.; Pinto, C.M.; and Biasi, I. 1997. Distemper in Brazilian wild canidae and mustelidae: Case report. Braz. J. Vet. Res. Anim. Sci. São Paulo 34(3):156–158.
27. Santiago, M.E.B.; Margatho, L.F.F.; Baldassi, L.; Calili, E.M.B.; Moulin, A.A.P.; and Melville, P.A. 1997,. Surto de diarréia em filhotes de lobo guará (*Chrysocyon brachyurus*) determinado por *Clostridium perfringens*. Arquivos do Intituto Biológico, São Paulo 64(suppl.):42.
28. Santiago, M.E.B. 1998. Protocolo de Manejo do Lobo Guará (*Chrysocyon brachyurus*). Sorocaba, Sociedade de Zoologicos do Brasil.
29. Serra-freire, N.M.; Teixeira, R.H.F.; Amorim, M.; Gazeta, G.S.; Nunes, A.L.V.; Yada, H.S.; and Teixeira, C. 1995. Babesiose associada ao parasitismo por carrapato em lobo guará. Arquivos da Sociedade de Zoológicos do Brasil 14/15/16:9.
30. da Silva, F. de A.; and Gilioli, R. 1998. Prevalência de infecções parasitárias e bacterias patógenas intestinais em *Chrysocyon brachyurus* (lobo-guará) mantidos em zoológicos no estado de São Paulo. In 6° Congresso Brasileiro da Ciência de Animais de Laboratório. Campinas, Universidade de Campinas, p. 94.
31. Silva, R.A.; and Breckenfeld, S.G. 1968. Ocorrência da raiva em lobo guará (*Chrysocyon brachyurus,* Illiger, 1815). Pesquisa Agropecuária brasileira 3:369–371.
32. Teixeira, C.R.; Ince, A. P.; Bandarra, E.P.; Fiorio, W.A.B.; Nunes, A.L.V.; and Teixeira, R.H.F. 1995. Adenoma de glandula ceruminosas em *Chrysocyon brachyurus.* Arquivos da Sociedade de Zoológicos do Brasil :5–6.

27 Order Carnivora, Family Felidae (Cats)

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BIOLOGY

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INTRODUCTION

The carnivore family Felidae comprises 36–37 wild species, placed under a varying number of genera depending upon the taxonomic scheme used, that are distributed worldwide, with the exception of Australasia, Antarctica, and several oceanic islands.[29,48] In the Neotropical region, which comprises South and Central America and the Mexican tropical lowlands, there are 10 species: ocelot (*Leopardus pardalis,* 10.1 kg), margay (*Leopardus wiedii,* 3.4 kg), little spotted cat or oncilla (*Leopardus tigrinus,* 2.4 kg), Geoffroy's cat (*Oncifelis geoffroyi,* 3.9 kg), kodkod or huiña (*Oncifelis guigna,* 2.2 kg), pampas cat (*Lynchailurus colocolo,* 3.5 kg), Andean cat (*Oreailurus jacobita,* 4 kg), jaguarundi (*Herpailurus yaguarundi,* 5.1 kg), puma (*Puma concolor,* 39.2 kg), and jaguar (*Panthera onca,* 61.4 kg).[31,37] All members of the family share morphological, physiological, and behavioral adaptations for a specialized predatory life history, and all occupy top consumer niches in the ecosystems in which they occur.

In part as a consequence of these characteristics, cats have often come into conflict with human interests, and in many cases have been directly hunted for sport or profit and deliberately persecuted for diverse reasons. As a result of these interactions, all felids (with the exception of the domestic cat, *Felis catus*) are currently considered to be threatened to some extent, and several species are viewed as being critically endangered.[30]

BIOLOGY

Evolution and Taxonomy

Felids are thought to have diverged from other carnivore families around 35 million years before the present (m.y.b.p), and the first appearance of morphologically modern cats in the fossil record was at approximately 25–30 m.y.b.p, in Eurasia. Modern lineages of felids seem to have diverged from each other and dispersed more widely geographically around 10 m.y.b.p in a rather rapid series of diversification events.[47] This pattern of rapid divergence makes it difficult to reconstruct the phylogenetic relationships among the living species, a problem that is accentuated by the visible conservation of morphological features across all members of the family and frequent episodes of convergence in which species that are not closely related develop similar characteristics due to coinciding environmental pressures.[24] These difficulties have led to the multiple conflicting taxonomic schemes proposed for this family in the last few centuries, which were based on divergent interpretations of the available evidence. Classic taxonomic schemes have divided the living cat species into as few as 2, and as many as 19 genera.[29,30,48] Recent studies using morphological analyses[43] and molecular approaches[4,21,40] have clarified many aspects of felid evolutionary relationships. Most of the newest findings have not yet been formally translated into nomenclature propositions for this family, so for the purposes of this chapter we will usually follow the taxonomy suggested by Wozencraft[48] and used by Nowell and Jackson.[30]

The most recent molecular studies[21,40] support the existence of eight major lineages of modern cats, which in many cases agree with previously established taxonomic partitions. Three of these major phylogenetic groups are present in South America, namely the ocelot lineage, the puma lineage, and the *Panthera* lineage. The ocelot lineage is essentially a Neotropical group, comprised by seven species: the ocelot, margay, little spotted cat, Geoffroy's cat, kodkod, Pampas cat, and the Andean mountain cat. This group seems to have evolved and diversified in this region in the last 3–6 million years, during and after the formation of the Panamanian land bridge between North and South America.[22] The phylogenetic relationships and evolutionary history of these species have been extensively investigated in recent years using molecular approaches.[13, 21, 22, 23, 39] These studies have supported the notion that the ocelot and margay are sister species and that the Andean mountain cat is more closely related to this pair than to any of the other species in this lineage. The kodkod and Geoffroy's cat are very closely related to each other, and these two are in turn related to the little spotted cat. The pampas cat seems to be a considerably old species, apparently more closely related to the little spotted cat/Geoffroy's cat/kodkod group than to the ocelot/margay/Andean cat group.[22,23]

The puma lineage is composed of three species: the puma, the jaguarundi, and the cheetah (*Acinonyx jubatus*). The former two are more closely related to each other, and currently occur in South America, whereas the latter is presently restricted to Africa and remnant populations in the Middle East. Fossil evidence, however, indicates that cheetah-like cats, which were probably related to this group, occurred in North America as late as 10,000 years ago.[47] The puma and the jaguarundi seem to have last shared a common ancestor around 5-6 m.y.b.p,[21] and it is at this point unclear whether these two species diverged in North America before entry in South America or during the colonization of the latter continent.

The *Panthera* lineage consists of the five big, roaring cats belonging to the *Panthera* genus, as well as the closely related clouded leopard *Neofelis nebulosa*. The only species of this group currently inhabiting South America is the jaguar, the largest of the Neotropical felids. The phylogenetic relationships of the jaguar and the other species within the *Panthera* genus have been historically impossible to determine and are at present still uncertain, but further research using several molecular methods is underway in an attempt to clarify this issue. The jaguar seems to have entered the American continent fairly recently, less than 1 m.y.b.p, and current jaguar populations seem to have expanded to colonize their present range in an even more recent process, around 280,000 years ago.[49]

Distribution

The geographic distribution of South American felids varies across species. Five species are predominantly tropical, four are mostly temperate, and the puma is a generalist. The tropical species, ocelot, margay, little spotted cat, jaguarundi, and jaguar, range overall from the lowlands of tropical Mexico to northern Argentina, except for the ocelot, which reaches southern Texas in the United States, and the little spotted cat, which extends only to Costa Rica.[18,32] In South America, tropical species currently range from the northern parts of the Continent in the Amazon and Orinoco basins, to the west side of the Andes in Ecuador (except for the little spotted cat), south to Salta Province and the Misiones area of northern Argentina, and the northern part of Rio Grande do Sul State in southern Brazil. Jaguarundi's distribution extends to central Argentina.[31,33] The area of jaguar occurrence has been considerably reduced in the eastern and southern portions of Brazil and on its southern limit.[25,46] Pumas' range from western North America to the Strait of Magellan in the southernmost part of the continent.[12]

Of the remaining four species, two have very limited distribution, the kodkod of central and southern Chile and adjoining areas of Argentina (Chubut and Santa Cruz to Rio Negro and Neuquen), and the Andean cat of the high Andes of southern Peru, central western Bolivia, northwestern Argentina, and eastern Chile (from Tarapacá to Santiago). The other two are widespread, mostly temperate species. The pampas cat ranges from the Strait of Magellan in the southernmost part of the continent to southern and central Brazil, Paraguay, and the mountain areas of Ecuador, northern Peru, and Bolivia, whereas Geoffroy's cat ranges east of the Andes in Bolivia, Paraguay, and southern Brazil to southernmost South America.[30,31]

Habitat

In South America, felids occur in every ecoregion found in the continent, except for the Peruvian-Chilean desert area. Habitats include tropical and subtropical rainforest, tropical deciduous and semideciduous forest, tropical thorny forest and shrubland, semiarid thorny scrub, savanna, wet/swampy savanna, premontane forest, montane forest, temperate coniferous forest, Monte, Patagonian steppes and scrub, Puna/Paramo, and the open grasslands of the Pampas. The puma and pampas cat are the species found in the greatest variety of these habitats, and Andean cat and kodkod in the fewest of them. Jaguar, ocelot, margay, little spotted cat, and jaguarundi are associated mostly with tropical habitats.[31]

Ecology

Ecological aspects of South American felids are poorly known, in comparison to cats elsewhere. Only jaguar, ocelot, and puma have been more thoroughly studied.[6,10,11,15,16,44] Neotropical felids are typically solitary, with a land tenure system in which home range of males will encompass those of 2–3 females (i.e., a polygynous mating system). All species hunt by stealth, killing prey by suffocation (large prey) or with a bite to the nape. Activity pattern is variable. It seems to be mostly nocturno-crepuscular, except for the jaguarundi, which is diurnal. However, varying amounts of daytime activity have been suggested for many species.[6,9,10,19,33,34,38]

Home ranges of males typically are nonoverlapping, whereas those of females go from some overlap to exclusive areas. Species home ranges vary according to their size (interspecifically), sex and habitat characteristics, especially the prey base (intraspecifically). For the larger cats ranges go from 9–10 km^2 to 259 km^2 for jaguars,[6,41] and for Neotropical pumas they are 56–155 km^2, as opposed to 109–1826 km^2 for this species in North America.[1,8] The medium-sized ocelot shows ranges of 0.76–121.1 km^2.[6,9] Meanwhile, the limited data set of margay, little spotted cat, and jaguarundi home ranges indicates about 10–15.9 km^2, 0.9–17.4 km^2, and 6.8–100 km^2, respectively.[33,34,42] For the temperate Geoffroy's cat and kodkod, ranges were 1.8–12.4 km^2 and 0.31–3.1 km^2, respectively.[19,20,45] No data is yet available for the remaining species.

Food habits vary considerably both inter- and intraspecifically. The mean weight of vertebrate prey (MWVP) taken (per study site) was positively correlated with felid body size. There are varying degrees of food-niche overlap among species. Nevertheless, there are always differences that would segregate them.[31, 35, 38] Jaguar staple prey consists of medium and large mammals such as peccaries, deer, large rodents, and armadillos, as well as chelonians. The species' average MWVP is, 32.02 kg (2.4–87.7 kg). Puma, in the Neotropics, also shows a varied diet, with a very similar prey base, but that is, on average, centered on medium-sized prey. Puma's average MWVP is 13.85 kg (1.5–52.3 kg).[35] The smaller species tend to prey mostly upon small rodents, but also include birds and reptiles to varying degrees. Their average biomass consumed in the wild is 700 g for the ocelot, 182.5 g for margay, 51 g for little spotted cat, 429.3 for jaguarundi, 64.3 for pampas cat, and 1.59 kg for Geoffroy's cat—due to the predominance of hares in the study site in southernmost Chile.[20,31,38]

Exploitation

The skin of cats has been used as ornaments in the American continent since Pre-Columbian times. However, at that time they were used primarily as special ceremonial attire, not as fur coats. Unfortunately, by the end of the 19th century a.d., cat skins began to be used in the European garment industry. After severe population declines of some African and Asiatic cats in the late 1950s, the fur trade shifted to South American species, especially the jaguar and ocelot.[26,27] The peak in the commercial trade of these cats in the late 1960s reached values of US$30 million/year for about a quarter of a million pelts. Commercialization in this era was centered in the Amazon basin, but was not restricted to it, and also included other species. In the 1970s, as a consequence of population decline of the larger species, the trade shifted to the smaller Neotropical cats. Between 1976 and 1985 the number of the smaller species' pelts reported to Convention on International Trade in Endangered Species of Fauna and Flora (CITES) totaled 1,091,056.[2] In this time period, Neotropical felids represented almost 50% of the world trade in cat skins.[27] In 1990, exports dropped to 1544 pelts. Most pelts were exported to western Europe, especially to Germany, France, and Italy.[2,3,30] At present, all spotted species are placed on Appendix I of CITES, which basically forbids their commercial use. Nevertheless, this does not fully preclude the occurrence of continued hunting and some incidence of illegal trade.[30,31]

Although the fur trade has dwindled, there is still commercial trade for the pet market of the smaller species. Statistics on this are not available, but at least in northern Brazil, ocelot, margay, and little spotted cat are occasionally found for sale. It seems that most animals in trade are caught and sold locally by residents, rather than by major dealers. The price range of these cats goes from US$40 to US$500. The lower price end is usually from owners and the upper end from dealers (T. G. Oliveira, personal observation).

Life history traits of the smaller Neotropical felids, such as longer gestation period, smaller litter size, and slower maturation, compared with equivalent species elsewhere in the world, have been suggestive of their low harvest potential. Additionally, the absence of data on population dynamics and harvest models and inefficient control and enforcement would further forbid the harvesting of these animals.[31]

Fur aside, South American cats, especially jaguar and puma, have the potential of being used economically for the growing industry of ecotourism, in the same way as lions and tigers in the Old World. It has been suggested that dependably baited jaguars could yield annual revenues of US$500,000 each in some National Parks and wilderness areas in South America. This would be the same amount generated by each male lion in East Africa.[17]

Conservation

Depending on the use of local, regional, or broader geographic scales, as well as different threat categorization schemes, Neotropical felid species and/or populations are considered to be vulnerable, endangered, or critically endangered.[30,31,32] Although the fur trade was, in the past, the major threat, currently the main conservation problems that these species face are derived from loss, alteration, and fragmentation of natural habitats, as well as direct persecution by humans due to conflicts (and often unjustified prejudice) over livestock depredation.[30] To counter these threats, it is important to integrate knowledge and approaches from diverse fields, devising conservation strategies that incorporate insights from disciplines such as ecology, demography, genetics, physiology, and behavior, and apply them in coordinated management efforts in the field and in captivity, as well as in associated environmental education programs.

Knowledge derived from basic sciences is crucial for both field- and captive-based conservation strategies, providing information on relevant topics such as population structure, history and dynamics, habitat requirements, genetic diversity, social interactions, physiological and behavioral characteristics, and dietary needs. Unfortunately, as described above, general aspects of the biology, ecology, and behavior of most Neotropical cats are currently known at a very basic level, at best. It is important to identify the geographic population units (subspecies) that should be managed separately,[13, 23] as well as to recognize the need for maintaining viable populations of these felids in the wild and in captivity and take action to do so.[14] Importantly, it has been shown that the size of most conservation units in the Neotropics, if isolated, would not be large enough for maintaining long-term viable populations of the larger species.[31] Applied field efforts that currently aim to implement conservation goals for felids include the prioritization, establishment, and enforcement of protected natural areas that are large enough to sustain viable and ecologically representative populations, as well as the management of conflicts between humans and cats over livestock depredation.[7,25,30,36] Applied captive-based conservation programs for South American cats include the attempt to establish, in a coordinated network of zoological institutions, viable populations of each of the geographical units (subspecies) of some of these species, which can be of use as potential sources for future reintroduction or genetic enhancement projects. Initial efforts in this direction have already produced very positive results.[5,28] Captive populations can also be extremely valuable for the development of basic research in diverse fields that are very relevant for conservation and for use in environmental education programs.

REFERENCES

1. Anderson, A.E. 1983. A Critical Review of Literature on Puma (*Felis concolor*). Special Report Number 54. Denver, Colorado Division of Wildlife, Research Section.
2. Broad, S. 1987. International trade in skins of Latin American spotted cats. Traffic Bulletin 9:56–63.
3. Broad, S. 1988. Species accounts. In S. Broad, R. Luxmore, and M. Jenkins, eds., Significant Trade in Wildlife: A Review of Selected Species in CITES Appendix II. Cambridge, IUCN/CITES, pp. 80–139.
4. Collier, G.E.; and O'Brien, S.J. 1985. A molecular phylogeny of the Felidae: Immunological distance. Evolution 39:473–487.
5. Comitê do Plano de Manejo da Jaguatirica. 1993. Plano de Manejo da Jaguatirica [Ocelot management plan]. Pedreira, Associação Mata Ciliar.
6. Crawshaw, P.G. 1995. Comparative ecology of ocelot (*Felis pardalis*) and jaguar (*Panthera onca*) in a protected subtropical forest in Brazil and Argentina. Doctoral thesis, University of Florida, Gainesville.
7. Crawshaw, P.G. in press. Jaguar conservation: The Pantanal and Iguaçu National Park in Brazil. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.

8. Crawshaw, P.G.; and Quigley, H.B. 1984. A Ecologia do Jaguar ou Onça-pintada no Pantanal [The ecology of the jaguar in the Pantanal]. Final report. Brasília, Instituto Brasileiro de Desenvolvimento Florestal.
9. Crawshaw, P.G.; and Quigley, H.B. 1989. Notes on ocelot movement and activity in the Pantanal region, Brazil. Biotropica 21:377–379.
10. Crawshaw, P.G.; and Quigley, H.B. 1991. Jaguar spacing, activity and habitat use in a seasonally flooded environment in Brazil. Journal of Zoology 223:357–370.
11. Crawshaw, P.G.; and Quigley, H.B. in press. Jaguar and puma feeding habits in the Pantanal of Mato Grosso, Brazil, with implications for their management and conservation. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.
12. Currier, M.J.P. 1983. *Felis concolor.* Mammalian Species 200:1–7.
13. Eizirik, E.; Bonatto, S.L.; Johnson, W.E.; Crawshaw, P.G.; Vie, C.; Brousset, D.; O'Brien, S.J.; and Salzano, F.M. 1998. Phylogeographic patterns and mitochondrial DNA control region evolution in two Neotropical cats (Mammalia, Felidae). Journal of Molecular Evolution 47:613–624.
14. Eizirik, E.; Indrusiak, C.B.; and Johnson, W.E. in press. Jaguar population viability analysis: Evaluation of parameters and case-studies in three remnant populations of southern South America. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.
15. Emmons, L.H. 1987. Comparative feeding ecology of felids in a Neotropical rainforest. Behavorial Ecology Sociobiology 20:271–283.
16. Emmons, L.H. 1988. A field study of ocelots (*Felis pardalis*) in Peru. Revue D'Ecologie (Terre Vie) 43:133–157.
17. Groom, M.J.; Podolsky, R.D.; and Munn, C.A. 1991. Tourism as a sustained use of wildlife: A case study of Madre de Dios, southeastern Peru. In J.G Robison and K.H. Redford, eds., Neotropical Wildlife Use and Conservation. Chicago, University of Chicago Press, pp. 393–412.
18. Hall, E.R. 1981. The Mammals of North America, Vol. 2. New York, John Wiley & Sons, pp. 393–412.
19. Iriarte, A.; and Sanderson, J. 1999. Home-range and activity patterns of kodkod *Oncifelis guigna* on Isla Grande de Chiloé, Chile. Cat News 30:27.
20. Johnson, W.E.; and Franklin, W.L. 1991. Feeding and spatial ecology of *Felis geoffroyi* in southern Patagonia. Journal of Mammalogy 72:815–820.
21. Johnson, W.E.; and O'Brien, S.J. 1997. Phylogenetic reconstruction of the Felidae using 16S rRNA and NADH-5 mitochondrial genes. Journal of Molecular Evolution 44:S98–S116.
22. Johnson, W.E.; Culver, M.; Iriarte, J.A.; Eizirik, E.; Seymour, K.; and O'Brien, S.J. 1998. Tracking the elusive Andean Mountain Cat (*Oreailurus jacobita*) from mitochondrial DNA. Journal of Heredity 89:227–232.
23. Johnson, W.E., Pecon-Slattery, J.; Eizirik, E.; Kim, J.; Menotti Raymond, M.; Bonacic, C.; Cambre, R.; Crawshaw, P.; Nunes, A.; Seuanez, H.; Moreira, M.A.; Seymour, K.L.; Simon, F.; Swanson, W.; and O'Brien, S.J. 2000. Disparate phylogeographic patterns of molecular genetic variation in four closely related South American small cat species. Molecular Ecology (in press).
24. Martin, L.D. 1989. Fossil history of terrestrial Carnivora. In J.L. Gittleman, ed., Carnivore Behavior, Ecology and Evolution, Vol. 1. New York, Cornell University Press, pp. 536–568.
25. Medellín, R.A.; Chetkiewicz, C.; Rabinowitz, A.; Redford, K.H.; Robinson, J.G.; Sanderson, E.; and Taber, A. Eds. in press. Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/ Wildlife Conservation Society.
26. Melquist, W.E. 1984. Status survey of otters (Lutrinae) and spotted cats (Felidae) in Latin America. Report to IUCN, Gland, Switzerland, IUCN.
27. McMahan, L.R. 1986. The international cat trade. In S.D. Miller and D.D. Everet, eds., Cats of the World: Biology, Conservation, and Management. Washington, D.C., National Wildlife Federation, pp. 461–488.
28. Morato, R.G.; and Barnabe, R.C. in press. Potential of reproductive techniques for jaguar conservation. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.
29. Nowak, R.M. 1991. Walker's Mammals of the World, Vol. 2, 5th Ed. Baltimore, Johns Hopkins University Press, pp. 643–1629.
30. Nowell, K.; and Jackson, P. 1996. Wild cats: Status Survey and Conservation Action Plan. Gland, Switzerland, IUCN/SSC Cat Specialist Group, p. 382.
31. de Oliveira, T.G. 1994. Neotropical cats: Ecology and Conservation. São Luís, EDUFMA, p. 220.
32. de Oliveira, T.G. 1997. Status dos mamíferos no Estado do Maranhão: Uma proposta de classificação das espécies ameaçadas de extinção [Status of mammals in the State of Maranhão: A proposal of classification for species threatened with extinction]. Pesquisa em Foco 5:65–82.
33. de Oliveira, T.G. 1998. *Herpailurus yaguaroundi.* Mammalian Species 578:1–6.

34. de Oliveira, T.G. 1998b. *Leopardus wiedii*. Mammalian Species 579:1–6.
35. de Oliveira, T.G. in press. Comparative feeding ecology of jaguar (*Panthera onca*) and Puma (*Puma concolor*) in the Neotropics. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.
36. de Oliveira, T.G. in press. Conservation assessment of jaguars (*Panthera onca*) in eastern Amazonia and northeastern Brazil. In R.A. Medellín, C. Chetkiewicz, A. Rabinowitz, K.H. Redford, J.G. Robinson, E. Sanderson, and A. Taber, eds., Jaguars in the New Millennium: A Status Assessment, Priority Detection, and Recommendations for the Conservation of Jaguars in the Americas. Mexico City, Universidad Nacional Autonoma de Mexico/Wildlife Conservation Society.
37. de Oliveira, T.G.; and Cassaro, K. 1997. Guia de Identificação dos Felinos Brasileiros [Identification guide of Brazilian Felids]. São Paulo, Sociedade de Zoolgicos do Brasil/Fundação Parque Zoolgico de São Paulo, p. 60.
38. de Oliveira, T.G.; and de Paula, R.C. 2000. Comparative analysis and ecological implications of the biomass eaten by the little spotted cat (*Leopardus tigrinus*), margay (*Leopardus wiedii*), jaguarundi (*Herpailurus yagouaroundi*), and Pampas cat (*Lynchailurus colocolo*) in the wild and in captivity. Animal Conservation (in review)
39. Pecon-Slattery, J.; Johnson, W.E.; Goldman, D.; and O'Brien, S.J. 1994. Phylogenetic reconstruction of South American felids by protein electrophoresis. Journal of Molecular Evolution 39:296–305.
40. Pecon-Slattery, J.; and O'Brien, S.J. 1998. Patterns of X and Y chromosome DNA sequence divergence during the Felidae radiation. Genetics 148:1245–1255.
41. Rabinowitz, A.R.; and Nottingham, B.G. 1986. Ecology and behaviour of the jaguar (*Panthera onca*) in Belize, Central America. Journal of Zoology 210:149–159.
42. Rodrigues, F.H.G.; and Marinho-Filho, J. 1999. Translocation of oncilla and jaguarundi in central Brazil. Cat News 30:28.
43. Salles, L.O. 1992. Felid phylogenetics: Extant taxa and skull morphology (Felidae, Aeluroidea). American Museum Novitates 3047:1–67.
44. Schaller, G.B.; and Crawshaw, P.G. 1980. Movement patterns of jaguar. Biotropica 12:161–168.
45. Sunquist, M.E.; and Sanderson, J. 1998. Ecology and behaviour of the kodkod in a highly-fragmented, human-dominated landscape. Cat News 28:17–18.
46. Swank, W.G.; and Teer, J.G. 1989. Status of the jaguar—1987. Oryx 23:14–21.
47. Turner, A.; and Antón, M. 1997. The big cats and their fossil relatives. New York, Columbia University Press, p. 234.
48. Wozencraft, W.C. 1993. Order Carnivora. In D.E. Wilson and D. M. Reeder, eds., Mammal Species of the World: A Taxonomic and Geographic Reference, 2nd Ed. Washington, D.C., Smithsonian Institution Press, pp. 286–346.
49. Eizirik, E.; Kim, J.H.; Menoti-Raymond, M.; Crawshaw, P.G., Jr.; O'Brien, S.J.; Johnson, W.E. In press. Phylogeography, population history and conservation genetics of jaguars (*Panthera onca,* Mammalia, Felidae). Molecular Ecology.

MEDICINE

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QUARANTINE

Animals newly introduced into zoological gardens must be quarantined as a protection for other animals in the collection. A careful analysis of the origin and history of the animal should be made. New cats should quarantined for at least 30 days. Samples of the first defecation should be collected in order to determine feeding habits, if from the wild, and to perform fecal analyses. Feces for fecal examination should be preserved with mercury, iodine, and formaldehyde, iodine and formaldehyde, or 2% potassium dichromate. For feeding analysis, feces should be frozen or stove dried.

During quarantine, a complete clinical examination should be performed, and the cat immunized. Most of these examinations require anesthesia, and this is an excellent time to obtain baseline laboratory data, measurements and evaluation of an anesthetic agent for future reference.

Quarantine Enclosures[2]

Quarantine enclosures should be at least 200 m away from other enclosures. Enclosures should be isolated, preferentially near the zoo entrance for ease in receiving incoming animals immediately on arrival and to avoid direct/indirect contact with any other zoo animal. The quarantine area should have a single entrance to keep enclosures totally isolated. As a precaution, the location that is chosen should be quiet and shaded most of the day.

Enclosures for small felines should be 2 m × 3 m (6 m^2) in area and should be 2 m high. Floors should be made of smooth concrete (for ease in cleaning) and may be partially covered with leaves, dried grass, or a wooden platform. All covering materials should be periodically removed and changed or sterilized, to minimize the risk of contamination. Corners of enclosures should be rounded with a slightly descending slope (3–5%) to facilitate water drainage. Enclosures should always be disinfected between occupants. Side and back walls should have a smooth, easy-to-clean covering. The front of the enclosure should have a wire screen, not directly welded to the walls. This screen should be welded to a metal frame, which can be easily removed when necessary. This procedure also prevents the screen from rusting too quickly.

Enclosures should have three doors (one for the animal in the holding cage and the other two for the animal keeper to enter the enclosure and the holding cage). The door to the holding cage should move laterally or in guillotine form. View ports on doors and walls allow a view of the animal by the keeper. The holding cage should be 1 m wide, 1 m long and 1.80 m high, so the animal keeper may enter easily. The door for the entrance of the animal should measure 0.6 m–0.7 m and be provided with artificial light. Although enclosures should be covered, they should receive morning sunlight.

Inside the enclosure there should be wooden platforms, logs, and branches that are easy to remove. Although the presence of a movable wooden platform in the holding cage improves the comfort of the animal, it does not substitute for a bed in the shelter inside the enclosure. A smooth and continuous wooden platform is recommended instead of one made with wood strips in order to avoid the accumulation of dirt and accidents with offspring, that may fall between the strips. A platform rotation routine (for cleaning one at a time) should be established. It is important that feeding pans are easily removed and cleaned daily. They should be removed and sterilized or discarded after the animal leaves quarantine.

FACILITIES FOR CAPTIVE BREEDING[2]

In a recent study[1] in 22 zoos in São Paulo State, Brazil (representing 99% of the zoos in that state), 38 enclosures housing 113 felines were assessed. Enclosures were evaluated as to their structure, safety, and environmental enrichment. Only 28% of the enclosures were considered to be good to excellent, and only 4% had adequate enrichment.

Management protocol sets[2] a minimum enclosure area of 25 m^2 for ocelots and 15 m^2 for other small cats. Holding cages should be 1.5 m^2 and the solarium 5 m^2. Enclosure height should be 2.5 m. Since margay are arboreal, this species may be kept in higher enclosures. Feed should be offered in individual, physically separated pans, in order to avoid competition, which may produce severe trauma. All of these animals are solitary in the wild. Females with offspring should always be kept away from the males.

EXHIBITION AND BREEDING ENCLOSURES[2]

Wild cat enclosures inside the zoo should be sited and designed for the welfare of the animals and to minimize stress. The location should be quiet and have shaded areas for most of the day, but provide sunshine for plant growth. The ground should be irregular, a mixture of sand, earth, grass, and plants growing here and there to provide privacy for the animals.

Enclosures should be at least 2 m away from the public. Plants may be placed around it to create a barrier. The smallest side of the enclosure should face the public. For ocelots, screens should be made of 12-inch diameter wire in a network of 2.0 cm; for other small cats, a 12-inch wire and a network of 1.5 cm is recommended. The screen should be of a neutral color (e.g., dark gray or dull black) to avoid reflection of light. Tempered glass may also be used as a barrier, but it should not be placed in direct contact with the animal, for safety reasons. Animals may be kept away from the glass with a crushed rock or a water course barrier. This will keep the glass clean and avoid the stressful to-and-fro pacing behavior. Less than 30% of the enclosure should have a solid cover.

Shelters should be placed in the highest point of the enclosure. Logs and branches should be placed in the enclosure for claw grooming and exercise. The drinking water source should be renewable and easy to clean. It should be located above the floor in order to avoid deposit of urine and feces. There should be a security corridor in wild cat enclosures in order to prevent escapes; a maternity den with a solarium and holding cage should also be provided. The solarium should have an area of at least 5 m^2, the ground should be earth and grass, and it should be covered with a screen.

RESTRAINT

Physical restraint of wild cats may be performed using leather gloves, nets, and squeeze cages. For ocelots, use nets of 60 cm (2 ft) diameter, 1.0 m (3 ft) deep, made of 12-inch diameter wire in 4.0 cm (1.5 in.) polypropylene squares. For other small wild cats, nets should be 50 cm (20 in.) in diameter and 80 cm (31 in.) deep, made of 10-inch diameter wire in 3.0 cm polypropylene squares. In both nets, the hoop should be made of covered metal, because animals may bite it during restraint and may otherwise sustain teeth fractures. If chemical restraint is used, the Management Plan for Brazilian Wild Felines[2] recommends ketamine hydrochloride associated with xylazine hydrochloride in the following doses, based on 500 anesthesia procedures from 1994 to 1999.

Felines	Drug	Dose (mg/kg)
Ocelots	Ketamine	12
	Xylazine	1
Other small cats	Ketamine	10
	Xylazine	2
Jaguar/Puma	Ketamine	10
	Xylazine	1

Tiletamine and zolazepam (Zoletil, Telazol) at a 4–7 mg/kg dose has also been used successfully in these animals. However, recovery times are longer than with ketamine/xylazine.

PREVENTIVE MEDICINE AND IMPORTANT DISEASES

PREVENTIVE MEDICINE[2]

Parasite Control

For control of endoparasites, fecal examination should be performed every 4 months. If treatment is necessary, a new fecal examination should be made after 1-2 weeks in order to evaluate drug efficacy. Ectoparasites (mites, ticks, lice, fleas, and others) should be controlled. After treatment, feces should be carefully removed, material used in perches and shelters should be discarded, and, only then, the enclosure should be cleaned. Formaldehyde, sodium hypochlorite, or quaternary ammonia solutions may be used, or physical disinfection may be achieved with a flame. It is important not to use any cleaning products containing phenolic radicals.

DISEASE

In a study of 113 animals of four species of Brazilian wild cats in 22 zoos in São Paulo, Brazil,[1] as many as 50% of the animals had disorders associated with management. Dental disorders included gingivitis and periodontitis caused by tartar accumulation. Tooth fractures were common, as a result of trauma, which led to pulpitis and necrotic tissue accumulation on the tooth canal.

Claw disorders were related to excessive wearing or failure to wear. In the latter case, wounds on the pads may occur. Plantar and palmar pad erosion was another foot disorder, resulting from inadequate sanitary conditions and excessively rough enclosure substrates.

Alopecia and pilus rarefaction were observed, in association with ectoparasites, excessive licking, and nutritional deficiencies. Thirty percent of the little spotted cats and jaguarundis were parasitized with *Ctenocephalides* spp.; in some of the animals severe anemia and death were the consequence of the infestation. A *Sarcoptes scabei* infestation in a little spotted cat caused changes in the ear.[6] Flea control was performed using topical monthly applications of fiproni, which was repeated three times, followed by physical disinfection of the enclosure using a flame.

Twenty-one percent of the little spotted cats were below the ideal weight, and 29.2% of the ocelots were obese.

Metabolic Disease

The primary metabolic disease of felines is metabolic bone disease, discussed elsewhere.

Infectious Diseases

According to the Brazilian health department,[4] there were 1205 cases of human rabies from 1980 to 1986. In 105 cases (9.7%), the disease was transmitted by wild animals; in only four cases were wild cats involved (*Leopardus* spp.). In São Paulo, Brazil, from January 1994 to December 1997, a study[3] performed to determine the prevalence of rabies antibodies in 547 wild animals verified that this prevalence was 8.8%. Among the six wild cats studied, only one little spotted cat had antibodies to the rabies virus. Two ocelots and one margay were seronegative.

Other infectious diseases of small felids include feline panleukopenia, viral feline rhinotracheitis, and salmonellosis. These diseases and their management are the same as for domestic cats.

Parasitic Diseases

The following parasites have been identified in wild felids and in zoos in South America: Spiruroidea type *Spirocerca;* Spiruroidea type *Physaloptera; Spirometra* sp.; *Toxocara cati; Toxascaris leonina; Capilaria* spp. and *Trichuris* spp*; Aelurostrongylus abstrusus Strongyloides* sp. larvae; *Platynosomum fastosum; Giardia* cysts; *Cystoisospora felis; Eimeria and Toxoplasma-Hammondia* oocysts, and *Sarcocystis* sporocysts. The most common clinical signs of parasitism are diarrhea, vomiting, anemia, and a rough fur coat. Anthelmintic drugs, such as mebendazole, ivermectin, pirantel, sulfas, and others, are the recommended treatment.

Ectoparasites include fleas, mites, and ticks.

HEMATOLOGY

Hematological parameters of Neotropical wild cats in captivity[2] are presented in Tables 27.1 and 27.2.

TABLE 27.1. Hematological data for some Neotropical wild felids in captivity

Wild Felids	No.	RBC (× 10⁶) X	RBC (× 10⁶) DP	Hb (mg/dL) X	Hb (mg/dL) DP	PCV (%) X	PCV (%) DP	MCV (fl) X	MCV (fl) DP	MCH (pg) X	MCH (pg) DP	MCHC (gd) X	MCHC (gd) DP	Plasma Protein X	Plasma Protein DP
Jaguar *(P. onca)*	32	6.99	1.02	10.73	1.56	32.95	4.90	47.20	3.30	15.40	1.12	32.61	1.33	7.42	0.78[a]
Puma *(P. concolor)*	20	7.41	0.77	11.77	1.36	34.75	4.15	46.90	3.18	15.90	0.82	33.95	1.78	7.39	0.41[b]
Ocelot *(L. pardalis)*	74	6.68	1.55	12.21	1.90	36.39	5.10	56.90	13.70	19.10	4.94	33.23	28.73	7.45	0.61[c]
Margay *(L. wiedii)*	16	7.18	1.26	12.23	2.31	37.10	6.40	51.70	3.69	17.00	0.85	33.93	2.46	6.91	0.51[d]
Jaguarundi *(H. yaguarundi)*	23	6.49	1.70	12.29	2.39	36.17	6.85	56.00	20.00	19.6	5.08	34.10	2.96	7.03	0.57[e]
Little spotted cat *(L. tigrinus)*	27	6.69	1.31	11.87	1.88	35.04	5.20	54.40	14.80	18.40	5.13	33.79	2.22	6.87	0.31[f]

Source: See reference 2.
[a]*n* = 21; [b]*n* = 16; [c]*n* = 44; [d]*n* = 7; [e]*n* = 12; [f]*n* = 16. Hb, hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell count; X, mean; DP, standard deviation.

TABLE 27.2. Hematological data for some Neotropical wild felids in captivity

Wild Felids	No.	WBC (× 10³) X	WBC (× 10³) DP	Neutrophils X	Neutrophils DP	Lymphocytes X	Lymphocytes DP	Monophils X	Monophils DP	Eosinophils X	Eosinophils DP	Basophils X	Basophils DP	Platelets X	Platelets DP
Jaguar (*P. onca*)	32	10.86	2.54	77.27	13.10	15.59	12.31	1.36	1.40	4.71	2.89	0	0	353,333.3	154,410.7[a]
Puma (*P. concolor*)	20	8.35	2.07	72.35	17.08	22.75	15.89	1.00	1.02	3.55	4.63	0	0	—	—
Ocelot (*L. pardalis*)	74	10.11	2.79	69.01	9.22	21.47	8.4	1.61	1.46	6.72	4.91	0	0	—	—
Margay (*L. wiedii*)	16	7.81	1.85	61.22	14.48	27.77	12.00	1.22	0.97	9.66	5.87	0	0	—	—
Jaguarundi (*H. yaguarundi*)	23	7.85	2.67	65.43	16.78	26.00	15.96	1.82	1.23	4.01	4.15	0	0	—	—
Little spotted cat (*L. tigrinus*)	27	8.17	2.72	67.74	15.80	27.25	15.89	1.62	1.92	2.18	2.51	0	0	343,625.0	64,409.27[b]

Source: See reference 2.
[a]*n* = 7; [b]*n* = 8. WBC, white blood cell count; X, mean; DP, standard deviation.

NUTRITION AND KITTEN CARE

Feeding Adults in Captivity

In the wild, Neotropical wild cats feed on small mammals and birds. In South American zoos, small cat diets include live or recently slaughtered whole animals, chicken necks, fish, domestic cat commercial diets, and beef. Live or recently slaughtered whole animals should be offered at least three times a week. The daily quantity should be 4–6% of the body weight. Beef offered to the animals should be frozen for at least 4 days at -20°C in order to destroy *T. gondii* cysts. Fasting one day a week is not recommended for small cats.

Reproductive Care

It is best for kittens to be cared for by the mother. There must be a maternity den (holding cage with a solarium) in the enclosure, where shelter boxes are available for the female. Material for the construction of the nest, such as grass or dry leaves, may be supplied. If there is a pool with running water, measures must be taken to prevent drowning accidents of the kittens. It is important to isolate the female when the first

TABLE 27.3. Milk replacers for young felines

Formula 1[a]		Formula 2	
Item	**Quantity**	**Item**	**Quantity**
Water	250 mL	Water	50 mL
Powdered cow milk	80%	Prepared powdered cow milk	200 mL
Soy milk	20%	Egg yolk	1
Egg yolk	1	Honey or dextrosol	1 teaspoon
Honey or dextrosol	1 teaspoon	Terragran junior	10 drops
Kalyamon B_{12} or similar product	1 teaspoon	Kalyamon B_{12}	10 drops
Vitamin complex	5 drops	Mineral oil	1 teaspoon
Mineral oil	1 teaspoon	Salt	a pinch

Source: See reference 2.
[a]Be cautious about changing the milk rapidly.

signs of pregnancy are observed, or the pair must be observed during the pregnancy; and the male removed near parturition.

Hand-Rearing Kittens

MILK REPLACERS, BASIC FORMULAS FOR KITTENS If possible, KMR, Esbilac (Pet/Vet Products Borden Inc., Nortfolk, Virginia, USA), or Mothers Help milk should be offered. They are normally found in pet shops. If it is not possible to offer one of these commercial milk substitutes, one of the formulas in Table 27.3 should be prepared and offered to the young animals.

During the first 12 hours of life, only a glucose solution should be offered. After this period, a mixture of 50% glucose solution and 50% milk may be offered (Table 27.3). From the third day on, only milk should be offered. The interval between feedings should be approximately 2 hours for the first 10 days and 3 hours after that. Feeding times should be distributed evenly over a period of 16 hours.[10] The quantity consumed may vary from 20 to 40 mL/kg of body weight. Analyses of offspring reared under this regimen suggest that ocelots and little spotted cats double their weight 20 by days after birth.[5]

The enclosure for orphaned kittens should be ventilated and the temperature kept at 29.4–32.0°C in the first 3 weeks, and at 21.2–23.9°C after that.[10] During milk feeding, offspring should be in a standing position. After each feeding, the abdomen and anus should be massaged with a warm damp cloth to stimulate defecation. When the kittens are approximately 1 month old, teeth and motor development should be observed, to determine readiness for gradually change to a solid diet.[5]

REFERENCES

1. Adania, C.H.; Diniz, L.S.M; Gomes, M.S.; Filoni, C.; and Silva, J.C.R. 1998. Avaliação das condições veterinárias e de manejo dos pequenos felinos neotropicais em cativeiro no Estado de São Paulo. Revista de Educucaçse Continuada-Regional de Mediciana-São Paulo 1(1):44–53.
2. Associação Mata Ciliar. Coordenadoria de Fauna. Plano de Manejo para Pequenos Felinos Brasileiros. 1998. Protocolo(Manejo Integrado para Pequenos Felinos Brasileiros 1997/2000. Jundiaí, Associação Mata Ciliar, p. 29.
3. Almeida, M.F. 1998. Prevalência de anticorpos antirábicos neutralizantes em animais silvestres do município de São Paulo. Masters thesis, Faculdade de Medicina da Universidade de São Paulo.
4. Ministério da Saúde. 1995.Programa Nacional de Profilaxia da Raiva. Informes Técnicos. Brasilia, Fundação Nacional de Saúde do Brasil.
5. Chieregatto, C.S.; and Gomes, M.S. Pers. comm.
6. Diniz, L.S.M; Costa, E.O.; and Benites, N.R. 1997. Processos dermatolgicos dos animais silvestres. Clínica Veterinária São Paulo 2(8):6–9.
7. Dubey, J.P.; and Beattie, C.P. 1988. Toxoplasmosis of animals and man. Boca Raton, Florida, CRC Press, p. 220.
8. Ferraroni, J.J.; and Marzochi, M.C.A. 1980. Prevalência da infecção pelo *Toxoplasma gondii* em animais domésticos, silvestres e grupamentos humanos da Amazônia. Memorias do Instituto Oswaldo Cruz 75(1(2):99–109.
9. Ferraroni, J.J.; Reed, S.G.; and Speer, C.A. 1980. Prevalence of *Toxoplasma* antibodies in humans and various animals in the Amazon. Proceedings of the Helminthological Society of Washington 47(1):148–150.
10. Fowler, M.E. 1986. Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders.
11. Frenkel, J.K. 1980. Protozoan diseases of zoo and captive mammals and birds. In R.J. Montali, ed., The Comparative Pathology of Zoo Animals. Washington D.C., Smithsonian Institution Press, pp. 329–342.
12. Jewell, M.L.; Frenkel, J.K.; Johnson, K.M.; Reed, V.; and Ruiz, A. 1972. Development of *Toxoplasma* oocysts in neotropical *Felidae*. American Journal of Tropical Medicine and Hygiene 21(5):512–517.

13. Miller, N.L.; Frenkel, J.K.; and Dubey, J.P. 1972. Oral infections with *Toxoplasma* cysts and oocysts in felines, other animals, and in birds. Journal of Parasitology 58(2):928–937.
14. Pizzi, H.L.; Rico, C.M.; and Pessat, O.A.N. 1978. Hallazgo del ciclo ontogenico selvatico del *Toxoplasma gondii* em felidos salvajes (*Oncifelis geoffroyi, Felis colocolo* y *Felis eira*) de la Provincia de Cordoba. Revista Militar Argentina 25(117):293–300.
15. Silva, J.C.R. 1997. Pesquisa de anticorpos anti-*Toxoplasma gondii* em felídeos silvestres de parques zoolgicos do Estado de São Paulo, mediante o emprego do teste de aglutinação do látex. São Paulo. Masters thesis, Universidade de São Paulo.
16. Sogorb, F.; Jamra, L.F.; and Guimarães, E.C. 1977. Toxoplasmose em animais de São Paulo, Brasil. Revista de Instituto de Medicina Tropical de São Paulo 19(3):191–194.

REPRODUCTION IN SMALL FEMALE FELIDS

Nei Moreira

INTRODUCTION

The 37 species of modern cats have evolved from approximately eight phylogenetic lineages within the past 10 to 15 million years. In relation to conservation, all South American small felids are included in CITES Appendix I or Appendix II. These species are in an endangered status in the wild mainly due to loss of habitat and hunting pressure.

Many of the small Neotropical felid species do not receive special attention at the zoos because they do not call great public attention; they have at most crepuscular or nocturnal activities and a reclusive nature, staying out of view or resting during daily visitation periods. Captive populations of small cats often comprise fewer than 50 animals that are widely dispersed geographically, making them difficult to study in scientifically meaningful numbers. Some animals that reproduce well in captivity are not bred, because there is not enough space in the zoos, and many times these individuals are kept in extra sectors.

In Brazil there is a nationally coordinated management plan for small felids that keeps and analyzes inventory information. It organizes and supports conservation programs with a realistic assessment of size, origin, health, and enclosure conditions of the captive population.

Historically, the small Neotropical felids have been known for their poor reproductive performance in captivity. The percentage of births in the captive population is still low and successful propagation unpredictable. Breeding records for the population of Brazilian zoo small felids show that there is a small percentage of births, associated with a high mortality rate in the first 30 days. Thus, reproductive performance is still unreliable and the circumstances surrounding successes and failures in captive breeding are poorly understood. Considering the high genetic value of the population of small felids in captivity in South America, with many individuals from the wild and/or with known origin and the increasing emphasis on the role of captive propagation as a conservation tool, successful breeding has become obligatory.

An improved understanding of the small Neotropical felids' reproductive physiology is needed to achieve more consistent reproductive performance in captivity, to increase the number of breeding individuals in the population, and to apply assisted reproduction (i.e., artificial insemination and in vitro fertilization) with more successful results. In cheetahs, many of the phenotypic effects attributed to inbreeding depression, such as infertility, reduced litter sizes, and increased susceptibility to disease, are limited to captive individuals and may be explained as physiological or behavioral artifacts of captivity.

Urine spraying is the predominant form of scent marking in free-ranging females, the frequency of which fluctuates during the year and seems to be related to changes in the reproductive cycle. Marking frequency increases dramatically during the breeding period, is low during pregnancy, and can be entirely absent when the female is rearing young. This strongly indicates a primary function in the advertisement of female reproductive condition, although an additional role in the maintenance of social spacing is suggested. In a female black-footed cat (*Felis nigripes*) these scent marks were not restricted to the borders of the animal's range, but corresponded to those areas used most intensely during the year.

GENERAL REPRODUCTIVE CHARACTERISTICS

General reproductive data are presented in Table 27.4. Historically, jaguarundis (*Herpailurus yaguarundi*) and ocelots (*L. pardalis*) have bred and reared young more consistently in captivity, when compared with another Neotropical small species, like *L. tigrinus* and *L. wiedii,* which are more difficult to breed in captivity. Pampas cat (*Oncifelis colocolo*) is rare in the world and Geoffroy's cat (*O. geoffroyi*) is rare in Brazil.

Puberty correlates with body weight, being sooner in females than males. For Geoffroy's cats, mean age of

TABLE 27.4. Reproductive data for small South American felids

Species (Status)	Scientific Name	Duration of Estrus (Days)	Estrous Cycle Length (Days)	Gestation (Days)	Number of Kittens per Litter (Mean ± SE)
Tigrina or Oncilla (CITES Appendix I; IUCN—Vulnerable)	*Leopardus tigrinus* (Schreber, 1775)	3–9[18]	16.4 ± 1.2[46]	73–78[24] 55–60[67] 62–74[36]	1.06[39] 1–2, one most common[24,36]
Ocelot (CITES Appendix I; IUCN—Vulnerable)	*Leopardus pardalis* (Linnaeus, 1758)	4.63 ± 0.63[39] 7–10[12]	25.11 ± 4.33[39] 42[12] 19.9 ± 1.8[46]	70–85[17,24,27,39] 79–85[43]	1.4[12] 1.67 ± 0.21[39] 1–2[13,27,43]
Margay (CITES Appendix I; IUCN—Vulnerable)	*Leopardus wiedii* (Schinz, 1821)	4–10[13,39,52]	32–36[17,39,52,53] 26.1 ± 3.5[46]	76–84[17,39,52]	1[10,14,39,54] 1–2[17,43,52]
Geoffroy's cat (CITES Appendix I)	*Oncifelis geoffroyi* (d'Orbigny and Gervais, 1844)	2.50 ± 0.50[39] 1–12[1,19,34]	20[39] 23–29[18]	62–78 (majority at 71–78 days)[1,24,39,60]	2.31 ± 0.13[39] 1.5 (1–3)[1,18]
Jaguarundi (CITES Appendix II; IUCN—Indeterminate)	*Herpailurus yaguarundi* (Lacépède, 1809)	3.17 ± 0.75[39]	53.63 ± 2.41[39]	70–75[30] 63–70[49] 86 (Cassaro, unpublished data)	1.83 ± 0.24[39] 1–4[24,27,30]
Pampas cat (CITES Appendix II)	*Oncifelis colocolo* (Monlina, 1782)			80–85[24]	1.31 ± 0.13[39] 1–3[4,14,56]
Kodkod (CITES Appendix II)	*Oncifelis guigna* (Molina, 1782)			72–78[73]	1–3[73]; 3–4[29]; 2–3[57]
Andean mountain cat (CITES Appendix I; IUCN—Rare)	*Oreailurus jacobita* (Cornalia, 1865)			63–70[59]	2–4[59]

Source: See references 12, 18, 24, 39, 51, 55, 67.
CITES, Convention on International Trade in Endangered Species of Wild Fauna and Flora; ICUN, International Union for the Conservation of Nature.

sexual maturity reported by International Species Information System (ISIS) was 50 months for males and 47 months for females, but both can be reduced, especially with a good nutrition. Breeding could continue until 16 years of age or older. The seasonal nature of the reproductive cycle in free-living domestic cats is dependent primarily on daylight length. Another important aspect when considering wild felids is food availability. A recent seasonality study with tigrina, ocelot, and margay females in captivity found that these species do not show a characteristic reproductive seasonality pattern, therefore breeding efforts can be conducted throughout the year.

The recent development of fecal steroid assays for assessing ovarian function in felids now makes it possible to generate a database, besides correlate with other factors such as captive environment, seasonality, nutrition, and behavior. Noninvasive fecal hormone monitoring avoids stress in serial blood collections and possible disturbances in hormone secretion and in the behavior due to restraint procedures.

A recent study has revealed that tigrinas, ocelots, and margays are polyestrous and the intervals and range between estradiol excretion peaks are presented in Table 27.5.

Tigrinas and ocelots, like most felids, appear to be primarily induced ovulators with ovulation occurring only after a copulatory stimulus. Estradiol levels fluctuate in nonmated females reflecting waves of follicular growth and regression, whereas progesterone concentrations remain at baseline in tigrinas and ocelots. Margay females can present high levels of fecal progestogen metabolites, reflecting spontaneous ovulation (Figure 27.1).

Estrous, courtship, and mating behaviors are reported to be rather uniform across felid species and similar to that of the domestic cat. Common behaviors associated with estrus include rolling, object rubbing, vocalizing, pacing, locomotor activity, grooming, urination, urine spraying, investigative activity (e.g., sniffing), and lordosis.

Estrus in most of these species has been identified as "silent", or females show just minor signs that are

TABLE 27.5. Intervals and range between estradiol excretion peaks for female tigrinas, ocelots, and margays

Species	Intervals between Estradiol Excretion Peaks (Days)[a]	Range between Estradiol Excretion Peaks (Days)
L. tigrinus (tigrina)	16.4 ± 1.2	11–27
L. pardalis (ocelot)	19.9 ± 1.8	7–51
L. wiedii (margay)	26.1 ± 3.5	11–55

[a]Mean ± SEM.

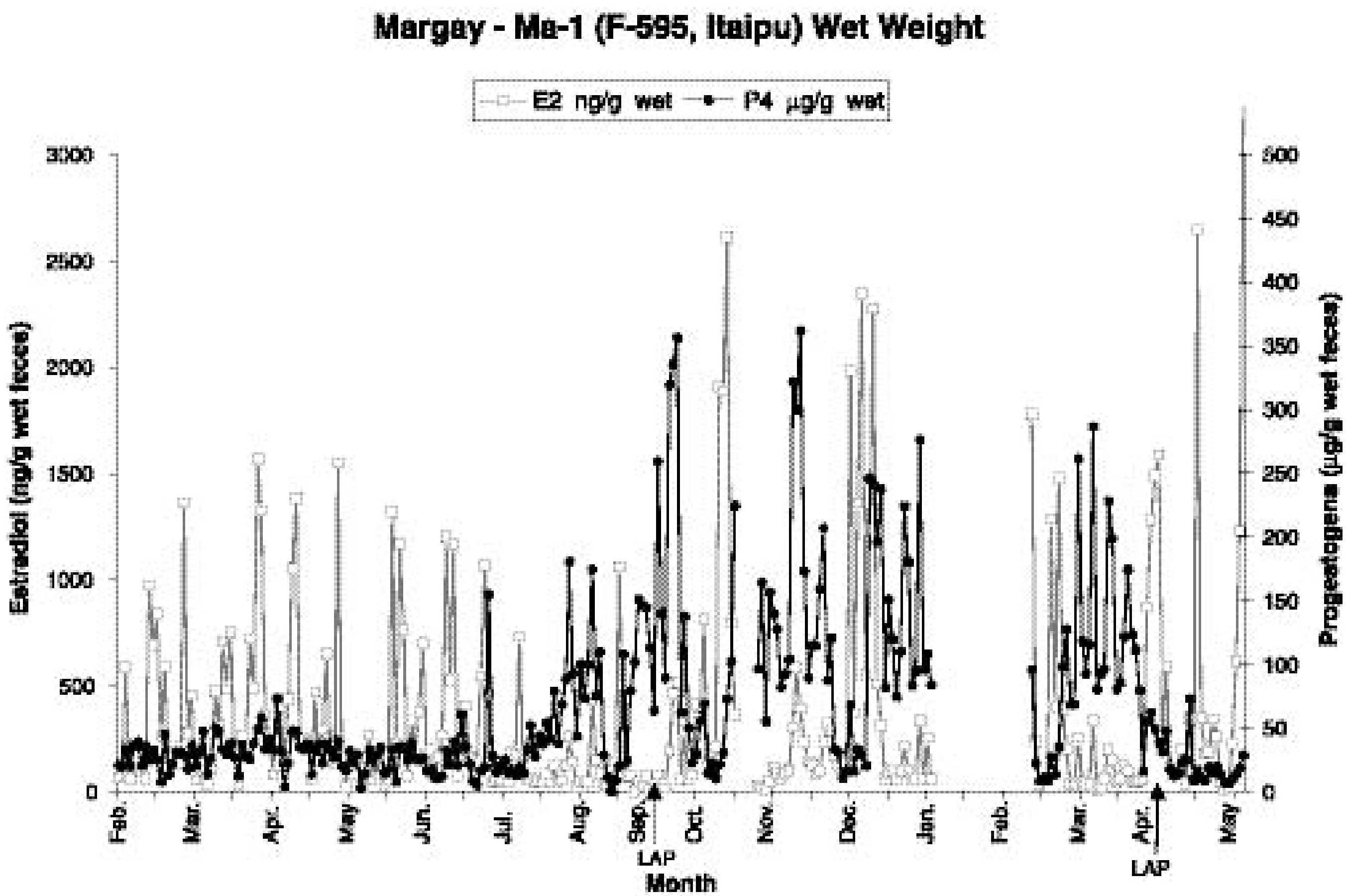

FIGURE 27.1. Longitudinal profile of fecal estradiol (open squares)and progestogen (closed squares) metabolites in a margay female housed alone. This female displayed ovarian activity throughout the year and spontaneous ovulation, especially from September to April. LAP, laparoscopic examination.

difficult to identify under practical conditions or routine procedures. Some species, with ocelots as an example, can show some estrous signs, like rubbing, vocalizing, rolling, urine spraying, and sniffing in a more perceptible way, which can be associated with an interest by males.

However, similar to cheetahs, the frequency and types of correlated behaviors vary across females, revealing no single behavior indicative of estrus, but rather a constellation of behaviors that some females show when estradiol concentrations are elevated. Although behavioral monitoring of estrus is possible in small wild felids, this may nevertheless be difficult (mainly in females housed alone) and time consuming due to individual variations and subtle changes in behavioral frequencies rather than changes in the types of behaviors displayed.

With estrus usually not observed, the appropriate timing of pair introductions is difficult and is associated with behavioral incompatibilities, which sometimes result in serious injuries or killing fights. Considering this, first-time breeding introductions must be carefully monitored. Another aspect that must be considered, when breeding these animals, is the possibility of the male or female killing the kittens in a stressful situation.

SUGGESTIONS FOR BREEDING MANAGEMENT (ENCLOSURES, ENRICHMENT, BREEDING PROTOCOL)

Similar to cheetahs, behavioral and management factors (extrinsic factors) are believed to be the main contributors to successful reproduction of these species in captivity. Environmental enrichment can increase reproductive success directly by providing the social and physical environments necessary for successful reproductive behavior and parental care and indirectly by providing the developmental environment required for the growth of behaviorally normal, and therefore reproductively viable, adults.

Many aspects must be considered when planning or adjusting a breeding facility for small felids. Since possible stress factors must be eliminated or avoided, calm caretaking routine must be provided and off-exhibit enclosures are preferred. Cats stressed by unpredictable manipulations had cortisol concentrations elevated and pituitary sensitivity to luteinizing hormone-releasing hormone (LHRH) reduced. Beyond this, physical and visual contact must be avoided between adjacent enclosures; this is important especially when considering pregnant and lactating females. In some zoos, these small species are installed near big cats, like jaguars, causing a chronic stressful condition, considering these big cats are potential predators in the wild. Carlstead et al. found that providing hiding places for leopard cats (*Prionailurus bengalensis*) that were stressed by the nearby presence of large cat species (*Panthera* spp.) resulted in less pacing and reduced cortisol levels. Meaningful insights for improving zoo environments and understanding how specific environments affect behavior and health require a synthesis of information obtained from field and laboratory.

A goal of the enrichment of zoo environments is to make the behavioral repertoire and activity budgets of captive animals as close to those of their wild counterparts as possible. Since zoo animals have a much simpler behavioral repertoire when compared with wild counterparts, modification of feeding conditions should be one of the high-priority targets in zoo environmental projects. As a feeding enrichment, dead prey items, such as day-old chicks, fish, and mice can be hidden around the enclosure, to increase the level and diversity of activity. Effective environmental and behavioral enrichment can reduce stereotypic behaviors and promote behavioral patterns, which resemble those of wild felids.

Logs and sloping branches are used to increase the usable space inside the enclosures and to provide access to the hanging boxes, not forgetting that these small cat species, especially margays, also have arboreal habits. A floor with natural soil, when possible, is preferred. Vegetation substrate is important to enrich the environment and grass can be supplied as an alternative feeding habit (E.L.A. Monteiro-Filho, personal communication, 1998). Shelves can be strategically mounted within the enclosures to enable each animal to rest in the sun or shade.

In our experience, some small felids occasionally show a severe hair loss, probably by plucking it out and it is stress-related, but also occurs at the warmest period of the year. Adult domestic cats were found to lose 28.1 g hair per kilogram of body weight yearly with two-thirds of the total hair loss found in the feces. Changes in the pelage of the cat throughout the year are optimally regulated to obtain the densest coat at the coldest period of the year and the sparsest coat at the warmest period of the year.

Considering pairs that will be introduced for the first time, it is advisable to put them in adjacent enclosures that provide some controlled visual, olfactory, and physical contact during a period before the introduction. Another safer method of introduction for mating purposes consists of allowing each individual unrestricted access to the other's outdoor enclosure, initially in the absence of the other individual, but culminating in joint access. The method of gradual acquaintance through an experimentally induced overlap of "home ranges" can be effective if the male has a history of aggression.

Social interactions can play an important part in either stimulating or inhibiting the estrual display. Females, particularly if housed in isolation, may show an estrous induction if housed near or with a male, or at least within sight, smell, and sound of a male. In contrast, estrus may be suppressed if a subordinate is housed with a dominant female. Since in the wild small Neotropical felids are mainly solitary, males and females in a preferential way may be housed alone in separate enclosures and introduced to each other only for mating. Another aspect to be considered is that when we keep a couple of small felids together during a long period, sometimes they can lose sexual interest. Considering this, and the frequent inability to reliably identify when females are in estrus for appropriate timing of breeding introductions, one suggestion would be the introduction of a male during a time period such as 60 days (W. Moraes, personal communication, 1993), avoiding the female giving birth in the male's presence.

Mate preference is an explanation for failure to mate, and this possibility should not be discounted.

The male could be reintroduced again after a period of, at least, the gestation length for the considered species. Usually, it is preferable to move the male to avoid stressful conditions for the possible pregnant female, since moving small wild felids between enclosures can be difficult, and animals may suffer injuries especially during capture. Ultrasound, abdominal palpation, radiology, or laparoscopy can be used to diagnose pregnancy. In our experience, we diagnosed by laparoscopy a 14-day gestation in a margay, followed by normal parturition. Laparoscopy is an invasive technique and x-rays are detrimental to gametogenesis. Abortion or parturition may be self-evident but may pass unnoticed if the fetuses are eaten by the female or by the male.

Because females appeared to be particularly sensitive to disturbances when rearing, a similar management protocol for the seclusion of female cats was developed for Pampas cats (*O. colocolo*) at Cincinnati Zoo. A pair is housed in the exhibit for 65 days, after which the female is transferred to an off-exhibit enclosure for the next 90 days. Total privacy for the female and young as an important factor for successful breeding is also described for margays.

Whenever possible, kittens should remain with their mothers. Even so, an aspect to be considered is that kittens can be killed or neglected by the mother. One alternative in females with a history of killing or neglecting kittens without a known cause is removing the litter and hand-rearing them. But a consequence of hand-rearing is that usually the kittens are weaker and more disease susceptible when compared with mother-reared kittens. Colostrum is important and, as of now, no milk replacement is as good as natural milk. Hand-rearing also avoids the important contact and training between mother and kittens, which, of course, interferes with future behavior. If the mother neglects the kittens, they should be raised together, with domestic kittens, and/or by a nonhuman surrogate mother in a rich and varied environment. This fact must be considered especially when there are plans to translocate them in the future. Seidensticker and Forthman concluded their study with a plea to include the maintenance of behavioral competence in zoo animals as a primary goal of zoo biology. In conclusion, close cooperation between countries and workers of ex situ (zoos and breeding centers) and in situ (field programs and wildlife agencies) becomes increasingly necessary for effective global conservation.

Although parental rearing of young is preferred, occasionally it is necessary to hand rear animals to ensure their immediate health and long-term well-being. Hand-rearing techniques for small felids are documented in the literature.

Reproductive Evaluation

The initial evaluation should include an assessment of any disease problems and management. Other approaches are to check reproductive steroids and to examine the genital tract during a laparoscopy. Vaginal cytology can be used to give an idea of the estral cycle phase, but it also may stimulate ovulation.

When there is a suspicion of endometritis or vaginitis, bacteriological examination of vaginal swabs is difficult to interpret, because the organisms involved can be secondary opportunists or constitute part of the normal vaginal flora. Ocelots seem to present neutrophilic preponderance in vaginal smears with higher frequency.

Assisted Reproduction

If used appropriately, assisted reproduction, including artificial insemination (AI), in vitro fertilization (IVF), embryo transfer, and genome resource banking, has an application for the management and conservation of small felids. It is possible that in the future assisted reproduction will enable the movement of genetic material, rather than living animals. These techniques can also be used to breed incompatible individuals and to infuse captive populations with new genetic material. Because cats are susceptible to certain infectious viruses, assisted reproduction may be one of the new means of breeding using washed and pathogen-free gametes. Basic physiological information is essential to developing and applying assisted reproductive techniques.

Pregnancy success and offspring survival are still low with the use of assisted reproduction in felids treated with exogenous gonadotropins: equine chorionic gonadotrophin(eCG), or follicle-stimulating hormone (FSH) and human chorionic gonadotrophin (hCG). The eCG is used to promote follicular development and hCG to induce ovulation. The dosage of eCG or FSH and luteinizing hormone (LH) are species specific and require adjustment accordingly. Underdosage is ineffective whereas overdosage carries the risk of superovulation and hormonal imbalances. High levels of estradiol have been demonstrated to prevent implantation by prolonging oviduct transport of the ova. Besides this, recent findings suggest that hCG promotes the ancillary follicle formation that is frequently observed after ovulation in cats treated with eCG plus hCG regimens, possibly disrupting the maternal environment and decreasing fecundity following assisted reproductive procedures.

Contraception

Melengestrol acetate (MGA) is the most widely used contraceptive in zoo felids. Contraceptive effects of levonorgestrel have also been described in domestic cat. The contraceptive actions of MGA do not occur by suppressing folliculogenesis, and MGA-contracepted felids likely have endogenous estrogens that may confound progestin effects on the uterus. The use of MGA is associated with uterine lesions, including uterine cancer, but not with ovarian disease. Mammary hyperplasia can also occur following progestogen treatment.

Frequent use of progestogens or repeated pseudopregnancies induce progestogenic dominance and increase the risk of endometritis. Prostaglandins are indicated for the treatment of endometritis, although they do not appear to have a marked luteolytic action in cats. Their beneficial action is mediated through their ecbolic effect. Broad spectrum antibiotics are generally given.

REFERENCES

1. Anderson, D. 1977. Gestation period of Geoffroy's cat *Leopardus geoffroyi* bred at Memphis Zoo. International Zoo Yearbook 17:164–166.
2. Baldwin, C.J.; Peter, A.T.; Bosu, W.T.K.; and Dubielzig, R.R. 1994. The contraceptive effects of levonorgestrel in the domestic cat. Laboratory Animal Science 44:261–269.
3. Brown, J.L.; Wasser, S.K.; Wildt, D.E.; and Graham, L.H. 1994. Comparative aspects of steroid hormone metabolism and ovarian activity in felids, measured non-invasively in feces. Biology of Reproduction 51:776–786.
4. Cabrera, A.; and Yeppes, J. 1960. Mamiferos Sud Americanos: Vida, Costumbres y Descripcion [South American mammals: Life, behaviors and description]. Buenos Aires, Historia Natural Ediar, Companhia Argentina de Editores.
5. Callahan, P.; and Dulaney, M.W. 1997. Husbandry and breeding of the Pampas cat *Oncifelis colocolo* at Cincinnati Zoo and Botanical Garden. International Zoo Yearbook 35:100–103.
6. Carlstead, K.; Brown, J.L.; and Seidensticker, J. 1993. Behavioral and adrenocortical responses to environmental changes in leopard cats (*Felis bengalensis*). Zoo Biology 12:321–331.
7. Carlstead, K.; Brown, J.L.; and Strawn, W. 1993. Behavioral and physiological correlates of stress in laboratory cats. Applied Animal Behaviour Science 38:143–158.
8. Carlstead, K.; and Shepherdson, D.J. 1994. Effects of environmental enrichment on reproduction. Zoo Biology 13:447–458.
9. Caro, T.M. 1993. Behavioral solutions to breeding cheetahs in captivity: Insights from the wild. Zoo Biology 12:19–30.
10. Crespo, J.A. 1982. Ecología de la comunidad de mamíferos del Parque Nacional Iguazú, Misiones [Ecology of mammal community of the Iguassu National Park]. Rev. Mus. Argent. Cienc. Nat. "Bernardino Rivadavia", Ecol. 3(2):45–162.
11. Czekala, N.M.; Durrant, B.S.; Callison, L.; and Williams, M.; Millard, S. 1994. Fecal steroid hormone analysis of reproductive function in the cheetah. Zoo Biology 13:119–128.
12. Eaton, R.L. 1977. Breeding biology and propagation of the ocelot (*Leopardus* [*Felis*] *pardalis*). Zool. Garten 47:9–23.
13. Eaton, R.L. 1984. Interference competition among carnivores: A model for the evolution of social behavior. Carnivore 9–16.
14. Eaton, R.L. 1984. Survey of smaller felid breeding. Zool. Gart. new ser. 54(1–2):101–20.
15. Edwards, M.S.; and Hawes J. 1997. An overview of small field hand-rearing techniques and a case study for Mexican margay *Leopardus wiedii glaucula* at the Zoological Society of San Diego. International Zoo Yearbook 35:90–94.
16. Ewer, R.F. 1973. The Carnivores. London, Weidenfeld and Nicolson.
17. Fagen, R.M.; and Wiley, K.S. 1978. Felid paedomorphosis, with special reference to *Leopardus*. Carnivore 1:72–81.
18. Foreman, G.E. 1988. Behavioral and genetic analysis of Geoffroy's cat (*Felis geoffroyi*) in captivity. Doctoral thesis, Ohio State University, Columbus.
19. Foreman, G.E. 1997. Breeding and maternal behaviour in Geoffroy's cats *Oncifelis geoffroyi*. International Zoo Yearbook 35:104–115.
20. Forthman-Quick, D.L. 1984. An integrative approach to environmental engineering in zoos. Zoo Biology 3:65–77.
21. Gilkison, J.J.; White, B.C.; and Taylor, S. 1997. Feeding enrichment and behavioural changes in Canadian lynx *Lynx canadensis* at Louisville Zoo. International Zoo Yearbook 35:213–216.
22. Graham, L.H.; Goodrowe, K.L.; Raeside, J.I.; and Liptrap, R.M. 1995. Non-invasive monitoring of ovarian function in several felid species by measurement of fecal estradiol-17β and progestins. Zoo Biology 14:223–237.
23. Green, R. 1971. Geoffroy's cat (*Felis geoffroyi*). Chester Zoo News (September):1–2.
24. Green, R. 1991. Wild cat species of the world. Plymouth, U.K., Basset.
25. Gross, T.S. 1992. Development and use of faecal steroid analyses in several carnivore species. In Proceedings of the 1st International Symposium on Faecal Steroid Monitoring in Zoo Animals. pp. 55–61.
26. Gruffydd-Jones, T.J. 1993. Disorders of the reproductive system. In J. Wills and A. Wolf, eds., Handbook of Feline Medicine. Oxford, Pergamon Press, pp. 213–222.
27. Guggisberg, C.A.W. 1975. Wild Cats of the World. New York, Taplinger.
28. Hendriks, W.H.; Tarttelin, M.F.; and Moughan, P.J. 1998. Seasonal hair loss in adult domestic cats (*Felis catus*). Journal of Animal Physiology and Animal Nutrition 79:92–101.

29. Housse, P.R. 1953. [Wild animals of Chile (in Spanish)]. Santiago, Ediciones Univerisdad de Chile.
30. Hulley, J.T. 1976. Maintenance and breeding of captive jaguarundis *Felis yagouaroundi* at Chester Zoo and Toronto. International Zoo Yearbook 16:120–122.
31. Kazensky C.A.; Munson L.; and Seal U.S. 1998. The effects of melengestrol acetate on the ovaries of captive wild felids. Journal of Zoo and Wildlife Medicine 29:1–5.
32. Kreger, M.D.; Hutchins, M.; and Fascione, N. 1998. Context, ethics, and environmental enrichment in zoos and aquariums. In D.J. Shepherdson, J.D. Mellen, and M. Hutchins, eds., Second Nature—Environmental Enrichment for Captive Animals. Portland, Maine, Smithsonian Institution Press, pp. 59–82.
33. Laurenson, M.K. 1993. Early maternal behavior of wild cheetahs: Implications for captive husbandry. Zoo Biology 12:31–43.
34. Law, G.; and Boyle, H. 1983. Breeding the Geoffroy's cat (*Felis geoffroyi*) at Glasgow Zoo. International Zoo Yearbook 23:191–195.
35. Law, G.; and Tatner, P. 1998. Behaviour of a captive pair of clouded leopards (*Neofelis nebulosa*): Introduction without injury. Animal Welfare 7:57–76.
36. Leyhausen, P.; and Falkena, M. 1966. Breeding the Brazilian ocelot-cat *Leopardus tigrinus* in captivity. International Zoo Yearbook 6:176–182.
37. Lindburg, D.G.; Durrant, B.S.; Millard, S.E.; and Oosterhuis, J.E. 1993. Fertility assessment of cheetah males with poor quality semen. Zoo Biology 12:97–103.
38. Mansard, P. 1997. Breeding and husbandry of the Margay *Leopardus wiedii yucatania* at the Ridgeway Trust for Endangered Cats, Hastings. International Zoo Yearbook 35:94–100.
39. Mellen, J.D. 1989. Reproductive behavior of small captive exotic cats (*Felis* spp.). Doctoral thesis, University of California, Davis.
40. Merola, M. 1994. A reassessment of homozygosity and the case for inbreeding depression in the cheetah, *Acinonyx jubatus:* Implications for conservation. Conservation Biology 8:961–971.
41. Michael, R.P. 1961. Observations upon the sexual behaviour of the domestic cat (*Felis catus* L.) under laboratory conditions. Behaviour 18:1–24.
42. Molteno, A.J.; Sliwa, A.; and Richardson, P.R.K. 1998. The role of scent marking in a free-ranging, female black-footed cat (*Felis nigripes*). Journal of Zoology 245:35–41.
43. Mondolfi, E. 1986. Notes on the biology and status of the small wild cats in Venezuela. In S.D. Miller and D.D. Everet, eds., Cats of the World: Biology, Conservation and Management. Washington, D.C., National Wildlife Federation, pp. 125–146.
44. Moraes, W.; Morais, R.N.; Moreira, N.; Lacerda, O.; Gomes, M.L.F.; Mucciolo, R.G.; and Swanson, W.F. 1997. Successful artificial insemination after exogenous gonadotropin treatment in the ocelot *(Leopardus pardalis*) and tigrina (*Leopardus tigrina*). In Proceedings American Association of Zoo Veterinarians. Houston, Texas, AAZV, pp. 334–336.
45. Moreira, N.; Moraes, W.; and Santos, L.C. 1994. Estudo de parâmetros reprodutivos de *Felis tigrina, Felis pardalis* e *Felis wiedii.* Nota prévia [Study of reproductive parameters in *Felis tigrina, Felis pardalis* and *Felis wiedii*. Previous note]. In Proceedings of the 18th Brazilian Congress of Zoological Society, Rio de Janeiro, Sociedade de Zoolgicos do Brasil, p. 59
46. Moreira, N.; Monteiro-Filho, E.L.A.; Moraes, W.; Swanson, W.F.; Graham, L.H.; Wildt, D.E.; Pasquali, O.L.; Gomes M.L.F.; Morais, R.N.; and Brown, J.L. 2000. Reproductive steroid hormones and ovarian activity in felids of the *Leopardus* genus. (in review).
47. Morimura, N.; and Ueno, Y. 1998. Behavior patterns of 9 mammals in the zoo: The comparison among species, and different environments. Japanese Journal of Animal Psychology 48:33–45.
48. Möstl, E.; Lehman, H.; and Wenzel, U. 1993. Gestagens in the faeces of mink and cats for monitoring corpus luteum activity. Journal of Reproduction and Fertility Supplement 47:540–541.
49. Nowak, R.M.; and Paradiso, J.L. 1983. Walker's Mammals of the World, Vol. 2. Baltimore, Johns Hopkins University Press.
50. Nowell, K.; and Jackson, P. Eds. 1996. Wild Cats: Status Survey and Conservation Action Plan. Gland, Switzerland, IUCN.
51. Oliveira, T.G. 1994. Neotropical Cats: Ecology and Conservation, São Luís, EDUFMA.
52. Pantiff, J.A.; and Anderson, D.E. 1980. Breeding the margay at New Orleans Zoo. International Zoo Yearbook 20:223–224.
53. Petersen, M.K. 1977. Behaviour of the margay. In R.L. Eaton, ed., The World's Cats. Seattle, Washington, Carnivore Research Institute, University of Washington, pp. 69–76.
54. Petersen, M.K.; and Petersen, M.K. 1978. Growth rates and other post-natal developmental changes in margays. Carnivore 1(1):87–92.
55. Quillen, P. 1981. Hand-rearing the little spotted cat or oncilla (*Felis tigrinus*). International Zoo Yearbook 21:240–242.
56. Rabinovich, J.; Capurro, A.; Folgarait, P.; Kitzberger, T.; Kramer, G.; Novaro, A.; Puppo, M.; and Travaini, A. 1987. Estado del conocimiento de 12 especies de la fauna silvestre Argentina e valor comercial [Knowledge status of 12 species of Argentina wildlife and commercial value]. Paper presented at the 2nd Workshop Elaboracin de propuestas de investigacin orientada al manejo de la fauna silvestre de valor comercial [Elaboration of investigation guided proposals to the handling of the wild fauna with commercial value], Buenos Aires.
57. Redford, K.H.; and Eisenberg, J.F. 1992. Mammals of the Neotropics: the Southern Cone, Vol. 2. Chicago, University of Chicago Press.

58. Risler, L.; Wasser, S.K.; and Sackett, G.P. 1987. Measurement of excreted steroids in *Macaca nemestrina*. American Journal of Primatology 12:91–100.
59. Robinson, R. 1970. Inheritance of the black form of the leopard. Genetics 41:190.
60. Scheffel, W.; and Hemmer, H. 1975. Breeding Geoffroy's cat *Leopardus geoffroyi salinarum* in captivity. International Zoo Yearbook 15:152–54.
61. Seidensticker, J.; and Forthman, D. 1998. Evolution, ecology, and enrichment. In D.J. Shepherdson, J.D. Mellen, and M. Hutchins, eds., Second Nature—Environmental Enrichment for Captive Animals. Portland, Maine, Smithsonian Institution Press, pp. 15–29.
62. Simon, F.; Cassaro, K.; and Quillen, P. 1997. Small felid breeding project at Sao Paulo Zoo. International Zoo Yearbook 35:159–164.
63. Slattery, J.P.; and O'Brien S.J. 1998. Patterns of Y and X chromosome DNA sequence divergence during the Felidae radiation. Genetics 148:1245–1255.
64. Swanson, W.F.; Morais, R.N.; Moreira, N.; Gomes, M; Moraes, W.; Brousset, D.; Gallindo, F.; Esquivel, C.; Yarto, E.; Canales, D.; Wasser, S.; and Brown, J.L. 1999 Collaborative reproductive research and training programs for the conservation of Latin American felids and primates. In Proceedings of the 7th World Conference on Breeding Endangered Species in Captivity. Cincinnati Zoo, p. 262.
65. Swanson, W.F.; and Wildt D.E. 1997. Strategies and progress in reproductive research involving small cat species. International Zoo Yearbook 35:152–159.
66. Swanson, W.F.; Wolfe, B.A.; Brown, J.L.; Martin-Jimenez, T.; Riviere, J.E.; Roth, T.L.; and Wildt, D.E. 1997. Pharmacokinetics and ovarian-stimulatory effects of equine and human chorionic gonadotropins administered singly and in combination in the domestic cat. Biology of Reproduction 57:295–302.
67. Widholzer, F.L.; Bergmann, M.; and Zotz, C. 1981. Breeding the little spotted cat. International Zoo News 28:17–22.
68. Wielebnowski, N. 1996. Reassessing the relationship between juvenile mortality and genetic monomorphism in captive cheetahs. Zoo Biology 15:353–369.
69. Wielebnowski, N.; and Brown, J.L. 1998. Behavioral correlates of physiological estrus in cheetahs. Zoo Biology 17:193–209.
70. Wildt, D.E.; and Roth, T.L. 1997. Assisted reproduction for managing and conserving threatened felids. International Zoo Yearbook 35:164–172.
71. Wooster, D.S. 1997. Enrichment techniques for small felids at Woodland Park Zoo, Seattle. International Zoo Yearbook 35:208–212.
72. Wozencraft, W.C. 1993. Order Carnivora. In D.E. Wilson and D.M. Reeder, eds., Mammal Species of the World, 2nd Ed. Washington, D.C., Smithsonian Institution Press, pp. 279–348.
73. Quillin, P. 1993. Kodkod (*Oncifelis guigna*): description and behavior. http://lynx.uio.no/catfolk/guigna01.html. Accessed June 7, 2000.

REPRODUCTION IN JAGUARS

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INTRODUCTION

The jaguar (*Panthera onca*), the largest cat of the Americas, inhabits different regions from North Mexico to Argentina.[23] However, the number of animals in the wild have been reduced by a variety of factors, the main one being the loss and fragmentation of their habitat, reducing jaguar populations to critical levels due to loss of genetic diversity. The consequences of genetic diversity loss have been well documented by many authors.[1, 20, 21, 22, 27]

Considering the importance of the maintenance of genetic diversity and the problem with captive jaguar populations, that have been reported to be mostly generic in Brazilian[15] and American (jaguar studbook) zoos, reproduction of wild-caught and captive jaguars of known origin needs to become an integral component of preservation efforts.

Assisted reproductive technologies such as AI, IVF, and embryo transfer (ET) can be viewed as important tools for safeguarding the jaguar, based on the highly successful applications to domestic livestock and humans[35] and the recent progress obtained in wild cat species.

In recent years, offspring have been produced using AI in the cheetah (*Acynonyx jubatus*),[7] clouded leopard (*N. nebulosa*),[9] tiger (*Panthera tigris*)[4], leopard cat (*P. bengalensis*)[34], puma (*P. concolor*),[2] and ocelot (*L. pardalis*),[30] and the use of IVF and ET have produced offspring in the tiger (*P. tigris*)[3] and Indian desert cat (*Felis silvestris*).[26] However, an assisted reproduction regimen that works well for one species can fail in another because of naturally occurring physiological specificities, even among closely related species within the same family and genus.[34]

We are describing here the progress of reproduction techniques that have been used for a number of cat species and the present and potential application of these techniques for jaguar conservation.

SEMEN COLLECTION, EVALUATION, AND CRYOPRESERVATION

In the jaguar, spermatozoa can be obtained from caudal epididymis of dead animals[18] or by electroejaculation in anesthetized jaguars.[8, 16]

TABLE 27.6. Semen characteristics of captive and free-living jaguars (*Panthera onca*) (mean + SEM)

	Reference 13[a]	Reference 8	Reference 16	Unpublished Data[b]
Number of ejaculates	6	5	43	3
Ejaculate volume (mL)	2.1 ± 0.5	2.7 ± 0.6	7.4 ± 0.6	4.6 ± 1.3
Sperm concentration ($\times 10^6$/mL)	39.1 ± 11.0	12.0 ± 1.9	6.2 ± 0.4	4.7 ± 2.4
Sperm motility (%)	71.7 ± 3.0	82.0 ± 5.8	62.6 ± 1.7	76.6 ± 11.1
Sperm progressive motility	—	4.1 ± 0.3	2.7 ± 0.1	3.6 ± 0.5
Normal sperm	61.4 ± 3.2	58.2 ± 11.1	46.7 ± 0.9	65.0 ± 5.1

[a]There is no value for sperm progressive motility.
[b]Data from free-living jaguars captured for radiotelemetry studies between 1995 to 1998, Porto Primavera, Brazil.

The spermatozoa recovery from dead animals involves flushing or macerating the ductus deferens and cauda epididymis in a physiological salt solution or cryoprotective diluent.[8]

Semen collection by electroejaculation has been done in 28 cat species[5] using the standard protocol developed by Wildt et al.[33]

Semen collection procedure is performed in anesthetized animals. In the jaguar, the tiletamine-zolazepam combination in a 10 mg/kg proportion is recommended.[16] A 29 × 2.3-cm probe is used and 15 cm is introduced into the rectum.[16] The electroejaculator is the same as that used for bovines.

Semen from each collection is immediately evaluated for total volume and pH, motility, rate of forward progression, and morphology as described by Howard.[8] The results of evaluation of semen collection from captive and free-living jaguars are shown in Table 27.6.

Cryopreservation of semen samples has been done using the standard protocol for cat species described by Platz and Seager [24] or a protocol for tigers described by Nelson et al.[19] In the first case, after diluting sperm with Hams' F10 media (1:1), it is centrifuged at 300*g* for 10 minutes, the supernatant is removed, and the sperm pellet is resuspended with PDV semen cryodiluent (20% egg yolk, 11% lactose, 4% glycerol). Sperm are loaded then into straws (0.25 mL) and placed at refrigerated (4–7°C) for 2 hours. They are then frozen in N_2 vapor and then plunged into liquid N_2 for storage. In the second protocol the dilution is made using Hepes-TALP (1:1) and the semen cryodiluent is a Tris-egg yolk–based diluent (Biladyl A; Minitube Co., Germany). After 1 hour of refrigeration at 4°C Biladyl A containing 4% glycerol is added.

Post-thaw evaluation of jaguar semen has demonstrated poor semen quality with 30% motility and mean status of 2 for captive and free-ranging jaguars. No differences have been observed between protocols (unpublished data).

Other procedures for testing sperm function are the homologous (cat) or heterologous zona-free hamster[8] or bovine ovum penetration assay.[18,19] Assays using heterologous zona-free hamster ovum reveal a penetration rate for post-thaw jaguar sperm of 26.5%[25]. These assays are also effective for studying factors influencing IVF.[8] Paz et al.,[25] observed that the optimum rate of ovum penetration by jaguar spermatozoa is greater using Percol gradients (26.5%) than swim-up (20%). Domestic cat oocytes can be used to assess capability of the spermatozoon to bind to the zona pellucida and to penetrate into the zona.[8] The combination of zona-free hamster or bovine assays and intact zona cat assay can be useful to analyze three critical stages of sperm-ovum interaction: binding to the zona pellucida, penetration through the zona, and sperm nuclear decondensation within the vitellus.[6,8]

BASIC REPRODUCTIVE PHYSIOLOGY

An understanding of basic reproductive physiology is important for the effective management of captive and free-living wildlife species.[12] The use of noninvasive techniques will permit us to improve our knowledge in this area. However, at the moment, few data have been generated to understand the basic reproductive physiology of the jaguar. After serum measurements by radioimmunoassay of progesterone and LH and serial laparoscopy, the duration of the estrus was 12.0 ± 1.0 days and the estrous cycle was 47.2 ± 5.4 days for one female jaguar.[32] Sexual maturity was reported to be between 2 and 2.5 years old in the female jaguar [14,32] and 3 years old in the male jaguar.[14] Male jaguars can produce sperm throughout the year without variations in the semen quantity and quality.[17]

OOCYTE RECOVERY, IN VITRO MATURATION, IN VITRO FERTILIZATION, AND EMBRYO TRANSFER

Oocyte recovery is based on two techniques: (1) postmortem collection or after ovariectomy, and (2) follicle aspiration using laparoscopy.

In the first case, ovarian oocytes can be recovered after 36 hours from ovaries stored ex situ in a physiological medium.[11] Previously, Johnston et al.[10] reported that cat follicular oocytes remain capable of maturing to metaphase II when recovered within 24-32 hours of initial storage.

In the second case, laparoscopy is preceded by an exogenous gonadotrophin treatment to induce ovarian activity. The use of equine chorionic gonadotrophin (eCG) followed by human chorionic gonadotrophin (hCG) treatment has been successful for many cat species.[2,3,7,9,30] However, multiple treatments with eCG/hCG can progressively decrease ovarian responsiveness owing to the formation of eCG and hCG neutralizing immunoglobulins.[29] A new treatment protocol for tigers using porcine FSH (pFSH) and LH (pLH) has been used as repeated stimulations every 100 days for the recovery of mature oocytes (N. Loskutoff, personal communication). Based on the results of ovarian stimuli in tigers, we are using pFSH and pLH for ovarian stimulation in the jaguar. The preliminary results are shown in the Table 27.7.

From the recovered oocytes, 74 (81%) were classified as good, 6 (7%) were classified as fair, 4 (4%) were classified as poor. Seven of them (8%) were degenerated.

The coculture of fresh jaguar sperm and recovered oocytes resulted in 2- to 4-cell embryos (unpublished data) that have been cryopreserved using the vitrification technique as described by Vatja et al.[31] The coculture of thawed jaguar sperm and recovered oocytes were unable to produce embryos. New trials will be performed mainly to test different protocols for semen cryopreservation. Offspring production is another step once embryo cryopreservation technique and time of embryo transfer problems are resolved; they can be barriers for the complete success of the IVF technique.

CONCLUSIONS

Assisted reproduction techniques have been successful for many cat species. For jaguars, we are successfully collecting and evaluating semen using standard protocols. However, we have observed that the semen-freezing protocol used for many cat species were not optimal to preserve jaguar semen. Meanwhile, as data accumulate on the basic reproductive physiology of the jaguar and the results of ovarian stimulation and oocyte retrieval accumulate, we will be in a better position to choose the best time for the artificial reproduction procedures such as AI, IVF, and ET. The pFSH and pLH treatment has successfully induced ovarian stimulation in jaguars. Embryo production by IVF using thawed sperm continues to be a challenge for our team. Based on the successful results after assisted reproductive techniques for many cat species, we believe that AI, IVF, and ET can be very important tools for preservation of the jaguar.

REFERENCES

1. Barone, M.A.; Roelke, M.E.; Howard, J.G.; Brown, J.L.; Anderson, A.E.; and Wildt, D.E. 1994. Reproductive characteristics of male Florida panthers; Comparative studies from Florida, Texas, Colorado, Latin America, and North American zoos. Journal of Mammalogy 75(1):150–162.
2. Barone, M.A.; Wildt, D.E.; Byers, A.P.; Roelke, M.E., Glass, C.M.; and Howard, J.G. 1994. Gonadotrophin dose and timing of anaesthesia for laparoscopic artificial insemination in the puma (*Felis concolor*). Journal of Reproduction and Fertility 101(1):103–108.
3. Donoghue, A.M.; Johnston, L.A.; Seal, U.S.; Armstrong, D.L.; Tilson, R.L.; Wolf, P.; Petrini, K.; Simmons, L.G.; Gross, T.; and Wildt, D.E. 1990. In vitro fertilization and embryo development in vitro and in vivo in the tiger (*Panthera tigris*). Biology of Reproduction 43(5):733–743,
4. Donoghue, A.M.; Johnson, L.A.; Armstrong, D.L.; Simmons, L.G.; and Wildt, D.E. 1993. Birth of a siberian tiger cub (*Panthera tigris altaica*) following laparoscopic intrauterine artificial insemination. Journal of Zoo Wildlife Medicine 24(2):185–189.
5. Howard, J.G.; Bush, M.; Hall, L.L.; and Wildt, D.E. 1984. Morphological abnormalities in spermatozoa of 28 species of non-domestic felids. In Proceedings of the

TABLE 27.7. Preliminary results of ovarian stimulation in the jaguar female treated with 50 IU pFSH followed by 25 IU pLH, intramuscularly

No. Females Superovulated/ No. Females Treated	Total No. Visualized Follicles ≥ 2 mm in Diameter	Total No. of Recovered Oocytes
6/6 (100%)	117	91

10th International Congress on Animal Reproduction and Artificial Insemination. p. 57.
6. Howard, J.G.; Bush, M.; and Wildt, D.E. 1991. Teratospermia in domestic cats compromises penetration of zona-free hamster ova and cat zonae pellucidae. Journal of Andrology 12(1):36–45.
7. Howard, J.G.; Donoghue, A.M.; Barone, M.A.; Goodrowe, K.L.; Blumer, E.; Snodgrass, K.; Starnes, D.; Tucker, M.; Bush, M.; and Wildt, D.E. 1992. Successful induction of ovarian activity and laparoscopic intrauterine artificial insemination in the cheetah (*Acinonyx jubatus*). Journal of Zoo Wildlife Medicine 23(4):288–300.
8. Howard, J.G. 1993. Semen collection and analysis in carnivores. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 390–398.
9. Howard, J.G.; Byers, A.P.; Brown, J.L.; Barret, S.J.; Evans, M.Z.; Schwartz, R.J.; and Wildt, D.E. 1996. Successful ovulation induction and laparoscopic intrauterine artificial insemination in the clouded leopard (*Neofelis nebulosa*). Zoo Biology 15(1):55–70.
10. Johnston, L.A.; O′Brien, S.J.; and Wildt, D.E. 1989. In vitro maturation and fertilization of domestic cat follicular oocytes. Gamete Research 24:343–356.
11. Johnston, L.A.; Donoghue, A.M.; O′Brien, S.J.; and Wildt, D.E. 1991. Rescue and maturation in vitro of follicular oocytes collected from nondomestic felid species. Biology of Reproduction 45(6):898–906.
12. Lasley, B.L.; and Kirkpatrick, J.F. 1991. Monitoring ovarian function in captive and free-ranging wildlife by means of urinary and fecal steroids. Journal of Zoo Wildlife Medicine 22(1):23–31.
13. Mies-Filho, A.; Telechea, N.L.; Bohrer, J.L.; and Wallawer, W.P. 1974. Produção espermática em onça pintada (*Panthera onca*). Arquivos da Faculdade de Veterinária da Universidade Federal do Rio Grande do Sul 2(1):55–65.
14. Mondolfi, E.; and Hoogesteijn, R. 1986. Notes on the biology and status of the jaguar in Venezuela. In S.D. Miller and D.D. Everett, eds., Cats of the World: Biology, Conservation and Management. Washington, D.C., National Wildlife Federation, pp. 85–124.
15. Morato, R.G.; and Gasparini, R.L. 1994. Levantamento preliminar sobre a situação da onça-pintada (*Panthera onca*) em cativeiro. Anais 18° Congresso da Sociedade de Zoolgicos do Brasil. p. 21.
16. Morato, R.G.; Guimarães, M.A.B.V.; Nunes, A.L.V.; Carciofi, A.C.; Ferreira, F.; Barnabe, V.H.; and Barnabe, R.C. 1998. Semen collection and evaluation in the jaguar. Brazilian J Vet Res An Sci. 35(4):30–34.
17. Morato, R.G.; Guimarães, M.A.B.V.; Ferreira, F.; Verreschi, I.T.N.; and Barnabe, R.C. 2000. Reproductive characteristics of captive male jaguars. Brazilian J Vet Res An Sci. (in press).
18. Morato, R.G.; Nelson, K.; Finnegan, J.; Volenec, D.; and Loskutoff, N.M. 2000. Effect of cryoprotectant and thawing method for cryopreserving epididymal spermatozoa from the jaguar (*Panthera onca*) for heterologous in vitro fertilization. Arquivos da Faculdade de Veterinária da Universidade Federal do Rio Grande do Sul (in press).
19. Nelson, K.L.; Crichton, E.G.; Doty, L.; Volenec, D.E.; Morato, R.G.; Pope, C.E.; Dresser, B.L.; Brown, C.S.; Armstrong, D.L.; and Loskutoff, N.M. 1999. Heterologous and homologous fertilizing capacity of cryopreserved felid sperm: A model for endangered species. Theriogenology, 51(1):290.
20. O'Brien, S.J.; Roelke, M.E.; Marker, L.; Newman, A.; Winkler, C.A.; Meltzer, L.; Colly, L.; Evermann, J.F.; Bush, M.; and Wildt, D.E. 1985. Genetic basis for species vulnerability in the cheetah. Science 227:1428–1434.
21. O'Brien, S.J.; Wildt, D.E.; and Bush, M. 1986. The cheetah in genetic peril. Scientific American 254(5):84–92.
22. O'Brien, S.J.; and Evermann, J.F. 1988. Interactive influence of infectious disease and genetic diversity in natural populations. Trends in Ecology and Evolution 3(10):254–259.
23. Oliveira, T.G. 1994. Cats: Ecological and Conservation. São Luís, Edusma, p. 244.
24. Platz, C.C., Jr.; and Seager, W.J. 1978. Semen collection by electroejaculation in the domestic cat. Journal of American Veterinary Medical Association 173(10):1353–1355.
25. Paz, R.C.R.; Zuge, R.M.; Morato, R.G.; Barnabe, V.H.; Barnabe, R.C.; and Felippe, P.A.N. 1999. Capacidade de penetração de sêmen congelado de onça pintada (*Panthera onca*) em ocitos heterlogos. In Anais do 23° Congresso Brasileiro de Zoolgicos. p. 37.
26. Pope, C.E.; Gelwicks, E.J.; Wachs, K.B.; Keller, G.L.; Maruska, E.J.; and Dresser, B.L. 1989. Successful interspecies transfer of embryos from the Indian desert cat (*Felis silvestris*) to the domestic cat (*Felis catus*) following in vitro fertilization. Biology of Reproduction 40(suppl.):61.
27. Raals, K.; Brugger, K.; and Ballou, J. 1979. Inbreeding and juvenile mortality in small populations of ungulates. Science 206:1101–1103.
28. Roelke, M.E.; Martenson, J.S.; and O'Brien, S.J. 1993. The consequences of demographic reduction and genetic depletion in the endangered Florida panther. Current Biology 3(6):340–350.
29. Swanson, W.F.; Horohov, D.W.; and Godke, R.A. 1995. Production of exogenous gonadotrophin-neutralizing immunoglobulins in cats after repeated eCG-hCG treatment and relevance for assisted reproduction in felids. Journal of Reproduction and Fertility 105(1):35–41.
30. Swanson, W.F.; Howard, J.G.; Roth, T.L.; Brown, J.L.; Alvarado, T.; Burton, M.; Starnes, D.; and Wildt, D.E. 1996. Responsiveness of ovaries to exogenous gonadotrophins and laparoscopic artificial insemination with frozen-thawed spermatozoa in ocelots (*Felis pardalis*). Journal of Reproduction and Fertility 106(1):87–94.
31. Vatja, G.; Hold, P.; Kuwayama, M.; Booth, P.J.; Jacobsen, H.; Greve, T.; and Callesen, H. 1998. Open-pulled straws (OPS) vitrification: A new way to reduce cryoinjuries of bovine ova and embryos. Molecular Reproductive Development 51(1):53–58.

32. Wildt, D.E.; Platz, C.C.; Chakraborty, P.K.; and Seager, S.W.J. 1979. Oestrous and ovarian activity in a female jaguar. Journal of Reproduction and Fertility 56(2):555–558.
33. Wildt, D.E.; Bush, M.; Howard, J.G.; O'Brien, S.J.; Meltzer, D.; Van Dyk, A.; Ebedes, H.; and Brand, D.J. 1983. Unique seminal quality in the South African cheetah and a comparative evaluation in the domestic cat. Biology of Reproduction 29(4):1019–1025.
34. Wildt, D.E.; Monfort, S.L.; Donoghue, A.M.; Johnston, L.A.; and Howard, J.G. 1992. Embryogenesis in conservation biology—Or how to make an endangered species embryo. Theriogenology 37(1):161–184.
35. Wildt, D.E.; Seal, U.S.; and Rall, W.F. 1993. Genetic resource banks and reproductive technology for wildlife conservation. In J.G. Cloud and G.H. Thorgaard, eds., Genetic conservation of salmonid fishes. New York, Plenum Press, pp. 159–173.

REPRODUCTION IN SMALL FELID MALES

Rosana Nogueira de Morais

Of the 10 felid species endemic to Latin America, 8 are assigned as "small cats," based on an average body weight of less than 20 kg. The scarce available data about the status of wild and captive populations for the small felids were recently reviewed and discussed during the South American Felid Conservation Assessment Management Plan (CAMP). Most of the taxa were either assigned to the endangered or vulnerable categories by Mace-Lande criteria.[3] Captive propagation was recommended for most species, however implementation of these ex situ conservation strategies are hampered by a host of factors, including the small-size and dispersed captive populations (< 50 individuals), lack of basic reproductive information, and poor husbandry conditions, which may all be partly responsible for the very low breeding success in captivity. More recently, reproductive investigations have been conducted to begin generating a database for South American small felids.[4,5,8] Techniques used in the domestic cat and larger felids, such as electroejaculation, laparoscopy, and noninvasive fecal steroid metabolite analysis, have been applied successfully in the ocelot (*L. pardalis*), Tigrina (*L. tigrinus*), and Margay (*L. wiedii*) to study ovary and testicular function in these species. Small felid-breeding programs in different Brazilian institutions are also trying to improve husbandry conditions to overcome captive stress. Consistent success had been reported for tigrinas and Margays both at Itaipu Breeding Center (unpublished data) and at the Sao Paulo Zoo.[1] A few data on ejaculate characteristics is also available from individuals maintained in North American institutions.[2]

REPRODUCTIVE PHYSIOLOGY

Age at sexual maturity in males is assumed to range from 10–12 months in the tigrina (*L. tigrinus*) to 24-36 months in the jaguarundi (*H. yaguarundi*), although systematic data is limited for most species. Two Brazilian tigrina males that submitted to regular reproductive examinations showed 70% of motile sperm in the ejaculate when total testicular volume reached a value of about 3.0 cm^3, at 16 months of age. Ejaculate volume by this age was also similar to the mean value obtained for adult males (R. Morais, O. Lacerda, M.L.F. Gomes, unpublished data).

Basic adult male reproductive anatomy resembles that of the domestic cat and is similar among species. Testes are located in the scrotum caudodorsal to the penis and mean testicular volume per kilogram of body mass in adult males range from 1.4 cm^3 in tigrinas to 2.2 cm^3 in the ocelot (*L. pardalis;* Table 27.8). At histological examination, the ocelot testis presents an arrangement similar to that described for the domestic dog, with the intertubular tissue displaying abundance of Leydig cells, with very little intertubular connective tissue (Morais et al., unpublished data; Figure 27.2). The felid penis is directed backward and the cranial two-thirds of the penis is covered with visible spines, which are testosterone dependent and differ in size among species. The exception is the male Margay (*L. wiedii*), which has no spine along the penile shaft. This finding maybe related to the occurrence of spontaneous ovulation (nonreflex ovulation) in Margay females, suggesting that the neuroendocrine events initiated during coitus may not be essential to induce the preovulatory luteinizing hormone (LH) surge in this small cat species.

Male reproductive traits for six small cat species are summarized in Table 27.8. Excepting for ocelots, high numbers of structurally malformed sperm cells are observed for all species. Jaguarundis (*H. yaguarundi*) have been found to have less than 30% of morphologically normal spermatozoa. This low sperm quality is due to a host of poorly defined factors, including species specificity, nutrition, stress, seasonality, and inbreeding. Genetic impoverishment is not likely to be critical since most of the captive males in Latin American zoos are wild caught, although that does not exclude the possibility of a reduced genetic diversity in the wild populations.

Reproductive studies with tigrinas, margays, and ocelots have demonstrated that consistent supplementation of meat-based diets with vitamins and minerals may improve significantly sperm production. Total sperm per ejaculate and percent of normal sperm collected by standardized electroejaculation were incremented in animals maintained on supplemented diets

TABLE 27.8. Characteristics of electroejaculated semen and testicular volume of captive small felids in Latin American zoos

Species	Number of Ejaculates	Ejaculate Volume (mL)	Sperm Concentration ($\times 10^6$/mL)	Total Sperm/ejaculate ($\times 10^6$)	Sperm Motility Index[a]	Normal Sperm (%)	Testis Volume[b] (cm^3)
Ocelot	38	0.62 ± 0.08	53.8 ± 17.8	34.3 ± 11.9	70.4 ± 2.3	58.4 ± 5.8	30.4 ± 3.1
(L. pardalis)	(45)[c]	1.34 ± 0.10	103.0 ± 10.4	134.4 ± 17.1	77.6 ± 1.2	79.5 ± 2.3	32.1 ±1.3
Margay	27	0.31 ± 0.05	14.2 ± 5.3	6.4 ± 2.8	62.8 ± 5.3	39.5 ± 7.7	10.3 ± 2.0
(L. wiedii)	(43)[c]	0.50 ± 0.04	76.2 ± 10.5	32.9 ± 1.3	70.9 ± 1.2	56.6 ± 2.8	6.2 ± 0.2
Tigrina	18	0.11 ± 0.02	78.5 ± 33.8	9.4 ± 3.7	62.1 ± 5.7	35.6 ± 6.0	3.6 ± 0.7
(L. tigrinus)	(58)[c]	0.27 ± 0.01	447.5 ± 58.9	108.9 ± 13.3	74.0 ± 1.7	57.0 ± 3.5	4.2 ± 0.2
Geoffroy's cat *(O. geoffroyi)*	24	0.21 ±0.03	66.5 ± 24.4	8.5 ± 1.7	64.0 ± 4.7	46.9 ± 5.0	5.5 ± 0.4
Pampas cat *(O. colocolo)*	02	0.08 ± 0.01	364.0 ± 326.0	22.6 ± 20.2	81.3 ± 6.3	56.5 ± 0.5	2.8 ± 0.7
Jaguarundi *(H. yaguarundi)*	21	0.08 ± 0.02	7.2 ± 4.0	1.0 ± 0.5	57.8 ± 2.5	25.7 ± 4.6	6.0 ± 1.6

Source: Data compiled from references 4, 5, and 8.
[a]Sperm Motility Index = [% motility + (20 × sperm progressive motility)]/2.
[b]Testicular Volume = (length × width^2 × 0.524) and total testes volume = right volume + left volume.
[c]Males receiving supplemented diet.
Values given are mean ± SEM.

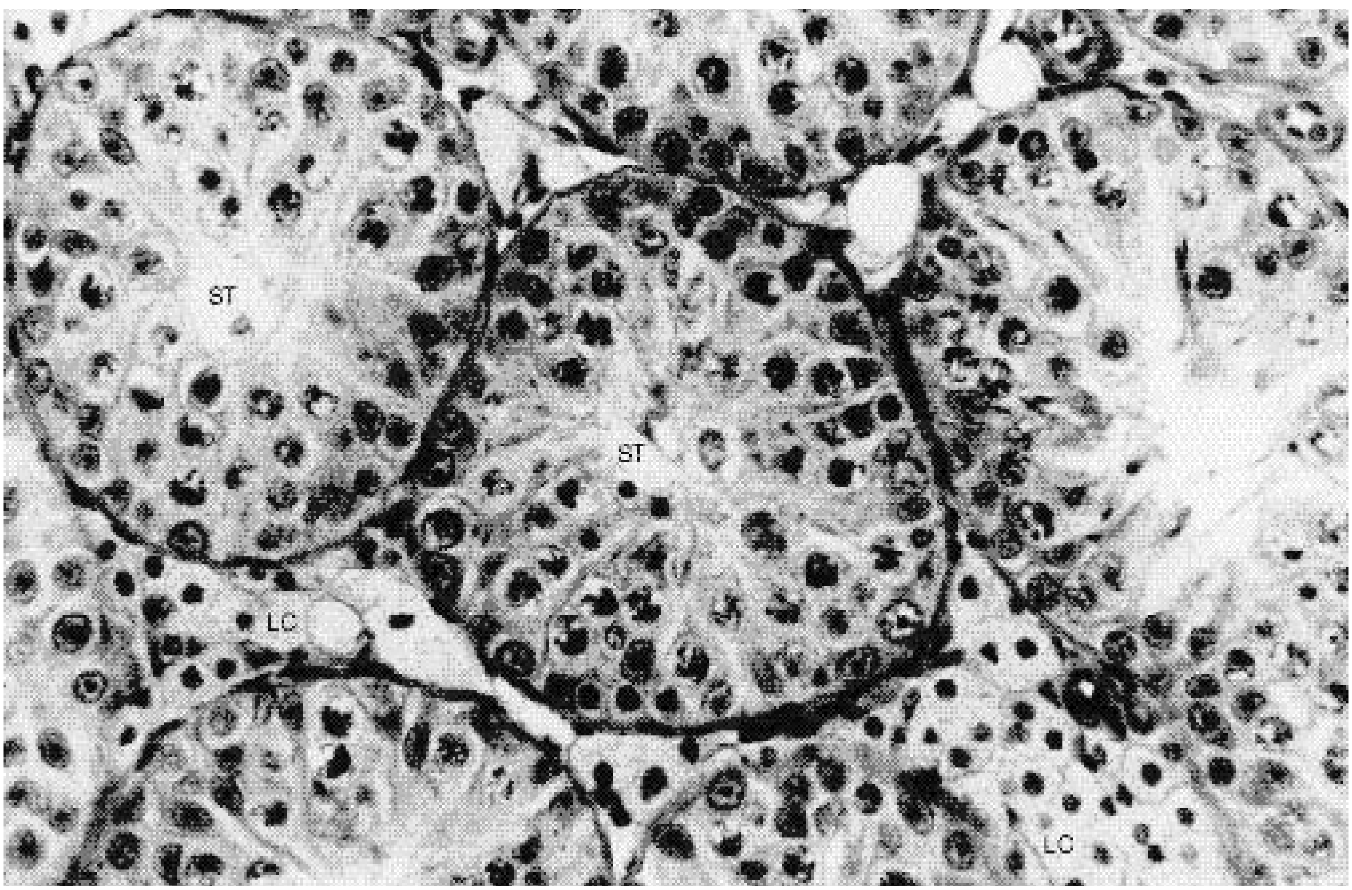

FIGURE 27.2. Testicular histomorphology of adult ocelot showing cross-sections of seminiferous tubules (ST) in intense spermatogenic activity. Note clusters of Leydig cells (LC) in the interstitial space, with cytoplasmic lipid vacuoles. (Hematoxylin and eosin stained; ×400.)

when compared with animals receiving almost all-meat or all–chicken neck diets (Table 27.8). Seasonal influence on testicular function was also investigated and minimal fluctuation was seen, with slight increments of spermatogenic activity found from September to February (spring and summer). These data coincide with keeper records for some Brazilian institutions. During the last 10 years, 16 small-cat litters were recorded at the Curitiba Zoo, southern Brazil, and based on reported average gestation length for tigrinas, margays, ocelots, and jaguarundis, 87% of conceptions have taken place during spring and summer. Data from the Brazilian ocelot studbook for all Brazilian institutions are also similar. Although data from fecal estradiol profiles demonstrated that females also sustain ovarian activity throughout the year,[4] breeding success rate might be enhanced during this time of the year as a result of an increase on both testicular and ovarian function.

Housing conditions, nevertheless, may impair testicular and/or epididymal function by preventing males from displaying behavioral adjustments to environmental factors such as ambient temperature. A male tigrina, maintained under poor husbandry conditions, may have had impaired testicular thermoregulation in response to heat, resulting in decreased sperm quality, as indicated by lower values for percent of normal spermatozoa and sperm motility in the ejaculate (Figure 27.3). Several other physical and/or social stressful conditions in captivity can compromise reproduction. Preliminary data on noninvasive monitoring of adrenal and testicular activity have shown that small-sized cats maintained under similar environmental and husbandry conditions may respond differently to various stress factors. When comparing fecal androgen and corticoid metabolite excretion rate in tigrinas, margays, and ocelots, we have found that tigrinas and margays seem to be more sensitive to captive conditions.[5]

SPERM CRYOPRESERVATION

Cryopreservation of gametes has a tremendous potential as an auxiliary tool in species conservation. However, even closely related species within a taxon may display marked physiological differences in reproductive mechanisms. Sperm viability and function should be evaluated for each species individually to assess similarities and differences among species. Recent studies evaluated comparatively the impact of semen cryopreservation on acrosome integrity, sperm motility, and sperm longevity in the ocelot, margay, and tigrina. Semen samples obtained by electroejaculation were evaluated for percentage of motile sperm (motility) and sperm forward progressive motility (status; range 0-5) to calculate the sperm motility index (SMI; Table 27.9). Further, samples were frozen and thawed according to previously described protocols.[9] Percentage of cells with intact acrosome (IA) was determined through analysis of smears stained with rose bengal-fast green.[7] After thawing, semen was reevaluated for the same variables. Mean values for SMI and AI of fresh samples did not

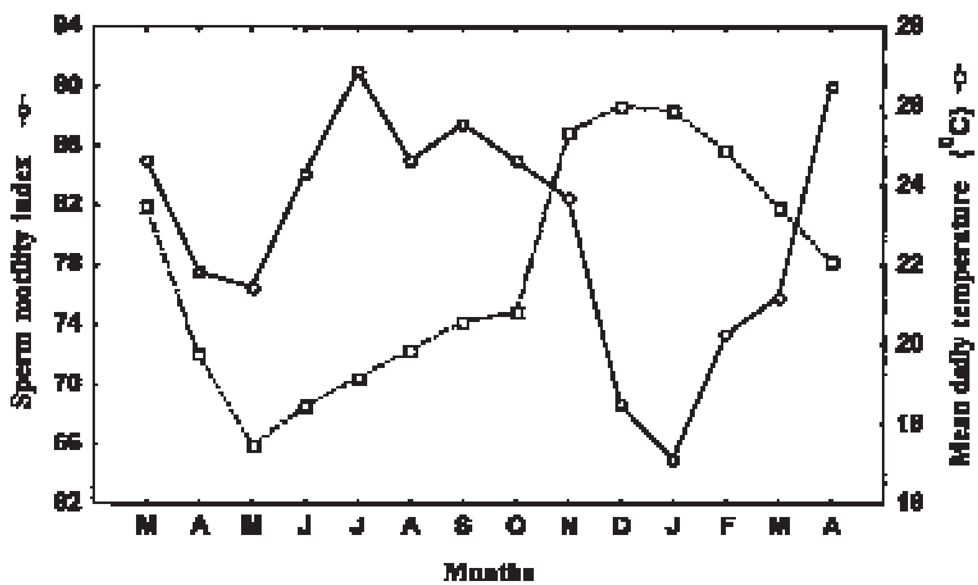

FIGURE 27.3. Sperm motility index in a male tigrina (*L. tigrinus)* at monthly intervals, in relation to mean daily temperature (°C) fluctuations during the same period (March 1995 to April 1996). Sperm motility index: [% motility + (20 × forward progressive motility)]/2.

differ between species, nor did values for SMI obtained after thawing. However, the average of post-thaw IA in the ocelot was significantly lower than in the tigrina and in the margay. Differences within species between values before and after thawing (SMI and IA) were significant ($P < 0.05$), with a reduction of 13-25% in SMI and 42-58% in IA (Table 27.9).

After thawing, in vitro spermatozoa longevity for the same species was also evaluated. Samples were incubated in centrifuge minitubes, containing either Hams'F10 or Hams'F10 with buffer (HEPES) over a 6-hour period on a slide warmer at 37°C, protected from light. Evaluations of the sperm motility index (SMI) and pH were done at 0, 30, 60 minutes, and every hour for 6 hours. Sperm longevity was determined by the time of spermatozoa survival (hours) multiplied by SMI of each sample (see Figure 27.4). Effects of media pH on spermatozoa function were not significant. Spermatozoa from tigrinas had prolonged motility in comparison to ocelots and margays. Data are summarized in Table 27.9.

Although results are preliminary, we conclude that cryopreservation decreased semen quality in all three species. However, significant differences were observed,

TABLE 27.9. Sperm viability and acrosomal status after semen cryopreservation of captive small felids

	Sperm Motility Index (SMI)[a]		Intact Acrosome (%)		Sperm Survival (h)	Sperm Longevity[b]
Species	Fresh	Post-thaw	Fresh	Post-thaw	Post-thaw	Post-thaw
Ocelot *(L. pardalis)*, *n* = 17	81.1 ± 1.6	59.7 ± 2.4	93.7 ± 2.0	39.1 ± 3.0	2.2 ± 0.3	71.2 ± 10.8
Margay *(L. wiedii)*, *n* = 10	74.2 ± 1.6	56.7 ± 2.3	93.7 ± 1.0	47.7 ± 3.1	1.3 ± 0.2	45.2 ± 4.9
Tigrina *(L. tigrinus)*, *n* = 20	76.5 ± 2.4	61.5 ± 2.2	91.9 ± 1.5	53.5 ± 3.8	3.5 ± 0.4	121.4 ± 15.8

Source: Data from reference 6.
[a]SMI = [% motility + (20 × sperm progressive motility)]/2.
[b]Sperm longevity = mean SMI for the total incubation time x mean sperm survival.
Values given are mean ± SEM.

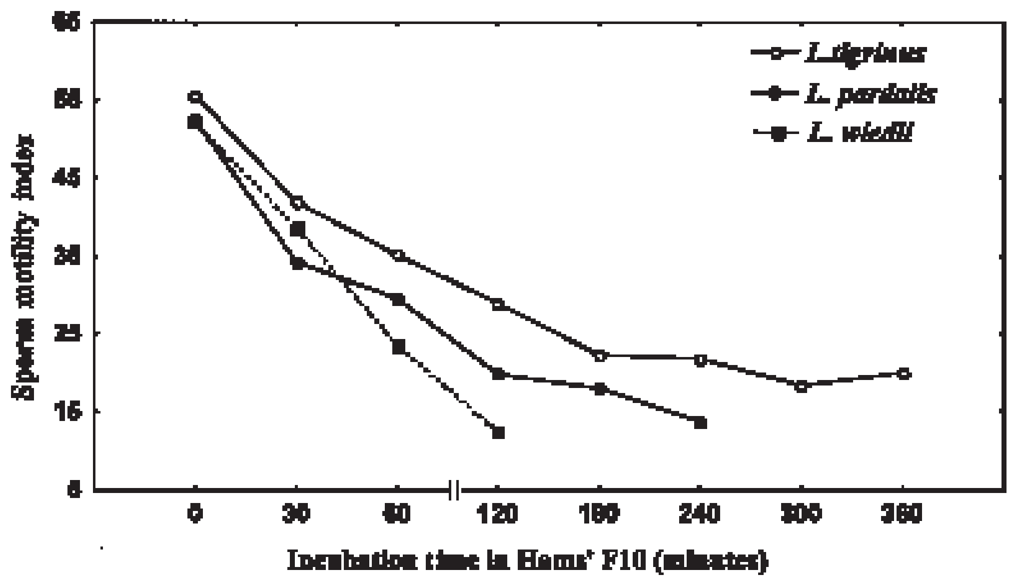

FIGURE 27.4. Longevity of tigrina (*L. tigrinus*) (*n* = 20), ocelot (*L. pardalis*) (*n* = 17), and margay (*L. wiedii*) (*n* = 10) spermatozoa maintained in culture in Hams' F10 medium at 37°C (*n,* number of ejaculates).

with tigrina sperm being slightly more resistant to the method used, for what would suggest a higher in vivo fertilizing potential. Our findings greatly encourage us to evaluate different protocols to be tested in each species until we are able to obtain maximal sperm viability after cryopreservation. Nevertheless, the results obtained in these studies are similar to those reported for other felid species, in which the fecundation capacity of frozen-thawed sperm has been proven after artificial insemination.[9]

REFERENCES

1. Faiçal, S.; Cassaro, K.; and Quilen, P. 1997. Small felid breeding project at São Paulo Zoo. International Zoo Yearbook 35:159–164.
2. Howard, J.G. 1991. Semen collection and analysis in carnivores. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia. W.B. Saunders, pp. 390–399.
3. Mace, G.M.; and Lande, R. 1991. Assessing extinction threats: Toward a reevaluation of IUCN threatened species categories. Conservation Biology 5:148–157.
4. Morais, R.N.; Moreira, N.; Moraes, W.; Mucciolo, R.G.; Lacerda, O.; Gomes, M.L.F.; Swanson, W.F., Graham, L.H.; and Brown, J.L. 1996. Testicular and ovarian function in South American felids assessed by fecal steroids. In Proceedings American Association of Zoo Veterinarians. Puerto Vallarta, Mexico, AAZV, pp. 561–565.
5. Morais, R.N.; Mucciolo, R.G.; Gomes, M.L.F., Lacerda, O.; Moraes, W.; Moreira, N.; Swanson, W.F.; and Brown, J.L. 1997. Adrenal activity assessed by fecal corticoids and male reproductive traits in three South American felid species. In Proceedings American Association of Zoo Veterinarians. Houston, Texas, AAZV, pp. 220–223.
6. Morais, R.N.; Gomes, M.L.F.; Ota, C.C.C.; Lacerda, O.; and Mucciolo, R.G. 1998. Estudo comparativo da longevidade *in vitro* de sêmen de pequenos felinos aps criopreservação [Comparative study of *in vitro* sperm longevity after cryopreservation in small felids] (abstract). In Proceedings Federação de Sociedades de Biologia Experimental. Caxambu, Brazil, FESBE, p. 285.
7. Pope, C.E.; Zhang, Y.Z.; and Dresser, B.L. 1991. A simple staining method for evaluating acrosomal status of cat spermatozoa. Journal of Zoo and Wildlife Medicine 22(1):87–95.
8. Swanson, W.F.; Wildt, D.E.; Cambre, R.C.; Citino, S.B.; Quigley, K.B.; Brousset, D; Morais, R.N.; Moreira, N.; O'Brien, S.J.; and Johnson, W.E. 1995. Reproductive survey of endemic felid species in Latin American zoos: Male reproductive status and implications for conservation. In Proceedings of the American Association of Zoo Veterinarians. Denver, AAZV, pp. 374–380.
9. Swanson, W.F.; Howard, J.G.; Roth, T.L.; Brown, J.L.; Alvarado, T.; Burton, M.; Starnes, D.; and Wildt, D.E. 1996. Responsiveness of ovaries to exogenous gonadotrophins and laparoscopic artificial insemination with frozen thawed spermatozoa in ocelots (*Felis pardalis*). Journal of Reproduction and Fertility 106(1):87–94.

28

Order Carnivora, Family Procyonidae (Raccoons, Kinkajous)

Adriana Sampaio Labate
Adauto Luis Veloso Nunes
Marcelo da Silva Gomes

BIOLOGY

Adriana Sampaio Labate

The Procyonidae family belongs to the order Carnivora. Originally, it was divided into two subfamilies, Procyoninae and Ailurinae, the latter represented by the giant panda (*Ailuropoda melanoleuca*) and the red panda (*Ailurus fulgens*). Some classifiers include these two species with the procyonids, others place them in the Ursidae family.

All the Procyoninae inhabit the New World, distributed in both tropical and Neotropical regions. Four genera are found in South America (*Procyon, Nasua, Potos,* and *Bassaricyon*), with from four to seven species (see Table 28.1).

The procyonids are middle-sized animals with short legs and a thick pelage. They walk on the sole of the foot, with five toes well developed in each foot. Agile hands enable them to be excellent climbers. The molars are large and well adapted to crush food items. The nose is generally pointed and the eyes are almost totally oriented to the front.

The dental formula is I3/3, C1/1, P4/4, M2/2, for a total of 40, except for the kinkajou (*Potos flavus*) which has P3/3.

Some species are totally arboreal, whereas others prefer to search for food on the ground. All of them use trees to nest, rest, or escape from danger. They are omnivorous, generally feeding on invertebrates, small vertebrates, fruits and nectar (kinkajous and olingos).

CAPTIVE MANAGEMENT AND RESTRAINT

Adauto Luis Veloso Nunes

The South American procyonids are easily kept in captivity. In general they are omnivorous, gregarious, and active. They are excellent climbers, with the ability to manipulate objects. Crab-eating raccoons *Procyon cancrivorus* are considered to be nocturnal, but in captivity they are also active during part of the day. They tolerate weather variations and are able to swim. Compatible males and females can be kept in large groups, making an attractive exhibit. Nesting boxes should be provided above the ground level, one for each breeding female, because they and their nestlings require calm and privacy.[2]

TABLE 28.1. Biological data of South American Procyonidae

Scientific Name	Name (English)	Name (Spanish)	Name (Portugese)	Weight (kg)	Identification	Distribution	Habitat/Diet
Procyon cancrivorus	Crab-eating raccoon	Osito lavador, mayato	Mão pelada, guaxinim	2–6	Black mask; long yellowish tail with black tip, carried low	Eastern Costa Rica to eastern Peru and Uruguay. Woody or brushy areas near water	Solitary, nocturnal, arboreal. Diet: Crabs, fish, mollusks, amphibians, insects, fruits, sugarcane, corn
Nasua nasua	Coatimundi	Coati	Coati	3–6	Long nose, white spots around eyes. Brownish pelage above, light below. Long tail with rings, carried vertical	Southern United States to Argentina	Humid forests, woods also found in dry regions in open pastures with stunted vegetation. Diet: fruit, small vertebrates
Potos flavus	Kinkajou	Cuchumbi, martucha	Kinkaju, jupará, macaco de meia noite	1.4–4.6	Long prehensile tail, short legs. Long, extensible tongue	Forests of southern Mexico to central Brazil	Arboreal, nocturnal, pairs or small groups. Diet: primarily fruits, but also insects
Bassaricyon gabbi	Olingo	Olingo	Japará	0.97–1.5	Smaller, but similar to kinkajou. Tail long, but not prehensile	Tropical forests from sea level to 2000 m. Central America through Brazil and Peru	Arboreal, solitary or pairs, nocturnal. Diet: fruits, nectar invertebrates

In the wild, coatis *Nasua nasua* prefer areas with dense vegetation, but they also have been seen in open fields. In captivity, overcrowding may result in aggression and subsequent severe injuries and/or deaths.

The kinkajou *P. flavus* is a shy animal with a strict nocturnal and arboreal behavior. Reverse photoperiod is required for exhibiting these animals. During the daylight they will be in their shelter almost all the time.

Considering the general characteristics of South American procyonids, the enclosure must have branches, perches, and trees which allow them to exhibit their abilities as climbers. The concrete floor must be deep to prevent escape by digging and be covered with soil, sand, vegetation, or leaves.

If the enclosure walls are constructed of mesh wire, it is necessary to have a closed roof. Some zoos in Brazil have experienced success using a smooth metal panel placed on top of the wire fence.

DIET

Table 28.2 lists diets that have been used for procyonids by zoos in Brazil, with good success in maintenance and breeding. In addition to small amounts of fish, frogs, fruits and vegetables provide palatability and variability in the diet.[2] An excessive amount of fat in the diet and lack of activity has frequently led to obesity in procyonids kept in zoos.[3,4] This problem may be managed with more naturalistic enclosures and adjustment in the diet. Vitamins are added empirically by some zoos in order to prevent deficiencies. An improper diet may cause anemia, slow growth, weight loss, osteodystrophy, rickets, paralysis, and poor coat.

TABLE 28.2. South American procyonid diets in use in some zoos in Brazil

	Compounds
Crab-eating raccoon *Procyon cancrivorus*	Dog food, banana, orange, sweet potato, boiled egg[a,d]
	Meat, eggs, corn, mice, day-old chick[b]
	Dog food, papaya, orange, banana, apple, boiled egg, day-old chick[c]
	Dog food, chicken necks, banana, day-old chick, Vionate[d]
Coatimundi *Nasua nasua*	Dog food, banana, orange, sweet potato, boiled egg[a,e]
	Dog food, papaya, orange, banana, apple, boiled egg, day-old chick[c]
	Dog food, chicken necks, banana, day-old chick, Vionate[d]
Kinkajou *Potos flavos*	Beef heart, day-old chick, boiled egg, banana, papaya, apple, Vionate[d]

[a]J.A.B. Bastos, personal communication, 1999.
[b]C. Giacomini, personal communication, 1999.
[c]A.P.O. Cottini, personal communication, 1999.
[d]C. Pessutti, personal communication, 1999.
[e]C.E.P. Saad, personal communication, 1999.

TRANSPORTATION

Plastic carrying cases used for dogs are suitable for moving procyonids. Special concern must be taken to prevent hyperthermia as a result of direct exposure to heat or sunlight. As a general rule, procyonids must be transported individually.

PHYSICAL AND CHEMICAL RESTRAINT OF PROCYONIDS

Restraint of procyonids is necessary for some procedures such as physical examination, vaccination, and medical care. Some procyonids become very tame if they are raised in close contact with humans, and they may be handled easily. However, this situation may change when they reach sexual maturity. If stressed or feeling pain during an examination they may cause serious injuries with their claws and teeth. The handler must be especially careful with coatimundis, which have extremely sharp canines.

Physical Capture

In the wild, procyonids have been captured with traps designed for animals of their weight and length. Once in a trap, they may be transferred to a carrying cage or may be handled with snares, nets, or small squeeze cages. If the animal can be attracted to a bait and becomes well positioned, they may be darted with a blow gun or an air rifle. Young animals may be restrained with leather gloves. Adults are difficult to handle with gloves only, and the initial approach must be made with nets or a squeeze cage.

Chemical Restraint

Chemical capture of the procyonids may be required, because physical restraint may not allow persons to handle adults safely. Many procedures may only be done with the animal under sedation (see Table 28.3).

TABLE 28.3. Protocols with success for South American procyonids

Drugs	(mg/kg)	Effects	Comments	References
Ketamine	20–30 IM	Superficial anesthesia	Induction 3–7 min. Return 45–90 min. Very poor muscle relaxation	1, 2, 3
Ketamine Xylazine	10 IM 2 IM	Anesthesia and analgesia	Induction 3–5 min. Anesthesia lasts 15–20 min. Return 60–90 min. Some muscle relaxation	6
Ketamine Xylazine Atropine	5–15 IM 1–2 IM 0.04 IM	Anesthesia and analgesia	Induction 3–5 min. Anesthesia lasts 15–20 min. Return 60–90 min. Some muscle relaxation	5[a]
Telazol (Tiletamine + zolazepam)	10 IM	Chemical capture and minor surgery	Induction 3–10 min. Sedation/ anesthesia lasts 20–60 min. Reflex present	[b] 2
Telazol (Tiletamine + zolazepam)	0.7–25 IM	Chemical capture and minor surgery	Induction 3–10 min. Sedation/anesthesia lasts 20–60 min. Reflex present	[c] 5

[a]M. P. Pinho, personal communication, 1999.
[b]A.P.O. Cottini, personal communication, 1999.
[c]C. Giacomini, personal communication, 1999.

REFERENCES

1. Beck, C.C. 1972. Chemical restraint of exotic species. Journal of Zoo Animal Medicine 3:3–66.
2. Boever, W.J.; Holden, J.; and Kane, K.K. 1977. Use of telazol (CI-744) for chemical restraint in wild and exotic carnivores. VM/SAC 72: 1722–1725.
3. Greg, D.A; and Olson, L.D. 1975. The use of ketamine hydrochloride as an anesthesia for raccoons. Journal of Wildlife Diseases 11:335–337.
4. IBAMA. 1989. Instrução normativa no. 001/89-P de 19 de Outubro de 1989. In Legislação sobre Zoológicos. Brasilia, IBAMA, pp. 9–27.
5. Mehren, K.G. 1986. Procyonidae. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 816–820.
6. Nielsen, L. 1999. Chemical Immobilization of Wild and Exotic Animals. Ames, Iowa, Iowa State University Press.
7. Ramsden, R.O.; Coppin, P.F.; and Johnston, D.H. 1976. clinical observations on the use of ketamine hydrochloride in wild carnivores. Journal of Wildlife Diseases 12:221–225.
8. Wallack, J.D. 1970. Nutritional diseases of exotic animals. Journal of the American Veterinary Medical Association 157:583–599.
9. Wallack, J.D. 1971. Exotic diets are not for exotic pets. Paper presented at the Gaines Small Animal Nutritional Workshop, University of Illinois.
10. Wallack, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals. Philadelphia, W.B. Saunders.

MEDICINE

Marcelo da Silva Gomes

CLINICAL EXAM

The clinical examination of Neotropical procyonids is similar to those of other wild carnivores or domestic dogs. The active and sometimes aggressive nature of these animals adds to the gravity of lesions resulting from bites, notably from adult coatis (*N. nasua*). Chemical restraint is usually required. When examining the skin, the handler should remember that the kinkajou (*P. flavus*) has two natural hairless areas on the face, frequently mistaken for *Sarcoptes* sp. infestation. The quantity and coloration of waxy discharge in the ear and the condition of the mucous membranes may show the presence of infection, or more frequently, infestation by *Otodectis* sp.

Accumulation of tartar on the teeth is common in captive procyonids, which may be related to a diet consisting of food items that are too soft. The accumulation of tartar occurs primarily on the superior molars and, later, on the inferiors. Fractures of the canines are common in captive specimens. A root canal or extraction of the affected tooth may be necessary.

The best results from auscultation are obtained with the subject seated on the examination table and the stethoscope positioned between the third and the sixth intercostal space on the left. Under restraint with ketamine, 10 mg/kg, and xylazine, 1 mg/kg, the authors have found the following parameters:

Time	Respiratory Rate/MinR.	Heart Rate/Min	Body Temperature (°C)
10 minutes	18–36	84–100	36.2–39.2
20 minutes	28–48	76–120	35.7–37.5
30 minutes	24–60	88–120	36.7–39.2

Depending on the restraint method used, the animal's nature, environmental temperature, and other characteristics, wide variation in these and other physiological parameters may be seen.

BIOLOGICAL SAMPLE COLLECTION

Biological samples may be collected in the same manner as in dogs.

The collection of blood samples is relatively easy, but requires firm physical restraint or chemical immobilization in adult animals. Venipuncture of the jugular, lateral saphenous, or cephalic vein may be used. The tendency for obesity in the coati and the thickness of the skin of the neck makes jugular venipuncture less desirable in that species. The majority of laboratories are able to perform hematologic analysis with 1.5–2.0 mL of blood. Normal parameters vary according to different authors, but a study with captive animals in the state of São Paulo, Brazil, has resulted in the values shown in Table 28.4 (unpublished data).

Cytocentesis, as in a dog, is commonly employed to collect urine samples.

INFECTIOUS AND PARASITIC DISEASES

Procyonids are affected by the majority of infectious and parasitic pathologies that attack domestic carnivores. This group is susceptible to rabies, canine distemper, infectious hepatitis, canine parvovirus, and feline panleukopenia. See other chapters in this book for disease descriptions.

Protozoa

Leishmaniasis, *Leishmania brasiliensis*, has been found in Panama in coatis without exhibiting cutaneous lesions. In the olingo, *Passaricyon gabbii* infection by dermatotopic forms of *Leishmania* was evidenced by a lack of pigmentation and incrustration generally in the internal face of the ear, and in the kinkajou, by lack of pigmentation in the nose and ears. There have been no reports of visceral leishmaniosis in this group.

Other protozoans such as *Trypanosoma evansi* cause significant hematologic alterations. *Eimeria* spp. and *Isospora* spp. have also been described in these animals.

Endoparasites

A variety of species of nematodes, cestodes, and trematodes have been found in animals that may or may not exhibit clinical signs. *Ancylostoma caninum* is a common parasite, and other parasites, such as *Dirofilaria imitis* in coatis and crab-eating raccoons and *Baylisascaris procyonis* in kinkajou have been reported. *Toxocara canis* has been found in captive coatis and crab-eating raccoons that had possibly had contact with domestic dogs. *Brugia guyanensis* has also been described in coatis and other wild animals in Peru. *Dioctophyma renale* has been described in *Nasua nasua,* although it is not found frequently.

TABLE 28.4. Hematologic parameters for South American Procyonidae

Animal	RBC (10^3 mm^3)	Hb (g/dL)	PCV (%)	MCV	MCH	MCHC
P. flavus	6.0(±0.8)	13.0(±0.9)	39.0(±3.0)	64.0(±10.0)	20.0(±1.7)	33.0(±3.0)
N. nasua	5.0(±0.9)	10.0(±1.8)	34.0(±4.2)	62.0(±7.8)	19.0(±2.0)	31.0(±3.0)
P. cancrivorus	5.0(±1.0)	11.0(±1.6)	35.0(±3.0)	64.0(±6.3)	20.0(±1.3)	31.2(±2.8)

RBC, red blood cells; Hb, hemoglobin; PCV, packed cell volume; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration.

Ectoparasites

Flea infestations are frequent in these animals, and *Ctenocephalides felis* is the usual species. In some cases, heavy parasite loads may cause anemia and death. Topical fipronil, together with an improved environment, tend to diminish infestations. In severe cases, administration of fluids, iron, and vitamin B complex may help to correct anemia.

Ticks, such as *Amblyoma collebs* in captive coatis and *Amblyoma cajamense, Amblyoma parvum,* and *Amblyoma ovale* in wild coatis from Mato Grosso, Brazil, may cause problems. Treatments are the same as used in domestic carnivores. These arthropods are potential vectors of *Ehrlichia* spp. and *Babesia* spp., although there have been no reported cases in South American procyonids, in contrast to North America.

NEOPLASIA

Reports of tumors are rare. A cutaneous hemangiosarcoma in an elderly *Procyon cancrivorus* was removed from the region of the left fifth intercostal space. Two years later, at necropsy, there was no evidence of metastasis. Lipomas have been found in elderly *N. nasua*.

TRAUMA

Interspecific and intraspecific aggression is common in procyonids in captivity, resulting in numerous lacerations. Aggression to neonates is also common; consequently a pregnant female should be separated from the group before delivery.

29
Order Carnivora, Family Mustelidae

BIOLOGY AND MEDICINE

Tatiana Lucena Pimentel
Marcelo Lima Reis
Ana Silvia M. Passerino

BIOLOGY

Taxonomy

It is believed that the family Mustelidae has a northern origin, since mustelids first appear in the Oligocene in North America and Europe. Research shows that they first arrived in South America in the late Pliocene and rapidly occupied the small-carnivore ecological niches. They are currently found on all continents except Antarctica and Australia.[7,28]

The family is divided into 25 genera and 65 species worldwide. Mustelids are further divided into six subfamilies, three of which occur in South America (Table 29.1). Seven genera and 14 species occur in South America.[36]

Identification

These small- to medium-sized carnivores are characterized by an elongated and slender body with relativity short legs; a broad head, small relative to body size; small rounded ears and eyes, tails shorter than the head and body, convenient for using burrows and holes. They are digitigrade or plantigrade, with five toes on all feet. The claws are compressed, curved, and nonretractile. The digits are usually webbed. Males have a baculum and are about one-fourth larger than females. The pelage is uniformly colored, spotted, or striped.

The dental formula is variable, and for most South American forms is I3/3, C1/1, P3/3, M1/2, showing a reduction in the premolar number, except for the otters (P4/3). Most species have large anal glands that produce strong-smelling musk used for territory marking or as defense against predators. [7,9,28,23,24]

Natural History

The family includes terrestrial, arboreal, and semiaquatic forms, often highly specialized (see Figure 29.1). They move quickly and with precise movements. The back is humped when the animal is standing. They may be nocturnal or diurnal and often shelter in crevices, burrows, and trees. The sense of smell and hearing are acute, but most species do not seem to see particularly well. They are predominantly carnivorous, and the group has a varied diet, which includes vertebrates (fish, amphibians, reptiles, birds, and small mammals) and invertebrates (crustaceans, mollusks, insects). They are voracious predators; some of them have an extremely powerful bite for their size and can

TABLE 29.1. South American mustelids, Order Carnivora, Family Mustelidae

Scientific Name	Common Name	Distributrion	Size	Habitat
Mustelinae				
Mustela felipei	Colombian weasel	Cordillera central Colombia and northern Ecuador	HB = length 21.7–22.0 cm T = 10.4–11.1 cm	Riparian areas
Mustela africana	Tropical weasel	East of the Andes in the lowland Amazon basin of Brazil, Ecuador, and Peru	HB = 24.0–38.0 cm, T = 16.0–23.0 cm W = 300g	Humid riparian forest
Mustela frenata	Long-tailed weasel	North, Central and South America from southern British Columbia to Guyana and Bolivia up to 4000 m	HB = 21.5–32.0 cm T = 11.5–20.7 cm W = 85–365 g	Prefers open, brushy, or grassy areas near water; tolerates a wide range of elevation
Eira barbara	Tayra	Southern Mexico to Paraguay and northern Argentina below 2400 m	HB = 52.6–72.2 cm T = 29.0–47.0 cm W = 4–7 kg	Prefers forest, but tolerates deciduous forest, tropical evergreen forest, gardens, farms
Galictis cuja	Little grison	East central Brazil, central Chile, southern Peru, Bolivia, Uruguay, Paraguay, and Argentina	HB = 37–53cm T = 12.5–19.0 cm W = 0.7-1.3 kg	Frequently found near open water with little surrounding vegetation or near forest margins
Galictis vittata	Greater grison	Central and South America below 1200 m	HB = 47.5–71.2cm T = 13.5–19.5 cm W = 1.4–3.3 kg	Found in deciduous forests, tropical rain forests and savannas, near rivers and streams
Lyncodon patagonicus	Patagonian weasel	Argentina and southern Chile	HB = 28.0–40.0 cm W = 2.3 kg	Pampas habitats—dry shrublands, as high as 2000 m

Conepatinae				
Conepatus semistriatus	Hog-nosed skunk	Southern Mexico to coastal Peru, Ecuador, Colombia, Venezuela and central-eastern Brazil	HB = 30–50 cm W = 1.4–4.5 kg	Savannas, scrub, secondary forest near clearings, gardens, pastures, and cultivated areas
Conepatus chinga	Patagonian hog-nosed skunk	Central and southern Peru, Bolivia, Chile, northwestern Argentina, southern Brazil and Paraguay	HB = 22.0–32.5 cm T = 18.0–42.0 cm W = 1–3 kg	Similar
Conepatus humboldti	Hog-nosed skunk	Chile, northeastern Argentina, and Paraguay, south to the Straits of Magellan	HB = 12.3–37.0 cm T = 11.0–20.0 cm W = 1–2.7 kg	Similar
Lutrinae				
Lontra longicaudis	River otter	Central and South America from northwestern Mexico to Uruguay	HB = 36.0–82.0 cm T = 30.0–57.0 cm W = 5-15 kg	Always near water, whether lagoons, rivers, streams, swamps, or other similar habitats
Lontra provocax	River otter	Central and southern Chile and adjacent Argentina	HB = 57.0–61.0 cm T = 35.0–40.0 cm W = 5–10 kg	Marine and fresh water rivers , lakes, and estuaries Marine habitats *Lontra felina*
Lontra felina	River otter	Pacific Coast from Peru to Tierra del Fuego	HB = 83.3–114.9cm W = 3.2–5.8 kg	Lowland forest rivers and lakes of many types
Pteronura brasiliensis	Giant otter	In the major river systems of South America, east of the Andes	HB = 86.4–140.0 cm T = 33.0–100.0 cm W = 24–35kg	

HB, head and body weight; T, tail length; W, whole body weight.

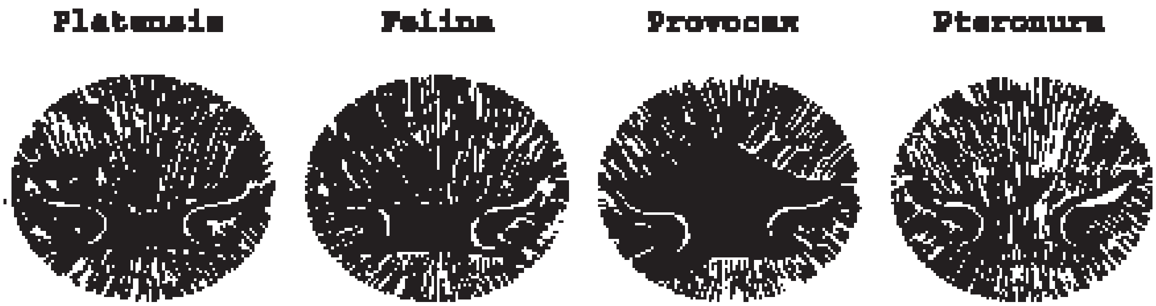

FIGURE 29.1. Drawings of South American mustelids' nostrils.

easily kill prey much larger than themselves.[9,28,24] The giant otters (*P. brasiliensis*) are strictly piscivorous.

DISEASES AND THERAPEUTICS

BOTULISM Most mustelids are susceptible to *Clostridium* botulinum type C toxin (to a lesser extent types A, B, and E). Generally fatal, the disease is caused by consumption of contaminated or uncooked meat.[29] Diagnosis is made by observation of the clinical signs and a history of peracute disease. Treatment should include removal of the contaminated food, injection of antiserum, and electrolyte fluid therapy.[35] Animals not on a commercially prepared diet should be vaccinated annually.[26]

LEPTOSPIROSIS Leptospirosis is a zoonosis caused by *Leptospira interrogans*. This organism can survive for long periods in surface water. Characteristic clinical signs may be muscle spasms, motor incoordination, fever, catarrhal discharge, hemoglobinuria, icterus, stomatitis, vomiting, progressive weight loss, and death.[10] The symptoms resemble a toxemia. It has been reported in giant otter.[21] It has been treated at Fundaçáo Polo Ecologico de Brasília (Brasília Ecological Pole Foundation; FunPEB) with dihydrostreptomycin 15 mg/kg plus penicillin benzathine 40,000 IU once a day for 10 days.

LISTERIOSIS Listeriosis, or circling disease, is not a commonly seen infection. The characteristic clinical signs include abortion, perinatal mortality, septicemia, and ataxia.[29] Treatment includes the use of parenteral broad spectrum antibiotics.[35]

SALMONELLOSIS This disease is characterized clinically by one or more of three major syndromes: septicemia, acute enteritis, chronic enteritis. Although it can be treated with antibiotics (furazolidone or chloramphenicol), it is often difficult to rid the animal of the organism. *Salmonella* species have been isolated from the feces of clinically normal animals and does not always cause disease (Petrini, personal communication).

Other infectious diseases reported in mustelids include tuberculosis, tularemia, pasteurellosis, canine distemper, parvoviral infection (feline panleukopenia, canine parvovirus, mink enteritis), pneumonia, otitis, and mycotic diseases.

Parasitic Diseases

The parasites of mustelid follow the same general pattern as other carnivores. A few of the parasites that have been reported to produce clinical disease include kidney worms *Dictophyma renale* and *Gnathostoma miyazakii,* lung flukes, tapeworms *Diphyllobothrildae, Giardia* spp., coccida, guinea worms *Dracunlus* spp., and heartworms, *Dirofilaria immitus*. Management and therapy is the same as for other carnivores.

Noninfectious Diseases

Noninfectious diseases include gastric ulcers, bite wounds, footpad abrasions, stress, dental problems (tooth decay, tartar accumulation, gingivitis, fractures).

CHEMICAL RESTRAINT, ANESTHESIA, AND SURGERY

To immobilize mustelids, an intramuscular injection is given in the upper hind leg using a plastic hand-held syringe while the animal is physically restrained with a snare pole, a net, a squeeze cage, or a restraint box. A variety of chemical agents are suitable. Ketamine hydrochloride may be used alone in mustelids at a dose of 20–40 mg/kg, intramuscularly. Dosages of up to 100 mg/kg have been used for major surgery.[35] At FunPEB the author combined ketamine (20 mg/kg) with xylazine, and 2 mg/kg is used to immobilize mustelids. Analgesia and anesthetic levels are excellent. At Hagenbeck Tierpark a 5-month-old male was immobilized

FIGURE 29.2. Blood sample being drawn from a giant otter. (FunPEB–Brasilia Zoo) (Photograph by Carlos Abs Bianchi.)

with ketamine (5.5 mg/Kg) and xylazine (1.1 mg/Kg). The animal slept after 6 minutes (Dr. M. Flugger, personal communication, 1999).

Tiletamine/zolazepam (Telazol, Zoletil) is used at a dosage of 1.5–10 mg/kg. Animals sedated with the higher doses may require hours to recover. Salivation and emesis are common side effects.[35] This works well for short physical examination. For longer procedures inhalation anesthesia with isoflurane is ideal. A precision vaporizer should be used attached to a mask or an endotracheal tube. Caution must be exercised because otters go into respiratory arrest easily. Other combinations that have been used include medetomidine/ketamine followed by reversal using atipamezole;[32] and ketamine 22 mg/kg with diazepam 0.4 mg/kg. The addition of diazepam decreases the undesirable side effects of ketamine anesthesia.[8]

The cardiac rate of an anesthetized male otter varied between 156 and 228 beats per minute (bpm), and the breathing rate varied between 14 and 22 bpm. The rectal temperature varied between 38.5 and 41.5°C (average of 39.37°C or 102.9°F).

Blood Sample

Venipuncture of the femoral or jugular vein is used to obtain blood samples in mustelids. Twelve milliliter (6 mL for pups) may be withdrawn from sea otters using a 19-gauge, 1-inch needle. Extra serum should be frozen because it is invaluable to researchers. If the animal is anesthetized, the jugular vein may be used. Place the animal in dorsal recumbency, pulling its forearm down firmly caudally and its head stretched cranially and slightly diagonally opposite the direction of the forearm. A depression should appear above the clavicle. The needle should be inserted perpendicular to the animal's body into the clavicular depression (Figure 29.2). If blood doesn't flow into the syringe, retract the needle slowly until blood does flow.

Hematological and chemical values for Amazonian otters are found in Table 29.2 and Table 29.3. Those values are within the range of various species of marine cetaceans and are similar to those described for sea otters.[5] Three young skunks *Galicis cuja furax* were anesthetized with ketamine for blood collection. The results are listed in Tables 29.4–29.6.

Surgery

In the Hoover[16] study, surgical technique was used to place intra-abdominal radiotelemetry devices in American river otters (*Lutra canadensis*). Food was withheld overnight (at least 12 hours), but the otters were allowed water. The immobilized otters were intubated for inhalation anesthesia and prepared for surgery. Each otter was given 15,000 IU/kg procaine penicillin G and benzathine penicillin G (Benza-Pen; Beecham Laboratories Division of Beecham Inc., Bristol, Tennessee, USA) by intramuscular injection immediately after surgery. For two otters with vaginal discharges, the treatment was repeated every 48 hours until they were released.

Urinalysis findings in the Hoover study included protein in 38% (trace to ≥ 300 mg/dL), bilirubin in 75% (1+ to 3+), blood in 100% (trace to 3+), and urobilinogen in 100% (0.1) of the 12 specimens tested. Glucose and ketones were not detected in any specimens. The findings on microscopic examination for 6 of 12 specimens that were adequate (≥ 5 mL) included leukocytes

TABLE 29.2. Hematological values for two species of Amazonian otters

	P. brasiliensis Animal No. 1	*L. longicaudis* Animal No. 2	*L. longicaudis* Animal No. 3	*L. longicaudis* Animal No. 4
RBC ($\times 10^6$ cells/μL)	5.5	6.2	5.1	5.1
Hb (g/dL)	17.3	18.1	15	17.3
PCV (%)	60	56	51	55
MCV (fl)	109.1	90.3	100	107.8
MCHC (g/dL)	28.8	32.3	29.4	31.4
MCH (pg)	31.5	29.2	29.4	33.9
WBC (cell/μL)	5200	5,100	10,000	6,700
BAND (cell/μL)	0	51	0	0
(%)	0	1	0	0
Neutrophils (cell/μL)	3,588	3,366	6,100	3,350
(%)	69	66	61	50
Lymphocytes (cell/μL)	1,248	867	2,300	2,345
(%)	24	17	23	35
Monocytes (cell/μL)	208	357	500	260
(%)	4	7	5	4
Eosinophils (cell/μL)	156	459	1,100	737
(%)	3	9	11	11

Source: See reference 5.
RBC, red blood cells; Hb, hemoglobin; PCV, packed cell volume; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; MCH, mean corpuscular hemoglobin; WBC, white blood cells.

TABLE 29.3. Clinical chemistry values for two species of Amazonian otters

	P. brasiliensis Animal No. 1	*L. longicaudis* Animal No. 2	*L. longicaudis* Animal No. 3	*L. longicaudis* Animal No. 4
Total protein (g/dL)	6.6	6.8	6.6	—
Albumin (g/dL)	2.6	3.2	3.4	—
Globulin (g/dL)	4	3.6	3.2	—
BUN (mg/dL)	39.2	31.6	32.5	41.3
Creatinine (mg/dL)	1.3	0.9	1	1.4
Glucose (mg/dL)	250	132	140	243
Bilirubin total (mg/dL)	0.5	—	0.5	—
Cholesterol (mg/dL)	207	248	207	270
AST (IU)	8.7	16.3	21.2	8.7
ALT (IU)	11.6	18.3	16.4	21.7
Uric acid (mg/dL)	—	2.7	1.9	1.3

Source: See reference 5.
BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase.

in 67%, erythrocytes in 83%, triple phosphate or amorphous urate crystals in 50%, squamous epithelial cells in 100%, and bacteria in 50% of the specimens.[16]

Parasitology

Parasitologic examinations of fecal samples revealed *Isospora* sp. (two otters), *Capillaria* sp. (four otters), and an unidentified strongyle (three otters). *Strongyloides lutrae* was recovered on culture of feces from one otter. Circulating microfilaria were seen in blood samples of nine otters. One nematode, later identified as *Dirofilaria lutrae,* was recovered from subcutis along the ventral midline incision of one otter during surgery and from two others at necropsy.[15]

REPRODUCTION

Table 29.7 provides data on the gestation periods and litter sizes for 14 South American mustelids.

TABLE 29.4. Skunk erythorogram

	RBCs	PCV	Hb	MCV	MCHC
F1 juvenile	5	38	12.48	76	32.84
F2 juvenile	4.5	39	13.18	86.66	33.79
Average	4.8	38.5	12.8	81.3	33.3
Standard deviation	0.35	0.71	0.49	7.54	0.67
F4 male adult	8.5	55	18.86	64.7	34.29
F5 female adult	6.5	45	14.7	69.23	32.66
F6 female adult	4.5	37	11.7	82.22	31.62
Ffemale 1	4.9	54	17.14	110.2	31.74
Ffemale 2	7	45	14.5	64.28	32.22
Average	6.28	47.2	15.38	78.13	32.51
Standard deviation	1.63	7.43	2.74	19.34	1.08

Source: See reference 12.
RBCS, red blood cells; PCV, packed cell volume; Hb, hemoglobin; MCV, mean corpuscular volume; MCHC, mean corpuscular volume concentration.

TABLE 29.5. Skunk leucogram

	WBC	Seg %	Band %	Lymphocyte %	Eosinophil %	Monocyte %	Basophil %	STP (g/dL)
01 juve.	9,200	46	1	39	8	3	0	6.2
02 juve.	8,900	47	0	46	6	1	0	8
Average	9,050	47	1	43	7	2	0	7
Standard deviation	212.13	0.71	0.71	4.95	1.41	1.41	—	1.27
0.4 M. A.	5,500	74	0	21	4	0	1	7
0.5 F. A.	9,600	76	2	18	3	1	0	6.6
0.6 F. A.	6,000	66	3	27	3	0	0	7
F.C. 03	5,000	61	0	18	20	1	1	7.2
Average	6,525	69	1	21	8	1	1	7
Standard deviation	2090.26	6.99	1.50	4.24	8.35	0.58	0.58	0.25

Source: See reference 26.
WCB, white blood cells; Seg, segmented neutrophils; Band, band neutrophils; STP, serum total protein; 01 and 02, juvenile animals 01 and 02; 04 M.A., adult male 04; 05 and 06 F.A., adult females 05 and 06; F.C. 03, female cub 03.

TABLE 29.6. Average values of biochemical parameters on 4 skunks

	Animal No. 1	Animal No. 2	Animal No. 3	Animal No. 4
Glucose (mg/dL)	115	125	116	105
Urea (mg/dL)	26	23	24	29
GGT (IU/L)	38	40	40	39
CK (IU/L)	118.01	84.24	60.37	76.28
LDH (IU/L)	191.80	168.40	162.85	156.10
AST (IU/L)	84.45	73.31	63.97	77.32

Source: See reference 26.
GGT, gamma-glutamyl transferase; CK, creatine kinase; LD, lactase dihydrogenase; AST, aspartate aminotransferase.

TABLE 29.7. South American mustelids, order Carnivora, family Mustelidae, reproductive data

Scientific Name	Common Name	Gestation (Days)	Litter Size
Mustelinae			
Mustela felipei	Colombian weasel	Approximately 30	3–9
Mustela africana	Tropical weasel	Approximately 30	3–9
Mustela frenata	Long-tailed weasel	23–34	3–9
Eira barbara	Tayra	63–70	2–4
Galictis cuja	Little grison	40–60	4–5
Galictis vittata	Greater grison	40–60	1-5
Lyncodon patagonicus	Patagonian weasel	40–60 (probably like Galictis)	
Conepatrinae			
Conepatus semistriatus	Hog-nosed skunk	Approximately 60	2–5
Conepatus chinga	Hog-nosed skunk	Approximately 60	2–5
Conepatus humboldti	Patagonian hog-nosed skunk	Approximately 60	2–5
Lutinae			
Lontra longicaudis	Neotropical river otter	56–70	1–5
Lontra provocax	Southern river otter	56–70	1–4
Lontra felina	Marine river otter	60–65	2–5
Pteronura brasiliensis	Giant otter	64–71	1–6

Source: references 1–4, 6, 7, 9, 11, 16–20, 22–25, 28, 30, 31, 34.

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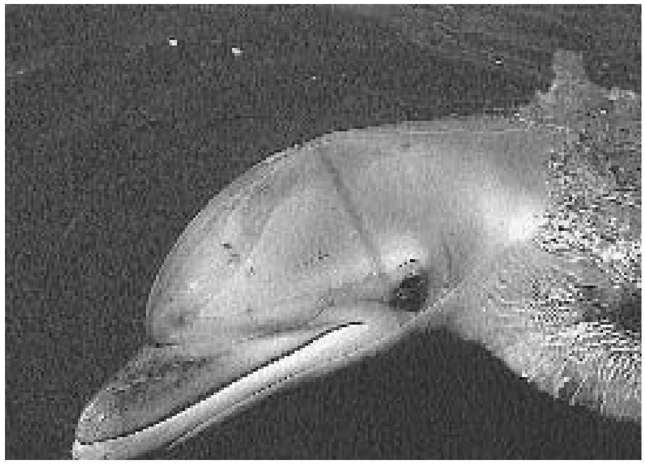
REFERENCES

1. Bardier, G. 1992. Uso de recursos y caracteristicas del hábitat del "lobito de rio" *Lutra longicaudis* (Olfers, 1818) (Mammalia, Carnivora) en el Arroio Sauce, se de Uruguay. Boletín de la Sociedad Zoológica del Uruguay 7:59–60.
2. Bertonatti, C.; and Parera, A. 1994. Lobito de rio. In Revista Vida Silvestre, Nuestro Libro Rojo, ed., Fundación Vida Silvestre Argentina, Buenos Aires, Ficha No. 34.
3. Blacher, C. 1987. Ocorrência e preservaço de *Lutra longicaudis* (Mammalia: Mustelidae) no litoral de Santa Catarina. Boletim da Fundaço Brasileira para Conservaço da Natureza 22: 105–117.
4. Cabello, C.C. 1978. La nutria de mar *L. felina* en la isla de Chiloé. In N. Duplaix, ed., Otters: Proceedings of the First Working Meeting of the Otter Specialist Group, Morges, International Union for Conservation of Nature and Natural Research, pp. 108–119.
5. Colares, E.P.; and Best, R.C. 1991. Blood parameters of amazon otters (*Lutra longicaudis, Pteronura brasiliensis*) (Carnivora, Mustelidae). Comparative Biochemistry and Physiology 99(4):513–515.
6. Duplaix-Hall, N. 1975. River otters in captivity: A review. In R.D. Martin, ed., Breeding Endangered Species in Captivity. New York, Academic Press, pp. 311–327.
7. Eisenberg, J.E. 1989. Mammals of the Neotropics, Vol. 1. The Northern Neotropics: Panama, Colombia, Venezuela, Guyana, Suriname, French Guiana. Chicago, University of Chicago Press.
8. Elmore, R.G.; Hardin, D.K.; Balke, J.M.E.; Youngquist, R.S.; and Erickson, D.W. 1985. Analysing the effects of diazepam used in combination with ketamine. Veterinary Medicine 5:55–57.
9. Emmons, L H.. 1997. Neotropical Rainforest Mammals. A Field Guide, 2nd Ed. Chicago, University of Chicago Press.
10. Farias, T.M.; da Silva, L.H.R.; and Pimentel, T.L. 1999. Occurrence of leptospirosis in giant otters at Brasilia Pole Ecological Foundation (FunPEB) Brazil. In 23rd Brazilian Congress of Zoos in Goiânia (GO) Brazil. Goiânia, SZB, p. 10.
11. Fuller, T.K.; Johnson, W.E.; Franklin, W.I.; and Johnson, K.A.. 1987. Notes on the Patagonian hog-nosed skunk (*Conepatus humboldti*) in southern Chile. Journal of Mammalogy 68(4):864–867.
12. Locatelli-Diltrich, R.; Schmidt, E.M.S.; Passerino, A.S.M; Andri, M.; Jardim, M.G.; and Kerazzoll, G. 1999. Hematological parameters and total plasmatic

protein of junvenile and adult skunks (*galictis cuja furax*). Anais do 10th Congresso, Basileiro de Pequeno Animals [Proceedings of the 10th Small Animal Brazilian Meeting in Aguas de Lindoia, State of São Paulo], p. 129
13. Hagenbeck, C.; and Wünnemann, K. 1992. Breeding the giant otter *Pteronura brasiliensis* at Carl Hagenbeck's Tierpark. International Zoo Yearbook 31:240–245.
14. Hall, E.R. 1951. American Weasel. Lawrence, Kansas, Museum of Natural History, University of Kansas.
15. Harris, C.J. 1968. Otters: A Study of the Recent Lutrinae. London, Weinfield and Nicolson.
16. Hoover, J.P.; Root, C.R.; and Zimmer, M.A. 1984. Clinical evaluation of American river otters in a reintroduction study. Journal of the American Veterinary Medical Association 185(11):1321–1326.
17. Kaufmann, J.H.; and Kaufmann, A. 1965. Observations of the behavior of tayras and grisons. Z Säugetierkunde 30:146–155.
18. Larivière, S. 1998. *Lontra felina*. In L. Carraway, E. Anderson, and K.F. Kooperman, eds., Mammalian Species. American Society of Mammalogists, 575:1–5.
19. Larivière, S. 1999. *Lontra longicaudis*. In L. Carraway, E. Anderson, and K.F. Kooperman, eds., Mammalian Species. American Society of Mammalogists, 609:1–5.
20. Larivière, S. 1999. *Lontra provocax*. In L. Carraway, E. Anderson, and K.F. Kooperman, eds., Mammalian Species. American Society of Mammalogists, 610:1–4.
21. Lewis, J.C.M. 1995. Veterinary considerations. In J. Partridge and M. Jordan, eds., Husbandry Handbook for Mustelids. Bristol, The Association of British Wild Animal Keepers, pp. 203–221.
22. Macdonald, S.; and Mason, C. 1992. A note on *Lutra longicaudis* in Costa Rica. International Union for the Conservation of Nature Otter Specialist Group Bulletin 7:37–38.
23. Mares, M.A.; Ojeda, R.A.; and Barquez, R.M. 1989. Guide to the Mammals of Salta Province, Argentina. Oklahoma City, University of Oklahoma Press.
24. Nowak, R.M. 1991. Walker's Mammals of the World, Vol. 2, 5th Ed. Baltimore, Johns Hopkins University Press.
25. Parera, A. 1996. Las Nutrias Verdaderas de la Argentina. Boletín Tecnico de la Fundación Vida Silvestre Argentina, Buenos Aires, Fundación Vida Silvestre Argentina.
26. Petrini, K. 1992. The medical management and diseases of mustelids. In Proceedings of the Joint Meeting of the AAZV and AAWV. Minnesota Zoological Gardens.
27. Poglayen-Neuwall, I. 1978. Breeding, rearing and notes on the behavior of tayras *Eira barbara* in captivity. International Zoo Yearbook 18:134–40.
28. Redford, K.H.; and Eisenberg, J.E. 1992. Mammals of the Neotropics, Vol. 2. The Southern Cone: Chile, Argentina, Uruguay, Paraguay. Chicago, University of Chicago Press.
29. Reed-Smith, J. 1994–1995. North American River Otter Husbandry Notebook. Grand Rapids, Michigan, John Ball Zoological Garden.
30. Schweizer, J. 1992. Ariranhas no Pantanal. Curitiba, EDIBRAN.
31. Sielfeld, W.K. 1983. Mamiferos marinos de Chile. Santiago, Ediciones de la Universidad de Chile.
32. Spelman, L.H.; Sumner, P. W.; Levine, J.F.; and Stoskohp, M.K. 1994. Anesthesia of North American river otters (*Lutra canadensis*) with medetomidine ketamine and reversal by atipamezole. Journal of Zoo Wildlife Medicine 25(2):214–223.
33. Tierpark, C.H. 1992. Breeding the giant otter *Pteronura brasiliensis*. International Zoo Yearbook 31:240–245.
34. Van Zyll de Jong, C.G. 1987. A phylogenetic study of the Lutrinae (Carnivora; Mustelidae) using morphological data. Canadian Journal of Zoology 65: 2536–2544.
35. Wallach, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals, Medical and Surgical Management. Philadelphia, W.B. Saunders.
36. Wilson, D.E.; and Reeder, D.M. 1992. Mammal Species of the World: A Taxonomic and Geographic Reference. Washington, D.C., Smithsonian Institution Press.



30
Orders Cetacea and Pinnipedia (Whales, Dolphins, Seals, Fur Seals, Sea Lions)

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BIOLOGY

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INTRODUCTION

The Order Cetacea (*ketos,* whale) is represented by mammals that are completely adapted to aquatic life. It is divided into two living suborders: Mysticeti (baleen whales) and Odontoceti (toothed whales). The taxonomic distinction between odontocetes and mysticetes appears to be well supported by morphological and physiological differences, including the previously mentioned presence and absence of teeth, and the echolocation system, which is exclusive to the odontocetes.

Recent phylogenetic analyses of DNA sequences suggest that the cetaceans and hippopotamids (artiodactyl) are sister groups, and that consequently the aquatic characteristics shared between these two groups are synapomorphies.[14] This molecular vision, however, contradicts paleontologic data, which emphatically support a monophyletic origin for the order Artiodactyla and a close relationship between the cetaceans and extinct primitive ungulates (mesonychids). Additionally, no DNA sequence data support the monophyly suggested by the paleontologic theory for Artiodactyla. As pointed out by Gatesy,[14] the simplest explanation for the DNA similarities between hippos and cetaceans is a common ancestor.

Controversial opinions also exist regarding the origin of pinnipeds. The term originates from Latin *pinna,* which means "fin" or "feather," and *pedis,* meaning "foot." In this chapter the classification proposed by Berta and Wyss[2] will be used. They suggest a monophyly for pinnipeds and thus consider them as an order. It is believed that the pinnipeds originated from terrestrial ancestors, related to the Ursidae, Procyonidae, and Mustelidae families, which are known as arctoid carnivores.

Cetaceans and pinnipeds developed a series of adaptive characteristics for the aquatic environment. The cetaceans are exclusively aquatic mammals and reflect a maximum expression of these characteristics. Although the pinnipeds have a close relationship with the water, they also use the terrestrial environment: for reproduction, to molt, or to rest. The main adaptations to the aquatic environment are hereby summarized and are generally present in all the cetaceans. The expression of these adaptations may be observed in pinnipeds, but usually on a smaller scale; the layer of blubber, for example, is thinner in the pinnipeds, and the fur is important in thermoregulation.

Respiratory System

These mammals evolved mechanisms to prolong breath holding and slow respiratory rates, which are much lower than that of terrestrial mammals. This physiological adaptation permits greater freedom in the aquatic environment and reduces temperature and water losses. A large capacity for storing oxygen in the blood allows for deeper dives. Some tissues, especially the muscles, are able to work anaerobically. During dives that last longer than 20 minutes, the muscles run out of oxygen stored in the myoglobin and produce ATP as a result of fermentation.[43]

Circulation

A slower heart beat (bradycardia) is another response of marine mammals to dives. These animals also have peripheral vasoconstriction, which works not only to conserve oxygen, but also body heat. They present differential distribution of blood, directing the blood flow and giving preference to the vital organs. They also have a greater volume of blood than other mammals. A system of countercurrent blood circulation, encountered in the flippers of cetaceans and pinnipeds, is important in thermoregulation, avoiding heat loss to the environment.

Thermoregulation

The cetaceans have almost no body hair, with only a few hairs being found along the snouts of young odontocete individuals and on the heads of some mysticetes. Apart from the above-mentioned countercurrent system, the cetaceans have a thick layer of blubber that works as thermal insulation. Because of the large body proportions, cetaceans have a smaller surface/volume ratio than most other mammals and therefore lose less heat to the surrounding water.

Osmoregulation

Cetaceans obtain necessary water from food.[43] The kidneys of cetaceans and pinnipeds are highly specialized, subdivided into hundreds or thousands of lobules or reniculi, each one of which is a miniature renal unit.[54]

Shape of the Body and Other Anatomical Adaptations

Cetaceans have no hind limbs, and the pelvic girdle is only vestigial. The body is fusiform and hydrodynamic. They have flexible backbones, which can resist the hydrostatic pressure in deep dives. They have extremely expansive veins, venous sinuses, and retia mirabilia, which may increase its volume with a greater blood flow, thus filling in spaces while the air is being compressed during a dive. The retia mirabilia may also help in thermoregulation.

TAXONOMY

The present classification of the suborder Mysticeti (baleen whales) includes four families; Balaenidae (bowhead and right whales), Neobalaenidae (pygmy right whale), Balaenopteridae (rorquals), and Eschrichtiidae (gray whale),[39] of which only the gray whale is not represented in South America. The suborder Odontoceti has nine families: Platanistidae (Ganges and Indus River dolphins), Pontoporiidae (franciscana and Yangtze River dolphin), Iniidae (Amazon River dolphin), Monodontidae (beluga and narwhal), Phocoenidae (porpoises), Delphinidae (marine dolphins), Ziphiidae (beaked whales), Physeteridae (sperm whale), and Kogiidae (pygmy and dwarf sperm whales).[39] Only the Monodontidae and Platanistidae families do not have South American representatives.

The order Pinnipedia has only three families: Odobenidae (walruses), Otariidae (fur seals and sea lions), and Phocidae (seals). Of these, only the Odobenidae family do not have representatives in South America.

DISTRIBUTION AND HABITAT

Among the mysticetes that occur in South America, the genera *Eubalaena* (southern right whale) and *Megaptera* (humpback whale) can be seen frequently from the coast in certain periods of the year. The other genera (*Balaenoptera* and *Caperea*, rorquals and pigmy right whale, respectively) rarely approach the coast of the South American continent and are typically oceanic animals.

Most of the mysticetes are migratory, moving during the summer to feed in Arctic waters or toward the Antarctic continent in the Southern Hemisphere. During the winter months, these animals migrate to temperate and subtropical waters to bear young and breed.

The Pontoporiidae (*Pontoporia blainvillei*) and Iniidae (*Inia geoffrensis*) are called river dolphins. However, despite the fact that *P. blainvillei* (franciscana dolphin) presents all the characteristics of the other river dolphins, its present distribution seems to be restricted to the Atlantic coastal region of South America.[35] *I. geoffrensis* (Amazon River dolphin) is exclusively a freshwater dolphin, with a large distribution in the Amazon and Orinoco River basins.[3] The dolphins of the

Delphinidae family have a predominantly marine distribution, with the exception of *Tursiops truncatus* (bottlenose dolphin), which may also occur in estuarine and coastal environments,[60] and the genus *Sotalia,* which has a freshwater species (*Sotalia fluviatilis*) distributed in the Amazon basin, and a marine/estuarine species (*Sotalia guianensis*) on the Atlantic Coast of the South American continent.[31] The South American porpoises of the genus *Phocoena* (Phocoenidae) have a coastal and oceanic distribution. All the other odontocete families (Kogiidae, Physeteridae, and Ziphiidae) that occur in the waters of South America, are predominantly oceanic.

Among the pinnipeds of the Otariidae family, the South American sea lion *(Otaria flavescens)* seems to have the greatest distribution, with groups of animals being recorded from southern Brazil on the Atlantic Coast[46, 47] to as far north as Peru on the Pacific Coast.[40] The northern limit of this species' range on the Atlantic Coast was reported at 13° S, which, however, was considered to be an erratic movement.[47]

The South American fur seal (*Arctocephalus australis*), the Galapagos fur seal (*Arctocephalus galapagoensis*), and the Juan Fernandez fur seal (*Arctocephalus philippii*) are the only species of the genus with an exclusively South American distribution. Erratic movements of *Arctocephalus tropicalis* (subantarctic fur seal) and *Arctocephalus gazella* (Antarctic fur seal) have been recorded during the austral winter months for the Atlantic Coast of South America,[36] but these species do not reproduce on South American land.[22]

Among the pinnipeds of the Phocidae family, the only species that reproduces in South America is the southern elephant seal (*Mirounga leonina*), which occurs on both the Atlantic and Pacific sides of the continent.[40] The other four genera of the Southern Hemisphere phocids are the Antarctic seals (*Hydrurga, Lobodon, Leptonychotes,* and *Ommatophoca*), which have a circumpolar distribution and do not reproduce in South America.[22] However, vagrant individuals of *Lobodon* and *Hydrurga* have been reported on the coasts of the South American continent.[36, 40, 45]

EXPLOITATION

The exploitation of cetaceans dates back millions of years. Whale bones were found in human settlements in Alaska approximately 1500 years b.c.[55] During the 19th century, the exploitation of large whales, already well in progress in the Northern Hemisphere, also spread to Antarctic waters. Most of the animals killed in the Antarctic belonged to the stock of whales of the South American coast.

The main products extracted from whales were oil and meat. The bones were used in the corset industry and to manufacture glue and gelatin.[55] With the reduction of whale populations and the discovery of petrol, a cheap and efficient substitute for whale oil, and with the technological development that occurred during the 20th century, the exploitation of the large whales decreased radically.

In South America many whaling stations were established at the beginning of the 20th century. Most of them ceased operation in the 1950s–1960s. In December 1946, a group of countries that practiced whaling formed the International Whaling Commission (IWC), with the aim of regulating captures.[6] Presently Argentina, Brazil, Chile, Ecuador, Peru, and Venezuela are members of the IWC (J.T. Palazzo, Jr., personal communication, 1999). Despite being a member of this commission, Brazil was the last South American nation to stop killing whales. A whaling station in the district of Costinha, in northeastern Brazil, began to operate in 1910. From 1951 the Companhia de Pesca Norte do Brasil (COPESBRA) took over operation of the station. COPESBRA was a subsidiary of Nippon Reizo Kabashiki Kaisha of Tokyo, Japan.[61] The activities of this station continued until 1986, ceasing only after the moratorium on whaling was declared.

Incidental catches of odontocetes in fisheries are common on both sides of the South American continent. This is the major threat faced by dolphins. However, it is known that some dolphin species are intentionally caught for bait or meat consumption around the coasts of South America. In Chile and Peru, some marine mammals have been deliberately killed for bait for centolla fisheries by artisanal fishermen.[26,58] The species that are mainly affected by fisheries, directly or accidentally, on the southeastern Pacific side are the dusky dolphin (*Lagenorhynchus obscurus*), the Burmeister's porpoise (*Phocoena spinipinnis*), the bottlenose dolphin, and the long-beaked common dolphin (*Delphinus capensis*).

Among the cetaceans of the western South Atlantic, the franciscana dolphin and the estuarine dolphin (*S. guianensis*) are probably the most vulnerable cetaceans to fisheries, especially because of their coastal habitat.[33, 49] There is some information on intentional killing of cetaceans on the Atlantic Coast of South America, however, its extent is unknown and requires further studies.

In 1978, the shipment of live specimens of Commerson's dolphin (*Cephalorhynchus commersonii*) was authorized by the Argentinean government for Japanese and German aquariums. Some dolphins died during transportation to Japan and another shipment of dolphins was authorized in the same year.[18] Currently, the live capture of cetaceans is forbidden in most South American countries.

There is a limited commerce in eyes, genitals, fat, and teeth of freshwater dolphins (*I. geoffrensis* and *S. fluviatilis*) because of Amazonian legends, which claim that these dolphin parts are love charms or have medicinal properties. However, apparently dolphins are not killed for this purpose; the parts are taken only from animals incidentally caught in fishing nets.[3,51]

Whale-watching activities are another kind of cetacean exploitation in the sense that the animals are used to make money. This kind of activity is fairly recent in South America, and probably started in Península de Valdés (Argentina) a few decades ago. Groups of tourists are permitted to approach the whales, under certain regulations and conditions.[28] Whale-watching or dolphin-watching in South America will probably increase in the near future, as is already the case for the Archipelago Fernando de Noronha concerning spinner dolphins, which can be seen year-round in the crystal blue waters of Dolphin Bay in that Archipelago.

Some species of South American pinnipeds were substantially important to the life of natives. Prehistoric people living near the southern tip of South America hunted sea lions (*O. flavescens*) for food and pelts. For hundreds of years, the fur seal (*A. australis*) was an important item in the diet of the Fuego-Magellanic canoe people who lived in the Beagle Channel and Chilean fiords, as it was for the Charrua Indians along the Uruguayan coast.[40]

More recently, extensive commerce in pinniped skins and oil became the primary motive for hunting. Commercial sealing of the Juan Fernández fur seal (*A. philippii*) in Chile began in 1687, and perhaps 3 million seals were killed during a 7-year period prior to 1824, by which time few remained alive. Sealing continued on some islands until the species was believed to be extinct.[40] Nowadays there is no commercial hunting of this species, although some are illegally caught for lobster bait.[41]

Whalers were the first to hunt the Galapagos fur seal (*A. galapagoensis*), but commercial sealers also operated in the Galapagos Islands. At least 22,500 were killed by U.S., British, and Spanish sealers between 1816 and 1933. U.S. whalers killed South American fur seals during the late 18th century.

The Uruguayan fur seal and sea lion industry was the longest sustained of this kind in the world. By 1950 the slaughters were directed to young males in order to protect the population as a whole, and also because the pelts of young seals sold for a better price in the international haute couture industry.[44] During the final years of hunting, the numbers of seals killed declined. Figures of the number killed, which had reached as high as 14,000 fur seals per year before 1982, dropped to 7000 per year in 1991, when the sealing industry in Uruguay ceased.[59] The oil from seals and sea lions killed in Uruguay was used in the tanning process and the meat was used for animal food.

Sea lions were also heavily exploited in Chile, and commercial sealing of sea lions in Argentina continued at peak levels throughout the 1950s, but declined in the 1960s for economic reasons.

British and U.S. sealers killed large numbers of fur seals and sea lions in western Patagonia between 1825 and 1865. U.S. sealers began killing the subantarctic fur seal (*A. tropicalis*) at Gough Island about 1800. Some sealing resumed between 1860 and 1890, but few seals were left alive by then. Sealing on this island ended completely by 1892. On Prince Edward Island sealing has been prohibited since 1948. Sealing at Crozet, St. Paul, and Amsterdam Islands was also important but did not last long because of the small populations.

South Georgia was an important sealing site. Commercial sealing for the Antarctic fur seal (*A. gazella*) began there by U.S. sealers in the early 1790s. The harvesting of southern elephant seals (*M. leonina*) began after the number of Antarctic fur seals was reduced in the early 1800s. Elephant seals were exploited for oil, but the colony of elephant seals was reduced so quickly that by 1900 sealing was no longer commercially profitable. Sealing continued through 1964, when shore-based whaling at South Georgia ended and sealing alone could not justify maintaining an oil-producing business there.[40]

As with the cetaceans, some pinnipeds in South America, as well as being intentionally killed, are also accidentally killed in fishing nets, and, on a smaller scale, taken directly for crab bait in the southeastern Pacific.

WILDLIFE POPULATION STUDIES

Estimation of Population Size

Line transect sampling is the most appropriate method for estimating the abundance of biological populations.[8] Modification of these techniques for use in surveys of large whales is described in detail elsewhere.[20] Line transects have been used to estimate minke whale distribution and abundance in the former whaling ground off the northeastern coast of Brazil.[63]

In the Amazon, the cue-counting method was applied to estimate the abundance of *I. geoffrensis* and *S. fluviatilis* using line and strip transects. The latter yielded more accurate results for estimating dolphin numbers in rivers wider than 500 m.[52]

The population density of the estuarine dolphin was also estimated using line transects in Guaraqueçaba Bay, Paraná State, southern Brazil. The results suggest that estimates must be made in subareas when conducted inside the estuary, according to the physiognomic differences.[5]

Photo identification, using mark and recapture methods, and aerial surveys are currently being used to estimate population densities of cetaceans in South America.[4,7,32] A new model to estimate population sizes of cetaceans based on the Bayes-hierarchy model was recently proposed, using humpback whales from the Brazilian coast as a model.[21]

Argentinean researchers perform aerial censuses of the South American sea lion in Patagonia, combining it with the use of hand-tally counters from elevated points, or counting the animals while the observer walks slowly around the sea lions during the breeding season.[42]

Marking and Sampling Techniques

Individual marks may be used to distinguish individuals for positive identification when they are reobserved. Natural marks are body markings and scars that permit the recognition of individuals. The relatively few studies of seals based on natural body patterns or scars have been limited to colonial species.[13] Paints, bleaches, and dye marking are temporary marks, which may be applied for short-term studies. There are limitations to these methods, such as problems associated with applying discernible marks.[16] Scar marking, as well as branding, have been used extensively to mark wild animals.

Tagging is the most widely used method of marking marine mammals. The principal tags used today are metal band tags or plastic disc tags, which are self-piercing and can be quickly applied using special pliers. The tags carry stamped serial numbers and a "Return to Organizer" label. The tags are applied to the hind edge of the fore flipper of fur seals and the hind interdigital web of phocid seals.[13] In cetaceans, the tag is usually attached to the dorsal fin. Tags can also be attached to free-ranging animals with a pole applicator or crossbow, which does not require capture.[23] Plastic tag marks have been used for population studies in Amazonian dolphins since the early 1980s. The tag marks usually remain on the animals for up to 2 years when they fall off during migration as a result of the force of water friction.[52] Besides the tag marks, freeze brands are also being used on Amazonian dolphins. They seem to be more durable marks, allowing long-term studies (V.M.F. da Silva, personal communication, 1999).

Commercial hair bleach and flipper tags were used in Peru to mark the South American fur seal for census and a study of behavior and population dynamics.[57]

Natural marks and color pellet marks were used to estimate the abundance and habitat use of the South American sea lion in southern Brazil. The short duration of the artificial marks upon re-entry of the animals into the water, proved that these marks are efficient only for studies in reproductive areas, where the animals stay on land for longer periods.[44]

Numbered plastic tags were used to mark the southern elephant seal in Península Valdés, Argentina. The marks were placed in the interdigital webbing of one or both hind flippers for movement and site-fidelity studies.[27]

The use of projectiles for biopsy sampling of cetaceans is well-known, providing nonlethal sampling of fresh, uncontaminated tissue for genetic and toxicological studies. This methodology was not used for pinnipeds until recently. A crossbow-launched biopsy system for sampling South American fur seals in Peru has shown excellent results.[15]

Telemetry and Electronic Technology

Early radio-tracking systems for dolphins consisted of a simple radio beacon attached to the animal and a directional antenna and receiving system. Each time the instrumented animal surfaced, a pulse signal was broadcast from the transmitter.[23] Transmitters can be monitored for two basic types of information; the presence or absence of a signal and the relay of various types of data (e.g., heart rate, temperature).[1] Most studies using radio transmitters involve monitoring the activity and movements of individuals. Dive recorders allow the study of dives, registering the dive depths.

Advances in microchip technology led to development of solid state and microprocessor-controlled recorders. Time depth recorders (TDRs) digitally measure depth at preprogrammed time intervals. The time and depth data are stored in random access memory chips, and data can be transferred directly to a computer. This type of equipment must be recovered to retrieve data.[1] A TDR radio transmitter was successfully attached to a female South American fur seal off the coast of Peru for a diving behavior study, remaining in the animal for 2 weeks.[57]

To improve the collection of data, researchers have been focusing on developing instruments that relay the data via satellite. Efforts to place satellite-linked TDRs on great whales have been partially successful in recent years.[29] This technology has also been applied to pinnipeds.

Telemetry studies have also been carried out with Amazonian dolphins. Very-high frequency (VHF) transmitters were attached to the dorsal fin of Amazon River dolphins in the Sustainable Development Reserve of Mamirauá (Brazilian Amazon). Such data as hour, date, transmitter frequency, signal intensity, and direction of the movements of the dolphins were recorded in a microprocessor. The data were downloaded periodically to clean the memory of the system and for analysis.[52] Satellite transmitters were also attached to three Amazon dolphins in order to obtain information on daily and seasonal movements.[53]

Behavior

Behavioral studies are like a dialogue with the animals, and, to be efficient, the right questions must be asked. Scan sampling is used to collect information about a large number of anonymous animals at frequent intervals. Event recording gathers information about parts of complex behavioral patterns, and focal animals are useful for quantifying a sequence of events.[56] Grouping and aggregations are directly related to how animals use the space. Also, social structure and reproductive strategies are represented by the distribution of animals in the environment.

Investigation of the territorial and reproductive behavior of the southern sea lion was carried out.[10] Diurnal rhythm or pattern describes the behavior on a temporal scale. Mother-pup relation is a complex and important category of study. It considers behavioral interactions between mother and pup, first responses of the pup, time until the placenta appears, presence of scavengers, and time of first suckling.[56] It is important to evaluate reactions to humans in order to understand the impact of development. Some interactions are positive, as is the cooperative fishing in Brazil.[37] Vocalizations between males and females in relation to mating and mother-young recognition are valuable. Significant variations in vocalizations were found between different populations of northern, and between northern and southern, elephant seals, showing the importance of this type of study.[24,25]

Behavioral and habitat studies of *I. geoffrensis* in the Sustainable Development Reserve of Mamiruá (Brazilian Amazon) using tag-marked and transmitter-marked animals showed that this species has a high site fidelity for a particular area, and most of the animals seem to be residents in Mamirauá Lake.[53,9]

Long-term studies have been conducted on the behavior of the spinner dolphin (*Stenella longirostris*) in the Archipelago of Fernando de Noronha (Brazil). Movements of the dolphins in Dolphin Bay were monitored on a daily basis. Underwater and aerial activities and social and reproductive behaviors of spinner dolphins have been studied for several years.[50]

Studies on the behavior and sound production of the estuarine dolphin have been conducted in the region of Cananéia in southeastern Brazil. The most frequent social organization observed was the family group, composed of two adults and one calf.[30] At least 11 basic behaviors were described while *S. guianensis* foraged, which most of the time were associated with production of a minimum of four different vocal and two nonvocal sounds.[30]

Parental care of *S. guianensis* has been described in detail, from studies in the lagunar complex of Cananéia (São Paulo State) and Paranaguá (Paraná State) in southeastern and southern Brazil, respectively.[38] Seven different kinds of parental care were described. There were significant differences in the frequency of behaviors between the two studied areas, probably as a result of geographic differences between the two locations.[38]

CONSERVATION PROGRAMS

National parks, reserves, and sanctuaries are usually created for the conservation of terrestrial species. The creation of protected areas in aquatic environments for the conservation of aquatic species, whether riverine or marine, has only recently been attempted.[39] In Golfo San Jose (Argentina), the sanctuary of the southern right whale (*Eubalaena australis*) is a good example. During the past 20 years, this endangered species has shown a significant trend of population increase in several areas in the Southwest Atlantic. The National Marine Park of Abrolhos in the northeast of Brazil is another good example of a marine environment protecting aquatic fauna and flora in South America, including humpback whales (*Megaptera novaeangliae*), which use the protected area of the park for reproduction.[12]

Efforts are being made in Chile to include the surrounding waters of the Humboldt Penguin National Reserve to protect resident populations of cetaceans, pinnipeds, and marine otters.[39] Brazil is also making efforts to create a whale sanctuary in the south Atlantic Ocean, including all the waters from the equatorial line to the limits of the already existing Antarctic sanctuary. The proposal was submitted to the IWC in 1994, but is still being discussed. A decision is to be made during the next IWC meeting in July 2000 (J.T. Palazzo, Jr., personal communication, 1999).

Gorgona National Park in the Colombian Pacific Ocean is another protected marine environment, constituting the main reproductive area of humpback whales in western South America.[11] The South American river dolphins are protected inside the Sustainable Development Reserve of Mamirauá in the Brazilian Amazon, which was created in 1990. Unfortunately, although most whales and dolphins of South America are protected by national laws, law enforcement is precarious in most countries.

In October 1998, the National Center of Research, Conservation and Management of Aquatic Mammals was created in Brazil. The responsibilities of the Center include the development and maintenance of a Brazilian data bank from information gathered through research and conservation studies of marine mammals. An objective is to provide data that encourages the conservation and management of Brazilian aquatic mammals.

In Chile, total protection for all fur seal species was declared in 1978.[41] The population of *A. philippii* has increased from about 200 animals in 1965 to more than 10,000 in 1991.[40,41]

The Galápagos fur seal *A. galapagoensis* has been protected in Ecuador since 1934. Despite being protected by law in Peru since 1969, the South American fur seal (*A. australis*) is still subjected to illegal hunting.[40] After a long period of heavy exploitation, the South American sea lion (*O. flavescens*), which has been protected by law in Argentina since 1974, has recently shown signs of population recovery[40].

The Antarctic seal (*A. gazella*) and the subantarctic seal (*A. tropicalis*) are protected in their breeding areas under conservation programs. The resulting increase of the populations of these two fur seals is probably responsible for an increase in the records of their appearance on the South American coast.[34]

All cetacean species are under the regulation of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES), listed in Appendix I (species threatened with extinction) or Appendix II (species that may become threatened with extinction unless trade is regulated).[48] Among the pinnipeds, all South American species of the genus *Arctocephalus* are listed in Appendix II of CITES. The other South American pinnipeds are not yet included in CITES regulations.[48]

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REFERENCES

1. Bengtson, J.L. 1993. Telemetry and electronic technology. In R.M. Laws, ed., Antarctic Seals Research Methods and Techniques. Cambridge, Cambridge University Press, p. 390.
2. Berta, A.; and Wyss, A.R. 1994. Pinniped phylogeny. In A. Berta and T.A. Deméré, eds., Contributions in Marine Mammals Paleontology Honoring Frank C. Whitmore, Jr. Proceedings of the San Diego Society of Natural History [Special issue] 29:33–56.
3. Best, R.C.; and da Silva, V.M.F. 1989. Amazon river dolphin, boto *Inia geoffrensis* (de Blainville, 1817). In S.H. Ridgway and R. Harrison, eds., Handbook of Marine Mammals, Vol. 4. London, Academic Press, pp. 1–23.
4. Bethlem, C.B.P.; Kinas, P.G.; Engel, M.H.C.; and Freitas, A.C.S. 1998. Estimativas de abundância da baleia jubarte no Banco dos Abrolhos, BA, Brasil [Abundance estimate of humpback whales of the Abrolhos Bank, Bahia, Brazil]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 25.
5. Bonin, C.A. 1997. Estimativa de Densidade Populacional do Golfinho *Sotalia fluviatilis guianensis* (Cetacea, Delphinidae) na Baía de Guaraqueçaba, Litoral do Estado do Paraná [Population density estimate of the estuarine dolphin in Guaraqueçaba Bay, Paraná State, Brazil]. Monografia de conclusão do Curso de Ciências Biolgicas. Paraná, Universidade Federal do Paraná, p. 45.
6. Bonner, N. 1998. Whales of the World. London, Blandford, p. 191.
7. Bordino, P.; and Tausend, P. 1998. Avistabilidad y estimacin preliminar de densidad del delfin franciscana *Pontoporia blainvillei* en Bahia Anegada, Argentina [Sightings and preliminary density estimation of franciscana dolphins in Anegada Bay, Argentina]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p.28.
8. Burmann, K.P.; Anderson, D.R.; and Laake, J.L. 1980. Estimation of density from line transect sampling of biological populations. Wildlife Monographs 72:1–202.
9. Camargo, Y.R.; and da Silva, V.M.F. 1998. Uso de habitat e comportamento do boto da Amazônia *Inia geoffrensis* na Reserva de Desenvolvimento Sustentável Mamirauá, AM, Brasil [Habitat use and behavior of the Amazon river dolphin in the Sustainable Development Reserve of Mamirauá, Amazon, Brazil]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 36.
10. Campagna, C.; and Le Boeuf, B.J. 1988. Reproductive behaviour of the southern sea lion. Behaviour 104(3–4):233–262.
11. Capella, J.; Florez-Gonzales, L.; and Bravo, G. 1994. Residencia y historia de reavistamientos de ballenas jorobadas, *Megaptera novaeangliae,* en el Pacífico Colombiano [Residence and history of re-sightings of humpback whales in the Colombian Pacific Ocean]. In Abstracts of the 6° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Florianpolis, Imprensa Universitária, p. 77.
12. Engel, M. 1994. Conservação e pesquisa das baleias jubarte, *Megaptera novaeangliae,* no Banco dos Abrolhos e adjacências de 1988 a 1993 [Conservation and research of humpback whales of the Abrolhos Bank and

adjacent waters]. In Abstracts of the 6° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Florianpolis, Imprensa Universitária, p. 98–99.

13. Erickson, A.W.; Bester, M.N.; and Laws, R.M. 1993. Marking techniques. In R.M. Laws, ed., Antarctic Seals Research Methods and Techniques. Cambridge, Cambridge University Press, p. 390.
14. Gatesy, J. 1997. More DNA support for a Cetacea/Hippopotamidae clade: The blood-clotting protein gene γ–fibrinogen. Molecular Biology and Evolution 14(5):537–543.
15. Gemmel, N.J.; and Majluf, P. 1997. Projectile biopsy sampling of fur seals. Marine Mammal Science 13(3):512–516.
16. Gentry, R.L. 1979. Adventitious and temporary marks in pinniped studies. In H.L. Hobbs and P. Russel, eds., Report on the Pinniped and Sea Otter Tagging Workshop, Appendix B. Arlington, Virginia, American Institute of Biological Sciences, pp. 39–43.
17. Geraci, J.R.; and Lounsbury, V.J. 1993. Marine Mammals Ashore: A Field Guide for Strandings. Galveston, Texas A&M University, p. 305.
18. Goodall, R.N.P.; and Cameron, I.S. 1980. Exploitation of small cetaceans off southern South America. Report of the International Whaling Commission 30:445–450.
19. Harrison, R.J.; and King, J.E. 1980. Marine Mammals, 2nd Ed. London, Hutchinson University Library, p. 192.
20. Hiby, A.R.; and Hammond, P.S. 1989. Survey techniques for estimating abundance of cetaceans. Report of the International Whaling Commission (Special issue) 11:47–80.
21. Kinas, P.G. 1998. Modelo Bayes-Hierárquico para estimar o tamanho de uma população [Bayes-Hierarchic model to estimate population size]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 101.
22. King, J.E. 1983. Seals of the World, 2nd Ed. London, British Museum of Natural History and Oxford University Press, p. 240.
23. Leatherwood, S.; Reeves, R.R.; Perrin, W.F.; and Evans, W.E. 1988. Whales, and Porpoises of the Eastern North Pacific and Adjacent Arctic Waters: A Guide to Their Identification. New York, Dover, p. 245.
24. LeBoeuf, B.J.; and Peterson, R.S. 1969. Dialects in elephant seals. Science 166:1654–1656.
25. LeBoeuf, B.J.; and Petrinovich, F. 1974. Elephant seals: Interspecific comparison of vocal and reproductive behavior. Mammalia 38:16–32.
26. Lescrauwaet, A.C.; and Gibbons, J. 1994. Mortality of small cetaceans and the crab bait fishery in the Magallanes area of Chile since 1980. In W.E. Perrin, G.P. Donovan, and J. Barlow, eds., Gillnets and Cetaceans. Report of the International Whaling Commission (Special issue) 15:485–494.
27. Lewis, M.; Campagna, C.; and Quintana, F. 1996. Site fidelity and dispersion of southern elephant seals from Patagonia. Marine Mammal Science 12(1):138–147.
28. Lichtschein de Bastica, V.; and Bastida, R. 1983. Whale watching in Argentina. In Proceedings of the Global Conference on the Non-Consumptive Utilization of Cetacean Resources. Boston, Connecticut Cetacean Society, pp. 1–4.
29. Mate, B.R. 1987. Development of Satellite-Linked Methods of Large Cetaceans Tagging and Tracking in OCS Lease Areas—Final Report. Minerals Management Service, Contract AA-730-79-4120-0109. Washington, D.C., U.S. Department of the Interior.
30. Monteiro-Filho, E.L.A. 1991. Comportamento de Caça e Repertrio Sonoro do Golfinho Sotalia Brasiliensis (Cetacea: Delphinidae) na Região de Cananéia, Estado de São Paulo [Hunting behavior and sound production by *Sotalia brasiliensis* in the region of Cananéia, São Paulo State, Brazil]. Doctoral thesis, Universidade Estadual de Campinas.
31. Monteiro-Filho, E.L.A.; Reis, S.F.; and Monteiro, L. 1999. Geometric analysis of tridimensional skull shape in *Sotalia:* Discrimination between freshwater and marine dolphins. Abstract submitted to the 8th Biennial Conference on the Biology of Marine Mammals.
32. Ott, P.H.; Secchi, E.; Crespo, E.A.; Kinas, P.G.; Bordino, P.; Dalla Rosa, L.; Danilewicz, D.S.; Martins, M.B.; Moreno, I.B.; and Möller, L.M. 1998. Abundance estimation of the franciscana dolphin, *Pontoporia blainvillei,* from aerial surveys and a preliminary analysis of fishery impact in southern Brazil. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 146.
33. Pinedo, M.C. 1994. Review of small cetacean fishery interactions in Southern Brazil with special reference to the franciscana, *Pontoporia blainvillei.* In W.E. Perrin, G.P. Donovan, and J. Barlow, eds., Gillnets and Cetaceans. Report of the International Whaling Commission (Special issue) 15:251–259.
34. Pinedo, M.C.; and Marmontel-Rosas, M. 1987. Primeiros registros do lobo marinho Antártico, *Arctocephalus gazella,* e novos registros de *Arctocephalus tropicalis* para o Rio Grande do Sul, Brasil [First record of the Antarctic fur seal and new records of the subantarctic fur seal of Rio Grande do Sul, Brazil]. In Proceedings of the 2° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Rio de Janeiro, FRCN, pp. 109–110.
35. Pinedo, M.C.; Praderi, R.; and Brownell, R.L. 1989. Review of the biology and status of the franciscana, *Pontoporia blainvillei.* In W.F. Perrin, R.L. Brownell, Z. Kaiya, and L. Jiankang, eds., Biology and Conservation of the River Dolphins. IUCN Occasional Paper 3. Ciland, IUCN, pp. 46–51.
36. Pinedo, M.C.; Rosas, F.C.W.; and Marmontel, M. 1992. Cetáceos e Pinípedes do Brasil. Uma Revisão dos Registros e Guia para Identificação das Espécies [Cetaceans and Pinnipeds of Brazil. A revision of the records and a guide for the identification of the species]. Manaus, Brazil, UNEP/FUA Imprensa Universitária, p. 213.

37. Pryor, K.; Lindbergh, J.; Lindbergh, S.; and Milano, R. 1990. A dolphin-human fishing cooperative in Brazil. Marine Mammal Science 6(1):77–82.
38. Rautenberg, M. 1999. Cuidados Parentais de *Sotalia fluviatilis guianensis* (Cetacea: Delphinidae) na Região do Complexo Estuarino Lagunar Cananéia-Paranaguá [Parental care of the estuarine dolphin in the Estuarine Lagunar Complex of Cananéia-Paranaguá, Brazil]. Masters thesis, Universidade Federal do Paraná, p. 54.
39. Reeves, R.; and Leatherwood, S. 1994. Dolphins, Porpoises and Whales: 1994–1998 Action Plan for the Conservation of Cetaceans. Gland, Switzerland, IUCN/SSC Cetacean Specialist Group, p. 91.
40. Reeves, R.; Stewart, B.S.; and Leatherwood, S. 1992. The Sierra Club Handbook of Seals and Sirenians. San Francisco, Sierra Club Books, p. 359.
41. Reijnders, P.; Brasseur, S.; van der Toorn, J.; van der Wolf, P.; Boyd, I.; Harwood, J.; Lavigne, D.; and Lowry, L. 1993. Seals, Fur Seals, Sea Lions and Walrus: Status Survey and Conservation Action Plan. Gland, Switzerland, IUCN/SSC Cetacean Specialist Group, p. 88.
42. Reyes, L.M.; Crespo, E.A.; and Szapkievich, V. 1999. Distribution and population size of the southern sea lion (*Otaria flavescens*) in central and southern Chubut, Patagonia, Argentina. Marine Mammal Science 15(2):478–493.
43. Ridgway, S.H. 1972. Homeostasis in the aquatic environment. In S.H. Ridgway, ed., Mammals of the Sea: Biology and Medicine. Springfield, Illinois, Charles C. Thomas, pp. 590–747.
44. Rosas, F.C.W. 1989. Aspectos da Dinâmica Populacional e Interações com a Pesca, do Leão Marinho do Sul, *Otaria flavescens* (Shaw, 1800) (Pinnipedia, Otariidae), no litoral sul do Rio Grande do Sul, Brasil [Aspects of the population dynamics and fisheries interaction of the South American sea lion in Rio Grande do Sul, Brazil]. Masters thesis, Universidade do Rio Grande, p. 88.
45. Rosas, F.C.W.; Capistrano, L.C.; Di Beneditto, A.P.; and Ramos, R. 1992. *Hydrurga leptonyx* recovered from the stomach of a tiger shark captured off the Rio de Janeiro coast, Brazil. Mammalia 56(1):153–155.
46. Rosas, F.C.W.; Haimovici, M.; and Pinedo, M.C. 1993. Age and growth of the South American sea lion, *Otaria flavescens* (Shaw, 1800), in southern Brazil. Journal of Mammalogy 74(1):141–147.
47. Rosas, F.C.W.; Pinedo, M.C.; Marmontel, M.; and Haimovici, M. 1994. Seasonal movements of the South American sea lion (*Otaria flavescens,* Shaw) off the Rio Grande do Sul coast, Brazil. Mammalia 58(1):51–59.
48. Schouten, K. 1992. Checklist of CITES Fauna and Flora: A Checklist of the Animals and Plant Species Covered by the Convention on International Trade in Endangered Species of Wild Fauna and Flora. Amsterdam, CITES.
49. Siciliano, S. 1994. Review of small cetaceans and fishery interactions in coastal waters of Brazil. In W.E. Perrin, G.P. Donovan, and J. Barlow, eds., Gillnets and Cetaceans. Report of the International Whaling Commission (Special issue) 15:241–250.
50. Silva, J., Jr.; Pereira, J.; Yamamoto, M.; Dupont, F.; Guiera, C.; Vasconcelos, G.; Mesquita, D.; Groch, K.; Damianp, C.; Almeida, L.; Marigo, J.; Pimentel, T.; Eleno, E.; Mello, G.; and Silva, F. 1998. Histria natural do golfinho rotador, *Stenella longirostris* (Gray, 1828), em Fernando de Noronha [Natural history of the spinner dolphin in the Archipelago of Fernando de Noronha, Brazil]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 199.
51. da Silva, V.M.F.; and Best, R.C. 1994. Tucuxi *Sotalia fluviatilis* (Gervais, 1853). In S.H. Ridgway and R. Harrison, eds., Handbook of Marine Mammals, Vol. 5. London, Academic Press, pp. 43–69.
52. da Silva, V.M.F.; Martin, A.R.; Rosas, F.C.W.; and Marmontel, M. 1994. Mamíferos aquáticos na Estação Ecolgica do Lago Mamirauá (EELM), Amazonas, Brasil [Aquatic mammals of the Mamirauá Reserve, Amazon, Brazil]. In Proceedings of the 6° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Florianpolis, Imprensa Universitária, pp. 28–31.
53. da Silva, V.M.F.; and Martin, A.R. 1996. Estudos do boto *Inia geoffrensis* na Amazônia Brasileira [Studies of the Amazon river dolphin in the Brazilian Amazon]. In Abstracts of the 7° Reunión de Trabajo de Especialistas en Mamíferos Acuáticos de América del Sur. Viña Del Mar, SOLAMAC, p. 40.
54. Simpson, J.G.; and Gardner, M.B. 1972. Comparative microscopic anatomy of selected marine mammals. In S.H. Ridgway, ed., Mammals of the Sea: Biology and Medicine. Springfield, Illinois, Charles C. Thomas, pp. 298–418.
55. Slijper, E.J. 1984. Whales and Dolphins. Ann Arbor, Michigan, University of Michigan Press, p. 170.
56. Stirling, I.; Gentry, R.L.; and McCann, T.S. 1993. Behaviour. In R.M. Laws, ed., Antarctic Seals Research Methods and Techniques. Cambridge, Cambridge University Press, p. 390.
57. Trillmich, F.; Kooyman, G.L.; Majluf, P.; and Sanchez-Grinan, M. 1986. Attendance and diving behavior of South American fur seals during El Niño in 1983. In R.L. Gentry and G.L. Kooyman, eds., Fur Seals: Maternal Strategies on Land and at Sea. Princeton, New Jersey, Princeton University Press, pp. 153–167.
58. Van Waerebeek, K.; and Reyes, J. 1994. Interactions between small cetaceans and Peruvian fisheries in 1988/89 and analysis of trends. In W.E. Perrin, G.P. Donovan, and J. Barlow, eds., Gillnets and Cetaceans. Report of the International Whaling Commission (Special issue) 15:495–502.
59. Vaz-Ferreira, R.; and Bianco, J. 1998. Explotacin, sobrevivência y preservacin de los otariidos en el Uruguay [Exploitation, survival and conservation of the otariids from Uruguay]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC,, p. 221.
60. Wells, R.S.; and Scott, M.D. 1999. Bottlenose dolphin *Tursiops truncatus* (Montagu, 1821). In S.H. Ridgway

and R. Harrison, eds., Handbook of Marine Mammals, Vol. 6. London, Academic Press, pp. 137–182.
61. Williamson, G. 1975. Minke whales off Brazil. Scientific Report of the Whales Research Institute 27:37–59.
62. Wyss, A.R. 1989. Flippers and pinniped phylogeny: Has the problem of convergence been overrated? Marine Mammal Science 5(4):243–360.
63. Zerbini, A.N.; da Rocha, J.M.; Andriolo, A.; Siciliano, S.; Moreno, I.B.; and Lucena, A. 1999. Report of a Cetacean sighting survey conducted in the former whaling ground off the northeastern coast of Brazil. Report of the International Whaling Commission (in review).

MEDICINE

Tatiana Lucena Pimentel and Artur Andriolo

INTRODUCTION

Marine mammals are frequently found beached after sustaining an injury from other animals or ships or boats. They have an excellent capacity for cellular regeneration and healing. Do not be too quick to euthanize a seriously injured animal. The radiograph of the head of a sea lion that had been shot showed approximately 60 lead pellets inside it. The animal survived after prolonged treatment (M. Koski, personal communication, 1996).

CAPTIVITY

Animals should be individually identified and records kept of every procedure and observation. It is important that the records be complete as to age, sex, marking, and weight. Strict adherence to regulations imposed by the country is necessary.

The maintenance of marine mammals in captivity in Brazil is under regulations called the Zoological Law (IBAMA, decree #93, article 31, 1998) that permits only zoos to maintain marine mammals. The São Paulo Zoological Park Foundation has three *O. flavescens* in exhibition; this species may be seen in other zoos and aquariums in South America, such as at the Montevideo Zoo, Uruguay. In 1985 two *Otaria flavescens* were born in the Mundo Marino, Buenos Aires, Argentina.[29] Another pinniped species that is regularly kept in zoos and aquariums in South America is *Arctochepalus australis* but there is no report of reproduction on this continent.[5]

Some of the challenges of exhibiting marine mammals is their size (whales), the lack of information about natural behaviors of free-living animals, and the failure of some species to adapt to captivity. An example of the latter is the *Pontoporia blainvillei,* which has not survived more than a few days in captivity.[6] An *Orcinus orca* was kept after rehabilitation for 5.5 years.[24] South American species have been kept in captivity in other countries. *Inia geoffrensis* has survived captivity in the United States for an average of 32.6 months[51]. *Sotalia* has been maintained since 1967 in U.S. zoos and aquariums, but there is little information about this species in captivity in South America.[3]

Pools

Natural seawater pens or large oceanarium pools are usually adequate. Decking with a nonabrasive surface is necessary for pinnipeds. Water quality in marine mammal exhibits is of critical importance in order to maintain healthy animals and keep the environment free of pathogens. The organisms that are pathogenic to captive animals may or may not be the same as those that cause disease in the wild.[2] The complete water flow-through system is adequate to maintain clean water when the zoo or oceanarium is along the coast. Water treatment is needed when the system is only partial flow-through or closed loop. Chlorination, ozonation, or ultraviolet radiation are methods used to disinfect the water. An adequate range of water temperature is important for the control of body temperature in pinnipeds.

Nutrition

The primary source of water for pinnipeds is from the fish they eat and secondarily from metabolic oxidation of dietary fats.[9,25] Seawater consumption is not of major importance.[40]

A good-quality fresh fish is essential. Whole fish should be fed whenever possible. The amount of fish to be fed to a pinniped should be equal to 8–10% of the body weight, divided into two or three feedings daily.[10] The supplement of 50–100 mg of thiamine (B_1) daily is recommended because of the activity of thiaminase in food fish.[18] Supplemental vitamin E (50–100 IU vitamin E/kg fish) may be used to compensate for potential oxidative losses.[19,20,37] When animals are sick or weak, pinnipeds and cetaceans may be force fed by placing fish in the mouth or via a stomach tube.[20]

Pinnipeds kept in freshwater pools may suffer from dehydration when fed low-sodium diets. Accidental ingestion of freshwater appears to result in sufficient leaching of electrolytes to cause clinical problems.[22,23] The sodium level in marine fish ranges from 30 to 150 mg/100 g of fish.[18] The practice of thawing fish in cold, freshwater reduces the sodium level in the feed fish at the rate of 25% in 3 hours. Classic signs of sodium deficiency are extreme muscular weakness, elevated hematocrit (up to 60%), and significantly lowered serum sodium chloride. Replacement therapy with oral sodium chloride at 2–3 g/kg of fish fed usually results in prompt recovery.[22,23] Good results are achieved by dipping the fish into a bucket with 2% saltwater (M. Flügger, personal communication, 1999).In captivity, *T. truncatus* eat marine fish (herring, capelin, and sardines) in four daily feedings, plus vitamins and mineral supplements (such as Sea Tabs, Pacific research Labs, Inc.; C.UJ. McKinnie, personal communication, 1999).

IMMOBILIZATION AND ANESTHESIA

Physical Immobilization

Immobilization is often required for wildlife studies, rehabilitation programs, and to manage animals in captivity.

CETACEANS Shallow-water seines and break-away hoop net techniques are used to capture cetaceans in the wild. The first uses a seine net that encircles a group of animals. The lead line should be just heavy enough to ensure that the net touches the seafloor but light enough to allow a tangled animal to surface. The break-away hoop net is best used in dolphins engaged in bow-riding behavior. The collector must place the hoop net in front of the animal's head when it surfaces to breathe.[7] For transportation, the animals should be positioned to ensure normal respiration in a moist, flat stretcher, supported by foam rubber or other padding.[7]

Animals in captivity can be trained to cooperate in husbandry routines that facilitate efficient maintenance, for example, training animals like dolphins to present their tail flukes so that veterinary personnel may obtain routine blood samples.[33] Nontrained animals may be stranded by draining the pool. Animals usually become calm when stranded and may be approached easily.[7]

PINNIPEDS Physical capture techniques employed for otariids include a variety of nets, hoop nets, snares,[7] and portable fences with restraint cages, restraining boards, or restraint bars.[8,15,16] The long-handled hoop net is particularly useful. The Wally net is an efficient device for catching larger seal lions. Otariids must be approached carefully, because they are quick and agile. The net is hurled over the animal, which becomes enveloped, and can be wrapped for management or short transportation.

Phocids may be easily approached, but because of their size and strength, it is necessary to use further restraint devices. The Wally net may be used for phocids, but other apparatuses are better. Another capture technique is the use of a sack with four ropes attached to each quarter around the sack mouth. Two persons each holding two ropes stand on either side of the seal and pull the sack over its head and fore flippers, which hold them immobile.[44] A special squeeze cage is available commercially to restrain pinnipeds. The flexibility of the guillotine, swinging doors, and the folding side, make this cage one of the best ways to physically restrain pinnipeds and permits approaches to different parts of the animal for measurement, drug administration, marking, and treatment. Wood or metal cages may be used efficiently to transport pinnipeds.

Chemical Immobilization and Anesthesia

CETACEANS Chemical immobilization and general anesthesia of cetaceans are rarely indicated. If necessary, it should be performed under strictly controlled conditions and with respiratory assistance. Chlordiazepoxide hydrochloride (0.5 mg/kg, intramuscularly [IM]) is the anesthesia in widest use and is recommended as safe.[7]

PINNIPEDS See Tables 30.1 and 30.2 for drugs used. To avoid death, it is important to estimate correctly the weight and the health state of the animal. To determine the correct dose of the agent, an accurate weight estimation is crucial before immobilizing an animal. It is important to ensure that the drug is injected into muscle, not into blubber.

Xylazine hydrochloride was used to immobilize southern elephant seal pups (1.0–4.9 mg/kg),[46] and adults (1.6–3.9 mg/kg).[47] It was considered an effective immobilizing agent for the pups with reversibility of any undesirable side effects that developed, and a good immobilizing agent for the adults with no undesirable secondary effects. Tiletamine-zolazepam (1:1) (Telazol, Zoletil) was used with success to immobilize elephant seals (young: 0.52 mg/kg; weaned: 0.61 mg/kg) in order to collect blood, perform measurements, and collect tissue.[30]

In captivity, induction in otariids is best achieved through the delivery of a volatile anesthetic such as halothane or isoflurane delivered from an appropriate face mask. Phocids may be induced with a barbiturate administered intravenously (sodium thiopental at 10 mg/kg body weight).[17]

TABLE 30.1. Principal drugs administered by intramuscular injection and dosages for immobilization of phocids, considering the effective drugs with low mortality

Drugs	Species	Dosage
Succinylcholine chloride	*Mirounga leonina*	2.5 mg/kg
Phencyclidine hydrochloride +	*Mirounga leonina*	0.3 mg/kg each
promazine hydrochloride	*Leptonychotes weddellii*	0.3 mg/kg each
	Lobodon carcinophagus	0.7 mg/kg each
	Hydrurga leptonyx	0.7 mg/kg each
	Ommatophoca rossii	0.6 mg/kg each
Xylazine hydrochloride	*Leptonychotes weddellii*	2.0 mg/kg
	Lobodon carcinophagus	2.5 mg/kg
Ketamine hydrochloride + xylazine	*Mirounga leonina*	4.5 + 0.9 mg/kg
Tiletamine + zolazepam	*Mirounga leonina*	1.0 + 1.0 mg/kg
	Leptonychotes weddellii	0.5–1.0 + 0.25–5 mg/kg

Source: Data summarized from reference 12.

TABLE 30.2. Principal drugs administered by intramuscular injection and dosages for immobilization of otariids, considering effectiveness but with undesirable side effects or with 5–10% of mortality

Drugs	Species	Dosage
Xylazine hydrochloride	*Otaria flavescens*	1.4 mg/kg pups 2.0 mg/kg adults
Ketamine hydrochloride +	*Arctocephalus gazella*	4.0–5.0 + 2.0 mg/kg
xylazine hydrochloride	*Arctocephalus australis*	3.0–4.0 + 0.4 mg/kg
	Arctocephalus galapagoensis	3.0–5.0 + 0.5 mg/kg
Tiletamine + zolazepam	*Arctocephalus gazella*	1.2–1.7 + 1.6 mg/kg

Source: Data summarized from reference 12.

DISEASES, DIAGNOSIS, AND TREATMENT

Physical examination, hematologic and serum biochemical tests, and fecal analysis for parasites should be performed on each animal admitted to a facility. Animals with suspected contagious diseases (e.g., salmonellosis or leptospirosis) should be isolated in special pens and maintained under standard quarantine procedures.[10]

Renal disease is diagnosed in animals exhibiting one or more of the following clinical signs: polydipsia, depression, and abdominal pain, combined with elevated blood urea nitrogen (BUN), creatinine, and phosphorus. The complete blood cell (CBC) count often shows a leukocytosis.[10,11] Leptospirosis is the most common cause of renal disease based on serologic examination. Animals with renal disease should be treated with oral and subcutaneous fluids (lactated Ringer's solution or 5% dextrose in water). If leptospirosis is suspected, antibiotic treatment must be initiated with tetracycline hydrochloride at a dosage of 22 mg/kg. If the animal is not eating, long-acting tetracycline (Liquamycin LA-200) at a dosage of 11 mg/kg IM should be used.[14]

Signs of pneumonia include a productive or nonproductive cough, wheezing, moist rales, crackles, or harsh lung sounds on auscultation. Animals are diagnosed as having verminous pneumonia if at least one of three fecal examinations is positive for the larvae of *Parafilaroides decorus* (lung worm).[13] Cases of pneumonia should be treated with a mucolytic agent, acetylcysteine 20% (Mucomyst), and a bronchodilator, albuterol sulfate (Proventil), nebulized to the animals for 15 minutes two to four times daily. [14] Smaller animals (< 100 kg) can be placed in a covered cage for this treatment whereas larger animals can be nebulized by covering their faces with a cone.

Most cases of pneumonia can also be treated with 22 mg/kg chloramphenicol for 14–30 days. Other, more recent, treatment regimens include enrofloxacin (Baytril) at 5.5 mg/kg, cephalexin (Keflex) at 6–12.5 mg/kg, or gentamicin (Gentocin) at 2.2 mg/kg IM. Animals with fecal examinations positive for lung worm larvae should be treated with ivermectin (Ivomec) at 0.2 mg/kg once every 10-14 days until the fecal examination is negative. Animals with feces containing more than 0-1 lung worm larva per low-power field should be treated with ivermectin concurrently with dexamethasone (Azium) or flunixin meglumine (Banamine) to reduce inflammation caused by the dying nematodes.

The initial dosage of dexamethasone is 0.2–0.4 mg/kg for 2–4 days. The dose is decreased to 0.1–0.2 mg/kg for 2–4 days and reduced once more to 0.05–0.1 mg/kg for 2–6 days, for a total treatment time of 6–14 days.[14] The severity of clinical signs and the numbers of parasites found during the fecal examination will determine the anti-inflammatory dosage and treatment length.

When verminous pneumonia complicates cases of renal disease, animals should be similarly treated with the addition of subcutaneous (SC) fluids and an initial antibiotic therapy of tetracycline. After 2 weeks of tetracycline therapy for the renal disease, many animals subsequently should be given chloramphenicol for 14–21 days to treat the pneumonia.

Seizures should be treated with fluid therapy and diazepam, starting with 0.1 mg/kg IM. If this dose of diazepam is ineffective, the animal may be given up to 0.2 mg/kg IM every 2–6 hours to control the seizures. The animals should be treated with 22 mg/kg chloramphenicol or 25 mg/kg trimethoprim/sulfadiazine (Tribrissen 960), if there is a leukocytosis.[14]

Skin diseases are treated by routine wound care plus 22 mg/kg ampicillin or 20,000-40,000 IU/kg penicillin G benzathine IM. Moderate to severe wounds should be cleaned by hydrotherapy and the ampicillin treatment supplemented with gentamicin at 2 mg/kg IM. Cephalexin at 6.25-12.5 mg/kg has been used more often since 1989 to treat animals with severe wounds.[14] Only clean wounds less than 6 hours old should be closed with sutures. Wounds to be sutured should be flushed and also packed with sulfanilamide powder as a precautionary measure. Nonabsorbable suture material (Vetafil) should be used. Access to swimming water should be limited until the sutures have been removed.[49]

Animals with suspected gastric ulcers should be treated with cimetidine hydrochloride (Tagamet) at 5 mg/kg.

Biological Data for Pinnipeds

The deep core body temperature of sea lions ranges from 36.5°C to 37.5°C. As in the Cetacea, the relatively thin, well-vascularized flippers are important organs of thermoregulation. Respiratory rates in pinnipeds range from 6 to 14 breaths per minute. The heart rate of the sea lion varies from 55 beats per minute while diving to 120 beats per minute during normal air breathing. A slightly higher range of 90–180 beats per minute is recorded in the pup. Heart rates for the harbor seal and elephant seal are 60 and 80 beats per minute, respectively.

Small blood samples may be collected from pinnipeds by clipping one of the nails. Blood samples of phocids (true seals and elephant seals) may be collected from the intravertebral extradural veins. The clinician should first find the pelvis and from this site, count the second or third intravertebral space, then insert the needle almost vertically and slightly cranially between any two of the lumbar vertebrae.[49] Blood samples may be collected from otariids (sea lion) and walruses from the flippers or iliac veins. Urine samples may be collected by using a French catheter (Table 30.3).

Rescue and Rehabilitation

In Brazil, if an *A. australis* strands on the beach it is easy to rehabilitate it because it eats in captivity, but *A. tropicalis* is extremely difficult to feed in captivity. In this case, the Jordan device can be used.

The animal should be rested, administered fluid, and given anthelmintic therapy. After 8–15 days of care in a rescue facility, the animal may be released to the wild, preferably on a deserted beach in order to provide tranquility to the animal. It needs to be left some distance from the water so it is able to walk toward the water, stopping as many times as it wants. It is extremely

TABLE 30.3. Urine values of pinnipeds

	Sea Lion	Fur Seal
Specific gravity	1.030–1.080	1.030 – 1.080
Color	Pale yellow to straw color	Pale yellow to straw color
PH	5–6	5–6
Sodium mEq/L	104–491	36–160
Chloride mEq/L	309–1127	38–140
Potassium	36–78	24–129

Source: See reference 49.

important that the animal not be hurried. It will keep its pectoral flippers opened and the entire body close to the sand. One might think that the animal is lazy, but actually it is sensing the thermoregulation necessary for entering the cold water, because of hyperthermia from the trip to the releasing area. As the animal nears the sea, the sand gets colder.

When a juvenile *M. leonina* (weighing about 600 kg) stranded on a Brazilian beach during the winter, a large effort was organized to rescue it. It required a winch, a tractor, and a truck to take it to the nearest zoo. The animal was darted (while it was far from the sea to make sure it would not escape to the water and run the risk of drowning) and chemically immobilized with tiletamine and zolazepam hydrochloride (1.7 mg/kg Zoletil IM). It was then lifted with the help of cranes and placed on a truck. The truck was covered with canvas to protect the animal from the sun and wind. The animal was surrounded by ice to prevent overheating. When it arrived at the zoo, clinical procedures were begun to guarantee its survival. Daily, for 8 days, it was chemically restrained for treatment using the same dose as for capture. Treatment included 8 days of antibiotic therapy and administration of B-complex vitamin and vitamin C, 4 days of anti-inflammatory treatment with flunixin meglumine (Banamine) with decreasing doses, and anthelmintic therapy with intramuscular and oral doses. (R.V. Monteiro, personal communication, 1999).

Although fish was offered every day, the animal only started to eat 45 days later. This species may fast in the wild for approximately 8 weeks (V. Ramos, Jr., personal communication, 1999). On arrival the animal had a corneal opacity, which disappeared after the treatment.

Pinniped Medicine

Pinnipeds may exhibit a period of depression and refuse food for weeks after initial capture.[42] Adults may show extreme alarm or aggression. Appetite may be stimulated by administration of B-complex vitamins with B_{12} administered intramuscularly if the animal does not start eating on its own.

Two common problems encountered in pinnipeds after shipment, are overheating and exhaustion. The place in which they are initially housed should be kept at or below 18°C. To quickly lower the temperature of severely hyperthermic arrivals, use sponge or hose baths or enemas with cool water.

Dehydration and electrolyte imbalance are commonly associated metabolic problems observed in newly acquired pinnipeds. Pinnipeds must feed regularly to ensure adequate intake.[22] Adult animals may go for longer periods without feeding and partially maintain proper hydration by the production of metabolic water as a by-product of lipid oxidation; however, blubber stores in a young orphan pinniped may not be adequate to provide proper hydration, hence, stomach tubing of fluids is sometimes necessary for newly acquired young pinnipeds.

Antibiotics and other drugs are easily administered to pinnipeds within food. Intramuscular injections are carried out using needles of sufficient length (7.5 cm or greater) to ensure disposition of the drug into the muscle mass. At the present time, few drug dosages have been established for pinnipeds, so the dosage rates recommended for dogs are used.[49]

Infectious Diseases

Staphylococcosis (*Staphylococcus aureus*) is characterized by subcutaneous abscessation followed by acute systemic infection. Depression, emaciation, dyspnea, emesis, and catarrhal-hemorrhagic enteritis are associated with the syndrome. Treatment should include the administration of penicillin or other antibiotics effective against the specific organism.[49]

Pasteurellosis (*Pasteurella multocida*) is characterized by sudden onset, anorexia, elevated rectal temperature, dyspnea, septicemia, and an elevated white blood cell (WBC) count. Treatment of uncomplicated cases of pasteurellosis respond well to oxytetracyclines (Terramycin) administered intramuscularly at the rate of 11 mg/kg of body weight daily. When pyothorax or peritonitis is present, drainage of the accumulated exudate is necessary. The thoracic or abdominal cavities should then be instilled with the appropriate antibiotic in aqueous solution plus proteolytic enzymes.[49]

Botulism (*Clostridium botulinum*) signs include anorexia, total paralysis, diarrhea, and sudden death.[48] Definitive diagnosis is made by injecting mice with serum from the suspect animals. Treatment of a suspect botulism case is largely supportive. Commercial antitoxins or toxoids for vaccination in endemic areas are readily available. Gastric lavage may be helpful if performed immediately after ingestion of suspect food.

The characteristic clinical signs of coccidioidomycosis (*Coccidioides immitis*) are anorexia, dehydration, and general debilitation in the chronic form and respiratory distress in the acute form.

The clinical signs of histoplasmosis(*Histoplasma capsulatum*) include a mucopurulent nasal discharge for 2 to 3 months, emaciation, and pronounced dyspnea.

Bacterial folliculitis and pustular dermatitis are seen in pinnipeds maintained under poor sanitary conditions. Treatment should include the administration of 25,000 IU of vitamin A daily and an appropriate long-acting broad spectrum antibiotic. Local treatment with antibiotic creams is useful.[49]

Dentoalveolar abscesses associated with the canine teeth have been reported in the California sea lion and walrus.[1,22,41] Purulent dental disease has also been observed in pinnipeds with traumatized oral cavities, exposing the pulp cavity of a tooth during capture or during confinement. The oral administration of penicillin or tetracycline is indicated, and, if elevation and extraction are necessary, the techniques for extracting pinnipd teeth are the same as those described for dogs. Clinical signs of these abscesses in the walrus include anorexia and swelling of the maxilla.[49] An animal being treated for this condition will probably eat as soon as the abscess is drained. The lesion should be flushed with antibiotics, and parenteral antibiotics should be administered.

Corneal opacities in pinnipeds are fairly common and have been reported in many California sea lions and harbor seals. Three etiologies have been proposed:[49] (1) salt deficiencies, (2) trauma to the cornea with a resultant ulcer, and (3) contaminated water in which the bacterial counts are high. The specific sign is a cloudy cornea, which sometimes has an ulceration. Most pinnipeds will keep the eye closed entirely when the lesion first appears. Treatment consists of instillation of antibiotic ointments into the eye daily and parenteral administration of antibiotics and high levels of vitamin A. Saltwater baths are also helpful.

Influenza virus, septicemia, candidiasis, histoplasmosis, dermatophytosis, botryomycosis, aspergillosis, and actinomycosis have been reported in pinnipeds and/or cetaceans.[21]

Parasites

Because of their association with water, mosquitoes of many genera often affect captive pinnipeds. Control consists of housing the pinniped in a mosquito-proof enclosure at night. Organophosphate impregnated fly strips (Vapona) may be hung in the enclosures to kill any insects that may accidentally enter the enclosure.[49]

Fecal flotation using a disposable fecal diagnostic system (Ovatector) is employed to identify parasites. Animals with feces positive for gastric nematodes should be treated with thiabendazole (Omnizole) at 45 mg/kg for 2 days or with fenbendazole (Panacur) at 10 mg/kg for 3 days. Treatment should be repeated in 2 weeks, if the parasites persist. Animals with feces positive for tapeworms, and liver flukes should be treated with praziquantel (Droncit) at 5–7 mg/kg for 1 day.[14]

Biological Data for Cetaceans

The normal deep core temperature of cetaceans is usually taken by rectal thermometer. Glass thermometers should not be used because the squirming animal may break it. A flexible thermometer should be inserted 30 cm into the rectum to obtain a true core temperature. Readings of 35.5–37.2°C are considered normal.

Heart rate may be determined by palpation or auscultation. The rate varies normally from 50 to 150 beats per minute.[36,38,43,49] The bradycardia occurs during the normal period of respiratory apnea,[36,38,43,49] and the relative tachycardia occurs during inspiration.[38,43] Since heart rate varies so much, it has little clinical value except during anesthesia.

Blood sampling for hematology or serum chemistry may be performed in many ways. Venipuncture may be made in the ventral or dorsal surface of the tail flukes. Because there is a depression or contrasting shade indicating the location of the vessel running from the base of the fluke toward the tip, this method is relatively easy. The brachial vessels of the pectoral fins may also be used to collect blood. The vessels are located at the dorsocranial border of the fin in a depression that follows its course. The puncture is made with a 20-gauge, 1-inch needle, perpendicular to the long axis of the fin. An assistant should hold the fin steady and apply digital pressure in the proximal aspect of the groove. Twenty to thirty milliliters of blood may be collected in this manner.[31]

Anemia as a result of bone marrow depression has been reported in the bottlenosed dolphin. A bone marrow biopsy may be collected to evaluate marrow activity. The fifth and eighth vertebrae cranial to the flukes are the sites of choice for bone marrow biopsy. An 11- to 13-gauge bone marrow biopsy needle 8.5 cm long is preferred.[45]

Urine may be collected by catheterization. For the female a simple canine or human catheter will work well.[38] The urethral orifice is located at the caudal aspect of the clitoris. A number 12 French catheter greater than 20 cm long is ideal for the collection of urine.[45] For the male, a 50-cm number 6–8 French canine catheter is recommended because of the sigmoid flexure.[38] See Tables 30.3 and 30.4 for urinalysis values.

Translocation

The attending veterinarian may monitor a cetacean's physical state during transport by keeping a record of the animal's respiratory rate, heart rate, and rectal temperature. During such restraint the heart rate will double its normal rate and a slight weight loss attributed to dehydration may be expected.[50] Hyperthermia must be anticipated during transit or when a cetacean is otherwise beached. Since a cetacean's skin is devoid of sweat glands, it must be kept moist.

Cetacean Medicine

Dosage based on uptake and excretion rates for selected antibiotics are as follows:[49] penicillin (IM),

TABLE 30.4. Urine values of bottlenosed dolphins

	Atlantic Bottlenosed Dolphin	Pacific Bottlenosed Dolphin
Specific gravity	1.020–1.055	1.030–1.065
Albumin	Negative	Negative
Glucose	Negative	Negative
PH	Acid	Acid
Color	Clear to Straw	Clear to straw
Sodium mEq/L	92–287	95–300
Chloride mEq/L	86–298	95–300
Potassium mEq/L	64–118	47–145

Source: See reference 40.

8800 IU/kg/24 h; gentamicin (IM), 5 mg/kg/12 h; tetracyclines (oral),88 mg/kg/4 h; chloramphenicol (oral), 44 mg/kg, then 22 mg/kg/12 h; and doxycycline (oral), 44 mg/kg, then 22 mg/kg/12 h.

Infectious Diseases

Characteristic clinical signs of erysipelas include anorexia, general weakness, and black tarry feces. The disease may be contracted by dolphins through contaminated food fish or blood-sucking flies. Care must be taken by an affected animal's handlers, because the disease is transmissible to humans. Treatment of erysipelas should include the administration of penicillin[49] for a minimum of 72 hours.[36] Response to treatment appears to be variable.[49] Commercial bacterins (both oral and parenteral) have been used to immunize dolphins against erysipelas.

Pseudomonas aeruginosa has been reported to cause bronchopneumonia, dermatitis, osteomyelitis, and septicemia in bottlenosed dolphins. Clinical signs of pseudomonas infections depend upon the location of the lesion. Necrosis and ulceration of the skin, respiratory distress, and depression have all been reported. Treatment should include the parenteral administration of antibiotics that show sensitivity to the organism; however, variable results have been reported.[49]

Tuberculosis treatment, if attempted, should consist of isoniazid given orally.

Actinomycotic mycetoma has been reported in the bottlenosed dolphin. Clinical signs included ulcerative lesions in the mouth and snout. Diagnosis is made by culturing the organism. Treatment should include topical application of sulfas to the lesion and parenteral administration of sulfadiazine, novobiocin, or oxytetracyline.

Cutaneous candidiasis (*Candida albicans*) in cetaceans may be diagnosed by culturing the organism from the skin ulcerations. Treatment with nystatin did not appear to be effective; however, levamisole phosphate injections intramuscularly cleared the lesions.[35]

Dolphin poxvirus has recently been identified to be the putative etiology of the peculiar skin lesions of porpoises known as "tattoos." They consist of discrete black lines of hyperpigmentation in the epidermis, and they form closed circles and other shapes that have the appearance of tattoo marks. They occur most commonly in the skin of the dorsal body, on the flippers, flukes, and dorsal fin.[21]

Skin squamous cell carcinoma (SCC) has occurred in dolphins,[21] and recently a sublingual SCC has been reported in a 22-year-old *T. truncatus* (M.S. Renner et al., unpublished data, 1999). It looks like a nonhealing sublingual mucosal ulcer.

Parasites

Ominous signs of internal parasites are loose stool, blood in the stool, or other digestive disturbances. Diagnosis is made by finding the parasite ova on fecal flotations. Treatment of nematodes may be accomplished by the oral administration of thiabendazole administered at the rate of 73 mg/kg body weight (no toxicity has been reported) and piperazine administered at the rate of 8–11 mg/kg. Dosages of 20 mg/kg produce hyperventilation, incoordination, and loss of swimming ability.[45]

Treatment of tapeworms can be carried out by administration of niclosamide orally at the rate of 110 mg/kg of body weight, with no toxicity reported. Trematode infestation of the gastrointestinal tract, liver, and nasal passages can be treated by oral administration of Lorothidol at 20 mg/kg; however, this drug can be toxic and should not be used routinely.

External parasites are occasionally found on newly acquired cetaceans. Barnacles (Cirripedia) and copepods may cause severe local skin lesions, secondary subacute local skin lesions, and secondary subcutaneous infections.[38] "Whale lice" (*Isocyamus delpini*) are among the more common offenders. These parasites cling to the host by sharp claws and feed on the skin;[21]

they are usually found clinging to old wounds, genital folds, and lips. They may be removed manually and the resulting wound treated with gentian violet in alcohol.

In unshaded areas or shallow pools, sunburn may occur on the dorsal area of cetaceans,[27,28] resulting in the formation of painful fissures around the blowhole and dorsal fin. Local application of zinc oxide ointment combined with provision of shade will prevent such problems from reoccurring.

Bite wounds in cetaceans appear as linear, parallel lacerations ("rake" marks) within or through the skin. Treatment of severe lacerations consists of the intramuscular administration of broad spectrum antibiotics to prevent septicemic complications, plus local treatment with methyl violet in an alcohol base. [34]

Subcutaneous cellulitis, abscessation, and ulcer formation often follow superficial wounds or chronic disease. *Streptococcus* spp. are common responsible agents. These lesions usually respond well to systemic treatment with chloromycetin. [34]

Corneal opacities and epidermal sloughing may indicate that the salinity of the water is too low. It is not desirable to have the sodium chloride content drop below 2.5%.[49] Increasing the sodium chloride level will usually reverse the clinical problem.

Mastitis occurs in cetaceans. The disease may be associated with parasitic infestation (*Placentoma* spp.) or bacterial infection (*Edwardsiella tarda*).[49] Treatment should include parenteral antibiotic therapy.

Dehydration, severe hemorrhage, or shock in cetaceans may be treated by administration of fluids. A catheter placed in the brachiocephalic vein or inserted intraperitoneally[IP] may be used to deliver the fluid replacement. Fluids may be administered at the rate of 4 L/h IP and somewhat slower intravenously.[49]

To treat respiratory disease intratracheal therapy is useful.[45] A 3-inch, 18-gauge needle is used for the bottlenosed dolphin. A ventral midline approach is employed. When the needle is in the trachea, air may be aspirated or a cough may be elicited by injecting several milliliters of saline. Gentamicin and cephaloridine have been administered intratracheally at the rate of 0.5 mg/kg and 7 mg/kg, respectively. The volume of the antibiotics is extended to 6–10 mL with sterile water. Aerosol therapy for infections of the upper respiratory tract and blowhole have been described.[45] A mist sprayer is employed to nebulize dilute antibiotic solutions directly into the upper respiratory tract.

Blowhole infections (*Candida albicans, Steptococcus* spp., and *Pseudomonas* spp.) are relatively common in the pilot whale and dolphins. For treatment levamisole phosphate injections should be used.[35]

During capture, cetaceans sometimes aspirate seawater with or without debris. Inhalation pneumonia caused by a number of etiologic agents appears to be the most common disease of the respiratory system in captivity.

If pneumonia is diagnosed, large doses of parenteral penicillin-streptomycin or chloromycetin are indicated. Intratracheal antibiotic therapy should be considered.[49] Supportive treatment should be administered according to the needs of the individual. It must be remembered that if the animal does not eat, it may become dehydrated; therefore, if a cetacean is anorexic, copious amounts of 5% dextrose in lactated Ringer's solution may be administered orally. (If prolonged fluid therapy is required, B-complex vitamins and an amino acid solution should be added to minimize weight loss).

Foreign bodies, usually sand or small stones, are often found in the fore stomachs of wild dolphins. The captive dolphin often takes in and swallows irregularly shaped objects that do not pass through the digestive tract. Some objects, such as rubber balls and footballs, have been left in the animal with no deleterious effects, but one bottlenose dolphin died from a gastric impaction produced by seven cotton gloves.[38]

When it is considered necessary to remove an object because of poor appetite or constipation, mineral oil (administered via stomach tube) should be tried first.[38] In small dolphins the use of an oral speculum has facilitated the manual removal of objects from the fore stomach.[38] Gastrotomy may be performed if necessary.

To perform a gastric lavage, an equine stomach tube should be passed through the esophagus into the stomach.[45] To collect gastric secretions and ingesta, suction may be used. Lavage techniques may be used to backflush from the stomach small, uniformly shaped foreign bodies.

Gastroscopy is now a routine diagnostic procedure in cetaceans.[45] Gastric contents and lesions may be visualized by employing a flexible fiber-optic colonoscope. The entire first stomach can be examined, and biopsy of the stomach lining, collection of gastric fluids for culture, and grasping of tissue or foreign objects can be performed.

The authors performed a gastroscopic search in a bottlenose dolphin that had swallowed a blinker used for training. Because it was extremely difficult to localize the object, a gastric lavage was performed to make the animal vomit it.

To perform a gastric palpation in a cetacean, its mouth should be opened with the help of an oral speculum.[45] The operator's hand (wearing a disposable obstetrical glove, well lubricated) should be passed directly into the stomach where the lining may be palpated or foreign objects grasped and removed. Clinical signs of gastric ulceration in a cetacean are depression, anorexia, and a contracted or "tucked up" abdomen. Diagnosis of gastric ulceration may be confirmed by gastroscopy, radiography, or by finding mammalian erythrocytes and leukocytes in the stomach contents. Treatment of gastric ulceration in cetaceans is symptomatic; e.g, magnesium hydroxide is administered as an antacid and the fish fed to the animal are ground into a fine slurry in a blender.[49] The clinical sign for a general enteric problem is a gas-laden, discolored stool.[36,38]

Bottlenosed dolphins may be protected against *Clostridium perfringens* enterotoxemia by vaccination with 5 mL of *C. perfringens* Type D bacterin toxoid; good titer responses are monitored by 7 days after the inoculation.

Hepatitis of a nutritional (i.e., choline or selenium deficiency) or toxic origin is a frequent finding in captive and free-ranging cetaceans. Clinical signs of hepatitis are emaciation, anorexia, and icterus. Diagnosis may be confirmed by liver biopsy techniques and indocyanine green dye retention tests.[45] Liver biopsy is performed with a 12.5-cm Manghini biopsy needle. Treatment of hepatitis is symptomatic and is based on enzyme replacement therapy and supportive therapy with parenteral and oral administration of B-complex vitamins and ascorbic acid.

Diabetes mellitus has been observed in cetaceans.[49] The disease was successfully controlled with insulin. Chromium should be administered orally to cetaceans with carbohydrate and glucose metabolic problems at a rate of 200–1000 µg per day.

Pancreatitis in the form of acute or chronic fibrosis commonly occurs in older cetaceans.[49] Characteristic clinical signs of pancreatitis include progressively pale, greasy feces and an increased serum amylase value. Treatment is symptomatic and should be aimed at enzyme replacement supported by the administration of selenium, zinc, B-complex vitamins, and ascorbic acid.

A trematode of the family Nasitrematidae has been observed adhered to the round window of the inner ear of a bottlenose dolphin with hearing loss and changes in acoustic behavior.[49] When the parasite was removed, the dolphin seemed to recover its hearing range.

Clinical signs of cerebral abscessation in cetaceans include incoordination, circling, loss of equilibrium, and sudden stranding.[49] When a bottlenose dolphin with a brain abscess was taken to deep water, it immediately swam back to the beach. To treat any neurological disease the veterinarian should lower the tank's water level to prevent drowning and administer chloromycetin.

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REFERENCES

1. Bartsch, R.C.; and Fruch, R.J. 1971. Alveolitis and pulpitis of canine tooth in a walrus. Journal of the American Veterinary Medical Association 159:575–577
2. Boness, D.J. 1996. Animal learning and husbandry training. In D.G. Kleiman, M.E. Allen, C.V. Thompson, and S. Lumpkin, eds., Wild Mammals in Captivity: Principles and Techniques. Chicago, University of Chicago Press, pp. 231–242.
3. Borobia, M.; and Rosas, F.C.W. 1991. Tucuxi, *Sotalia fluviatilis* (Gervais, 1853). In H.L. Capozzo and M. Junin, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 36–41.
4. Capozzo, H.L. 1991. Lobo marino peletero sudamericano, Arctocephalus australis (Zimmermann, 1783) [The fur seal, Arctocephalus australis (Zimmermann, 1783)]. In H.L. Capozzo and M. Junin, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 171–174.
5. Cappozo, H.L.; Campagna, C.; and Monserrat, J. 1991. Sexual dimorphism in newborn southern sea lions. Marine Mammal Science 7(4):385–394.
6. Corcuera, J.; and Monzn, F. 1991. Franciscana, *Pontoporia blainvillei* (Gervais and d'Orbigny, 1844). In H.L. Capozzo and M. Junin, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 16–22.
7. Cornell, L. 1986. Capture, transportation, restraint, and marking. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 764–770.
8. David, J.H.M.; Meyer, M.A.; and Best, P.B. 1990. The capture, handling and marking of free-ranging adult South African (Cape) fur seal. South African Journal of Wildlife Research 20:5–8.
9. Decopas, F.; Hart, J.S.; and Fisher, H.D. 1971. Sea water drinking and a water flux in a starved and fed harbor seals. Canadian Journal of Physiology and Pharmacology 49:53.
10. Dierauf, L.A. 1990. Pinniped husbandry. In L.A. Dierauf, ed., Handbook of Marine Mammal Medicine. Boca Raton, Florida. CRC Press, pp. 553–590.
11. Dierauf, L.A.; Vandenbrock, D.; Roletto, J.; Koski, M.; Amaya, L.; and Gage, L.J. 1985. An epizootic of leptospirosis in California sea lions. Journal of the American Veterinary Medical Association 187:1145–1148.

12 Erickson, A.W.; and Bester, M.N. 1993. Immobilization and capture. In R.M. Laws, ed., Antarctic Seals Research Methods and Techniques. Cambridge, Cambridge University Press, pp. 46–88.
13. Fowler, M.E. Ed. 1986. Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, p. 781.
14. Gage, L.J.; Gerber, J.A.; Smith, D.M.; and Morgan, L.E. 1993. Rehabilitation and treatment success rate of California sea lions (*Zalophus californianus*) and northern fur seals (*Callorhinus ursinus*) stranded along the central and northern California coast, 1984–1990. Journal of Zoo and Wildlife Medicine 24(1):41–47.
15. Gentry, R.L.; and Holt, J.R. 1982. Equipment and Techniques for Handling Northern Fur Seals. NOAA Technical Report NMFS 758. Washington, D.C., U.S. Dept. of Commerce, pp. 1–15.
16. Gentry, R.L.; and Johnson, J.H. 1978. Physical restraint for immobilizing fur seals. Journal of Wildlife Management 42:944–946.
17. Geraci, J.R.; and Sweeney, J. 1986. Clinical techniques. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 771–777.
18. Geraci, J.R. 1974. Thiamine deficiency in seals and recommendations for its prevention. Journal of the American Veterinary Medical Association 165:801–803.
19. Geraci, J.R. 1981. Dietary disorders in marine mammals: Synthesis and new findings. Journal of the American Veterinary Medical Association 179:1183–1191.
20. Geraci, J.R. 1986. Nutrition and nutritional disorders. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 771–777.
21. Howard, E.B. 1983. Pathobiology of Marine Mammal Diseases. Boca Raton, Florida, CRC Press, Vol. 1, pp. 1–233; Vol. 2, pp. 1–227.
22. Hubbard, R.C. 1968. Husbandry and laboratory care of pinnipeds. In R. Harrison, r. Hubbard, R. Peterson, C. Rice, and R. Shusterman, eds., The Behavior and Physiology of Pinnipeds. New York, Appleton-Century-Crofts, pp. 299–358.
23. Hubbard, R.C. 1969. Chemotherapy in captive marine mammals. Bulletin of the Wildlife Disease Association 5:218–230.
24. Iñiguez, M. 1991. Orca, *Orcinus orca* (Linnaeus, 1758). In H.L. Capozzo and M. Junin, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 92–95.
25. Irving, L.; Fisher, K.C.; and McIntosh, F.C. 1935. The water balance of a marine mammal, the (harbor) seal. Journal of Cellular and Comparative Physiology 6:387.
26. Jasmin, A .M.; Powell, C.P.; and Baucom, J.N. 1972. Actinomycotic mycetoma in the Bottlenose Dolphin (*Tursiops truncatus*) due to *Nocardia paraguayensis*. Veterinary Medicine of Small Animal Clinics 67:542.
27. Kenny, D.W. 1965. The gentle art of capturing whales. Modern.. Veterinary Practice 46:27–32.
28. Layne, J.N.; and Caldwell, D.K. 1964. Behavior of the Amazon dolphin, *Inia geoffrensis* (Blainville) in captivity. Zoologica 49:81–108.
29. Loureiro, J.D. 1988. Nacimiento en cautiverio de *Otaria flavescens* [The birth in captivity of *Otaria flavescens*]. In Abstracts of the 3° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Montevideo, Universidad Austral de Chile, p. 13.
30. Martínez, P.E.; Colares, E.P.; Muelbert, M.M.C.; Robaldo, R.B.; and Bianchini, A. 1998. Anestesia de elefante marino del sur, *Mirounga leonina,* utilizando Tiletamine-Zolazepam [Anesthesia of southern elephant seal, *Mirounga leonina,* using Tiletamina-Zolazepan]. In Proceedings Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 124.
31. Medway, W.; and Geraci, J.R. 1964. Hematology of the bottlenosed dolphin (*Tursiops truncatus*). American Journal of Physiology 207:1367–1370.
32. Medway, W.; Geraci, J.R.; and Klein, L.V. 1970. Hematologic response to administration of a corticosteroid in the bottlenosed dolphin (*Tursiops truncatus*). Journal of the American Veterinary Medical Association 157:563–565.
33. Mellen, J.D.; and Ellis, S. 1996. Animal learning and husbandry training. In D.G. Kleiman, M.E. Allen, C.V. Thompson, and S. Lumpkin, eds., Wild Mammals in Captivity: Principles and Techniques. Chicago, University of Chicago Press, pp. 88–99.
34. Miller, R.M.; and Ridgway, S.H. 1963. Clinical experiences with dolphins and whales. Small Animal Clinic 3:189–193.
35. Nakeeb, S.; Targowski, S.P.; and Spotte, S. 1977. Chronic cutaneous candidiasis in bottlenosed dolphins. Journal of the American Veterinary Medical Association 171:961–965.
36. Norris, K.S. 1966. Whales, Dolphins and Porpoises. Berkeley, University of California Press, pp. 278–754.
37. Oftedal, O.T.; and Boness, D.J. 1983. Considerations in the use of fish as food. In T.P. Meehan and M.E. Allen, eds., Proceedings of the 3rd Annual Dr. Scholl Conference on the Nutrition of Captive Wild Animals. Chicago: Lincoln Park Zoological Society, 149–161.
38. Ridgway, S.H. 1965. Medical care of marine mammals. Journal of the American Veterinary Medical Association 147:1077–1085.
39. Ridgway, S.H. 1966. Classifying small dolphinids according to brain weight. Psychology and Science 6:491.
40. Ridgway, S.H. 1972. Homeostasis in the aquatic environment. In S.H. Ridgway, ed., Mammals of the Sea: Biology and Medicine. Springfield, Illinois, Charles C. Thomas, p. 631.
41. Rosen, M.N. 1971. Pasteurellosis. In J. W. Davis, ed., Infectious Diseases of Wild Mammals. Ames, Iowa, Iowa State University Press, pp. 214–232.
42. Sedgwick, C.J.; and Costa, A.L. 1968. Considerations of emotional patterns in M-99 immobilization. Paper presented at the Annual Meeting of the American Association of Zoological Parks and Aquariums.
43. Slijper, E.J. 1958. Whales. New York, Basic Books.
44. Stirling, I. 1966. A technique for handling live seals. Journal of Mammalogy 47:543–544.

45. Sweeney, J.C.; and Ridgway, S.H. 1975. Procedures for the clinical management of small cetaceans. Journal of the American Veterinary Medical Association 167:540–545.
46. Vergani, D.F. 1985 Comparative Study of Population of the Southern Elephant Seal, *Mirounga leonina* (Linne, 1758) and Its Methodolgy. Direccion Nacional del Antartico Publication No. 15. Buenos Aires, Instituto Antartico Argentino.
47. Vergani, D.F.; Spairani, H.J.; and Aguire, C.A. 1986. Immobilization of Crabeater Seals, *Lobodon carcinophagus,* with the Use of Xylazine Hydrochloride at 25 De Mayo Island (Antartica) and Identification of Polymorphism in Transferrins. Direccion Nacional del Antartico Publication No. 317., Buenos Aires, Instituto Antartico Argentino.
48. Wagner, J.E.; and Mann, P.C. 1978. Botulism in California sea lions—A case report. Journal of Zoo Animal Medicine 9:142–145.
49. Wallach, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals: Medical and Surgical Management. Philadelphia, W.B. Saunders, pp. 686–725, 726–759.
50. Wilkie, D.W.; Bell, G.B.; and Coles, J.S. 1968. A method of dolphin transport and its physiological evaluation. International Zoo Yearbook 8:198–202.
51. Caldwell, M.C.; Caldwell, D.K.; and Brill, R.L. 1989. *Inia geoffrensis* in captivity in the United States. In W.F. Penin, R.L. Brownell, Z. Kaiya, and L. Jiankang, eds., Biology and Conservation of the River Dolphins. Occasional Paper of the IUCN Species Survival Commission. No. 3, Allen Press, Lawrence, Kansas, p. 35–41.

31
Order Sirenia (Manatees, Dugongs, Sea Cows)

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BIOLOGY

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INTRODUCTION

The order Sirenia contains what is commonly known as the sea cows, including the three living species of manatees and the dugong. They are essentially herbivores, and like cetaceans, they are exclusively aquatic mammals.[22] All sirenians are nonruminant with a rich microflora in the intestines that allows them to digest cellulose.[24]

The orders Sirenia, Proboscidea, Hyracoidea, and Tubulidentata together form what is sometimes called the subungulates. The amino acid sequence of the eye-lense protein of the subungulates suggests that these animals are closely related.[11] Sirenian ancestors date from the Eocene epoch, about 55 million years ago, and reached their peak in diversity during the Oligocene and Miocene epochs.[23] The oldest known sirenian fossil was found in Central America. The first sirenians lacked the extra teeth and tooth replacement characteristics of the modern sirenians, which is an adaptation to the abrasive plants on which they feed.

The living sirenians are represented by only two families, Dugongidae (dugongs) and Trichechidae (manatees). Apparently the dugongids were the first sirenians to occupy the Caribbean region. By the early Pliocene epoch, some trichechids invaded the Caribbean whereas others were isolated in the Amazon basin after the origin of the Andes, giving rise to the Amazonian manatee. In the Caribbean the trichechids were more successful, expelling the dugongids from this region by competition and invading South America during the same period.[23]

Different from most other mammals, which have seven cervical vertebrae, the manatees have only six cervical vertebrae. The only other mammal with six cervical vertebrae is the two-toed sloth.[23] Another peculiarity of manatees is the absence of marrow cavities in the ribs and long bones, which is probably a result of their low metabolic rate or retarded development.[9] Marrow for producing red blood cells is basically found only within the vertebrae and sternum. Red blood cells may also be produced by the liver.[23] The lack of marrow provides dense bones that help to keep manatees submerged.

Living manatees have premolar and molar teeth only, which are continuously replaced by new teeth sprouting at the rear of the row and moving forward. The worn teeth at the front of the row drop out, and the bony tissue separating the tooth sockets continuously breaks down and re-forms to allow the new teeth to move forward at a rate of 1 or 2 mm per month. This process occurs throughout the manatee's life.[4]

Manatees have a very low reproductive rate, producing only one calf every 2.5–5 years.[16] Gestation is estimated to last 12–14 months,[9,2,16] and sexual maturity is believed to occur between 5 and 10 years.[17]

TAXONOMY

The order Sirenia has its origin in the Greek legend of mermaids (the mythical siren). The modern sirenians are classified as follows:

Order Sirenia
- Family Trichechidae
  - *Trichechus manatus* (West Indian manatee)
  - *Trichechus inunguis* (Amazonian manatee)
  - *Trichechus senegalensis* (West African manatee)
- Family Dugongidae
  - *Dugong dugon* (dugong)
  - *Hydrodamalis gigas* (extinct Steller's sea cow)

The South American manatees, *T. inunguis* and *T. manatus manatus,* are distinguished by the absence of nails on the flippers, reduced number of dorsal vertebrae, smaller and more complex molars, and a thickened supraoccipital observed in *T. inunguis*.[7,26] A larger number of diploid chromosomes is also found in *T. inunguis* (2n = 56) when compared with *T. manatus* (2n = 48).[26] Among the sirenians, the Amazonian manatee is the smallest, reaching up to 3 m in length and a maximum weight of 450 kg.[25] This species usually has a white patch on the belly, although it may not be present in some individuals.[26] On the other hand, *T. manatus manatus* may reach more than 3.5 m and weigh up to 1000 kg.[23]

A recent study on mtDNA of manatees showed that *T. manatus* and *T. inunguis* shared a common ancestor more recently than was previously suspected and that the Florida manatee seems to be a recent derivative of the West Indies populations.[7] However, although the genetic results do not coincide completely with what was previously indicated by morphological and osteological studies, the mtDNA data are not sufficient in themselves to resolve the taxonomic questions of manatees, and further investigations are needed on this subject.[7]

DISTRIBUTION AND HABITAT

All modern sirenians have a tropical or subtropical distribution. Their low metabolic rate, about 36% of that predicted for a terrestrial mammal of similar size, and their apparent inability to reduce heat loss (thermal lability) make survival in water temperatures below 22°C impossible for long periods.[6] Their occurrence in local habitats is basically limited to calm waters and by the availability of food.[3] The low metabolic rate, on the other hand, allows manatees to dive for longer periods than would otherwise be possible, as they lack some of the physiological adaptations of cetaceans and pinnipeds for diving (see Cetacea and Pinnipedia, Chapter 30).

The Amazonian manatee (*T. inunguis*) is an exclusively freshwater species distributed in the Amazon basin.[25,26] The Antillean manatee (*T. manatus manatus*) is a marine and estuarine species, distributed exclusively on the Atlantic side, from Mexico to the northeast of the South American continent.[23,26] The present distribution of the Antillean manatee is smaller than the historic range, and it is probably not continuous along the coast.[13]

EXPLOITATION

The Amazonian manatee is part of the folklore and culture of the Amazon region and has been hunted for subsistence food and for medicinal purposes by indigenous people for hundreds of years. Commercial exploitation apparently started around 1542.[3] In the beginning, the animals were hunted for meat only. Between 1935 to 1954, the hide of the Amazonian manatee was used to manufacture machine belts, hoses, and other items that required resistant leather, which is found in manatees.[3,26] From 1955 on, with the introduction of synthetic products on the market, the leather industry was reduced. Presently, Amazonian manatees are captured by riverine people for local consumption, although there is still a limited illegal market of manatee meat in the Amazon.[26] In Ecuador, the Siona Indians have been hunting the Amazonian manatee for hundreds of years, with the skill passed from generation to generation.[31] However, the number of Indians are decreasing and their impact on the manatee populations does not seem to be a problem. Today, the main problems faced by manatees in Ecuador are oil exploration and the construction of new roads bringing settlers to the region, causing a demand for animal protein, and a consequent decline of manatee populations.[31] These also seem to be common problems throughout the Amazon region.

In the beginning, the Antillean manatee was also hunted by indigenous tribes for meat and homemade medicine. A more irrational slaughter occurred with the European colonization of South America in the 17th century, which, besides the meat, also killed manatees for leather.[13] Nowadays, it seems that no, or extremely little, commercialization occurs with this species on the northeast coast of Brazil.[12] However, because of exploitation and habitat destruction, the population of the Antillean manatee off the northeast coast of Brazil has been estimated to be approximately only 400 individuals.[12]

WILDLIFE POPULATION STUDIES

The timid behavior of manatees in exposing very little of their bodies when they breath (especially the Amazonian species), and the turbidity of the waters in their preferred habitat (coastal or interior waters) in South America, make the studies of wild populations limited. Telemetry seems to be the best way to study wild manatee populations.

Radiotelemetry studies have been conducted with wild Amazonian manatees since the early 1980s.[21] A total of 42 manatees were radio-marked with very high frequency (VHF) transmitters and released in a hydroelectric dam in the central Brazilian Amazon with the aim of using manatees as biological controllers of aquatic macrophytes.[27] More recently, telemetry studies using VHF and satellite transmitters are in development in the Sustainable Development Reserve of Mamirauá, in Brazil.[29] According to these studies, during the dry season in the Amazon, the manatees leave the "várzea" lakes and move to the main rivers. Apparently this movement starts when the water level declines about 2 m. The animals at this time move at a speed of about 1.35 km/h. They return to the lakes when the water levels are at least 6.5 m higher than its lowest level.[18]

Telemetry studies are also in development on the northeast coast of Brazil with the aim of determining the movements, behavior, and social interactions of the Antillean manatee.[14] In contrast to the studies of the Amazonian manatees in Mamirauá Reserve, the telemetry studies of the Antillean manatees off the northeast coast of Brazil are being carried out with manatees rehabilitated in captivity and released into the wild. Seven manatees have already been reintroduced, of which two have disappeared from the monitor and one died three days after being released, probably killed by predatory hunting (C.L. Parente, personal communication, 1999). Of the knowledge provided by telemetry about the dynamics of manatee populations, perhaps the most useful information is that concerning their habitat. The knowledge of the preferred habitats of animals so difficult to follow in the wild may permit the conservation of such areas and allow manatee survival.

CONSERVATION PROGRAMS

Steller's sea cow (*Hydrodamalis gigas*) and the Caribbean monk seal (*Monachus tropicalis*) are the only two species of marine mammals that have become extinct in modern times as a result of human hunting.[22] The South American sirenians (the Amazonian and Antillean manatees) are classified by the IUCN [10] as "vulnerable." However, in Brazil both species are being cited as "endangered."[1] In that country, research and conservation programs with the Amazonian manatee have been in development since at least the 1970s.[26] Research and conservation programs concerning the Antillean manatee off the coast of Brazil started a bit later (1980). The hunting of manatees continued without any regulation in Brazil until 1967, when the Law for the Protection of Fauna was declared (Law No. 5197/67).

Manatees are also protected by law in Peru and Colombia. In Ecuador they are protected only inside national parks.[3,31,25] In Ecuador and Peru, they seem to be highly endangered. The hunting and commercialization of manatee meat in those countries has led to a precarious status.[31] An international agreement among the South American countries where manatees occur is urgently needed to avoid the continuing depletion of manatee populations.[31] Even though they have been protected by law in many countries, there is no, or minimal, law enforcement.[28]

In 1996, the National Plan for Recovery and Conservation of the Manatee was created in Colombia. For the success of the plan, researchers are conscious that the socioeconomic and cultural status of the human communities in the areas of manatee occurrence have to be taken into account.[8] A primary threat faced by manatees is the environmental damage imposed by anthropic activities.

In 1974, the National Institute of Amazonian Research (INPA) in Manaus, Brazil, started a series of studies of the Amazonian manatee, involving its biology, ecology, physiology, and management. The maintenance of Amazonian manatees in captivity at INPA has been successful for 25 years. Many of the animals arrive at the institute as orphan calves because their mothers were victims of illegal hunting and are raised in captivity with artificial milk.[26] More than 40 manatees have been successfully rehabilitated at INPA. In April 1998, after an improvement of the diet of the adult captive manatees, the researchers at INPA recorded the first birth of an Amazonian manatee conceived in captivity,[30] which is undoubtedly an important step toward the conservation of the species.

In 1990, the Sustainable Development Reserve of Mamirauá was created in Brazil. This is the biggest

Conservation Unit in that country (1,124,000 ha) and the first reserve that integrates conservation of biological diversity and the sustainable development of human communities. The environmental conditions of Mamirauá Reserve encompass one of the preferred habitats of the Amazonian manatee, the "várzea" area.[15] Besides research projects, a management plan for manatees is also being implemented in Mamirauá. This includes environmental education involving the native people (caboclos) with the aim to reach a sustainable use of the natural resources of the reserve.[20,19,29]

The conservation program with the Antillean manatee on the northeast coast of Brazil started in the 1980s, and in 1990 the National Center for the Conservation and Management of Sirenians (NCCMS) was created. Data gathered from the NCCMS indicates that today the manatee of the Brazilian coast has a noncontinuous distribution, which is diminishing in size, and variety of habitats when compared with the historical distribution of the species.[12] Perhaps one of the main advances for the conservation of sirenians was the environmental education program created by NCCMS, which managed to stop the slaughter of manatees on the northeast coast of Brazil. During 19 years of existence, the NCCMS has rehabilitated 27 manatees and recorded four births in captivity, one of which was a case of twins (C.L. Parente, personal communication, 1999).

In October 1998 the National Center for Research, Conservation and Management of Brazilian Aquatic Mammals was created by the Instituto Brasileiro do Meio Ambiente e dos Recursos Naturais Renováveis (IBAMA). This new National Center incorporated all the responsibilities of the NCCMS, which automatically became extinct on the same date.

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REFERENCES

1. Bernardes, A.T.; Machado, A.B.M.; and Rylands, A.B. 1990. Fauna Brasileira Ameaçada de Extinção [Threatened Brazilian Fauna]. Belo Horizonte, Fundação Biodiversitas para a Conservação da Diversidade Biolgica.
2. Best, R.C. 1983. Apparent dry-season fasting in Amazonian manatees (Mammalia: Sirenia). Biotropica 15(1):61–64.
3. Best, R.C. 1984. The aquatic mammals and reptiles of the Amazon. In H. Sioli, ed., The Amazon, Limnology and Landscape: Ecology of a Mighty Tropical River and Its Basin. Dordecht, Dr. W. Junk Publishers, pp. 371–412.
4. Domning, D.P.; and Hayek, L.C. 1984. Horizontal tooth replacement in the Amazonian manatee (*Trichechus inunguis*). Mammalia 48(1):105–127.
5. Domning, D.P.; and Hayek, L.C. 1986. Interspecific and intraspecific morphological variation in manatees (Sirenia: *Trichechus*). Marine Mammal Science 2(2):87–144.
6. Gallivan, G.J.; Best, R.C.; and Kanwisher, J.W. 1983. Temperature regulation in the Amazonian manatee, *Trichechus inunguis*. Physiological Zoology 56(2):255–262.
7. Garcia-Rodriguez, A.I.; Bowen, B.W.; Domning, D.P.; Mignucci-Giannoni, A.A.; Marmontel, M.; Montoya-Ospina, R.A.; Morales-Vela, B.; Rudin, R.; Bonde, R.K.; and Mcguire, P.M. 1998. Phylogeography of the West Indian manatee (*Trichechus manatus*): How many populations and how many taxa? Molecular Ecology 7(9):1137–1149.
8. Garzón, F.; Montenegro, M.; Caicedo-Herrera, D.; Millán-Sanchez, S.; and Montoya-Ospina, R.A. 1996. Plan para la recuperacin y conservacin del manati Antillano (*Trichechus manatus*) en Colombia [Recovery and conservation plan for the Antillean manatee in Colombia]. In Abstracts of the 7° Reunión de Trabajo de Especialistas en Mamíferos Acuáticos de América del Sur. Viña del Mer, SOLAMAC, p. 70.
9. Harrison, R.J.; and King, J.E. 1980. Marine Mammals, 2nd Ed. London, Hutchinson & Co.
10. IUCN. 1996. Red List of Threatened Animals. Gland, Switzerland, IUCN.
11. de Jong, W.; Zweers, A.; and Goodman, M. 1981. Relationship of aardvarks to elephants, hyraxes, and sea cows from alpha-crystalin sequences. Nature 292(5823):538–540.
12. Lima, R.P. 1997. Peixe-boi Marinho (*Trichechus manatus*): Distribuição, Status de Conservação e Aspectos Tradicionais ao Longo do Litoral Nordeste do Brasil [The Antillean manatee: Distribution, status and traditional aspects along the northeast coast of Brazil]. Masters thesis, Universidade Federal de Pernambuco.
13. Lima, R.P.; and Borobia, M. 1991. Peixe-boi marinho, *Trichechus manatus* (Linnaeus, 1758) [The Antillean manatee]. In H.L. Cappozzoand M. Junin, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 182–187.
14. Lima, R.P.; Reid, J.; and Soavinski, R. 1996. Análise preliminar da utilização de Rádio-telemetria e telemetria satelital para conservação e manejo de Sirênios no litoral Nordeste do Brasil [Preliminary analyses of VHF and

satellite telemetry for the conservation and management of sirenians in the northeast of Brazil]. In Abstracts of the 7° Reunión de Trabajo de Especialistas en Mamíferos Acuáticos de América del Sur. Viña del Mer, SOLAMAC, p. 116.
15. Mamirauá. 1996. Mamirauá: Plano de Manejo [Mamirauá: Management Plan]. Brasília, SCM; CNPq/MCT.
16. Marmontel, M. 1988. The Reproductive Anatomy of the Females Manatee *Trichechus manatus latirostris* (Linnaeus 1758) Based on Gross and Histologic Observations. Masters thesis, University of Miami.
17. Marmontel, M.; Odell, D.K.; and Reynolds, J.E., III. 1992. Reproductive biology of South American manatees. In W.C. Hamlett, ed., Reproductive Biology of South American Vertebrates. New York, Spring-Verlag, pp. 295–312.
18. Marmontel, M.; and Rosas, F.C.W. 1996. Plano de Manejo para Preservação e Uso Sustentado do Peixe-boi da Amazônia (*Trichechus inunguis*) na Estação Ecolgica Mamirauá [Management plan for the conservation and sustainable use of the Amazonian manatee in the Mamirauá Reserve]. Manuscript, p. 22.
19. Marmontel, M.; and Rosas, F.C.W. 1996. El manati de Mamirauá: Plan de Manejo para una espécie amenazada en una Reserva de Desarollo Sostenible [The Amazonian manatee in Mamirauá: Management plan for a threatened species in a Sustainable Development Reserve]. In Abstracts of the 7° Reunión de Trabajo de Especialistas en Mamíferos Acuáticos de América del Sur. Viña del Mer, SOLAMAC, p. 68.
20. Marmontel, M.; Rosas, F.C.W.; Martin, A.R.; and Da Silva, V.M.F. 1994. Mamíferos aquáticos na estação ecolgica do Lago Mamirauá, Amazonas, Brasil: O peixe-boi da Amazônia [Aquatic mammals of the Mamirauá Reserve, Amazonas, Brazil: The Amazonian manatee]. In Proceedings of the 6° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Florianpolis, Imprensa Universitária, pp. 37–39.
21. Montgomery, G.G.; Best, R.C.; and Yamakoshi, M. 1981. A radio-tracking study of the Amazonian manatee (*Trichechus inunguis*) (Mammalia: Sirenia). Biotropica 13(2):81–85.
22. Reeves, R.R.; Stewart, B.S.; and Leatherwood, S. 1992. The Sierra Club Handbook of Seals and Sirenians. San Francisco, Sierra Club Books.
23. Reynolds, J.E., III; and Odell, D.K. 1991. Manatees and Dugongs. New York, Facts On File.
24. Ronald, K.; Selley, L.J.; and Amoroso, E.C. 1978. Biological Synopsis of the Manatee. Ottawa, IDRC.
25. Rosas, F.C.W. 1991. Peixe-boi da Amazônia, *Trichechus inunguis* (Natterer, 1883) [Amazonian manatee, *Trichechus inunguis*]. In H.L. Cappozzo and M. Junín, eds., Estado de Conservacin de los Mamíferos Marinos del Atlántico Sudoccidental. Informes y Estudios del Programa de Mares Regionales del PNUMA No. 138. Nairobi, PNUMA, pp. 178–181.
26. Rosas, F.C.W. 1994. Biology, conservation and status of the Amazonian manatee *Trichechus inunguis*. Mammal Review 24(2):49–59.
27. Rosas, F.C.W.; and Best, R.C. 1981. Utilização do peixe-boi amazônico, *Trichechus inunguis,* no controle biolgico de plantas aquáticas [The use of the Amazonian manatee in the biological control of aquatic plants]. In Abstracts of the 1° Encontro Brasileiro de Oceanólogos. Rio Grande, Imprensa Universitária, p. 29.
28. Rosas, F.C.W.; Colares, E.P.; Colares, I.G.; and da Silva, V.M.F. 1991. Mamíferos Aquáticos da Amazônia Brasileira [Aquatic mammals of the Brazilian Amazon]. In A.L. Val, R. Figliuolo, and E. Feldberg, eds., Bases Científicas para Estratégias de Preservação e Desenvolvimento da Amazônia: Fatos e Perspectivas, Vol. i. Manaus, Imprensa Universitária, pp. 405–411.
29. Rosas, F.C.W.; and Marmontel, M. 1996. Telemetria do peixe-boi da Amazônia na Reserva de desenvolvimento sustentável Mamirauá [Telemetry of the Amazonian manatee in the sustainable development reserve of Mamirauá]. In Abstracts of the 7° Reunión de Trabajo de Especialistas en Mamíferos Acuáticos de América del Sur. Viña del Mer, SOLAMAC, p. 68.
30. da Silva, V.M.F.; D'affonseca Neto, J.A.; and Rodriguez, Z.M.C. 1998. Concepção e nascimento do primeiro filhote de peixe-boi da Amazônia em cativeiro [Conception and birth of the first Amazonian manatee calf in captivity]. In Abstracts of the 8° Reunião de Trabalho de Especialistas em Mamíferos Aquáticos da América do Sul. Olinda, SOLAMAC, p. 57.
31. Timm, R.M.; Albuja, L.V.; and Clauson, B.L. 1986. Ecology, distribution, harvest, and conservation of the Amazonian manatee *Trichechus inunguis* in Ecuador. Biotropica 18(2):150–156.

MEDICINE

Tatiana Lucena Pimentel

INTRODUCTION

It is strongly recommended that before release into the wild, rehabilitated manatees have a period of readaptation in a semicaptive enclosure and that a thorough pre-release health examination be performed.

PHYSIOLOGY

The metabolic rate of manatees is one-third that of most other mammals.[12] Their normal cardiac frequency is approximately 40 beats per minute (bpm) when swimming or eating, but it can be reduced to 8 bpm in the case of prolonged threat. When under threat, the heart oxygen consumption is reduced to a minimum and at the same time their peripheral metabolism converts to an anaerobic phase. The blood oxygen is conserved and reserved for the vital organs, such as the heart, lungs, and brain.[2]

Manatees usually breathe approximately once every 1 minute, when in rapid locomotion and once every 2.5 minutes when playing or when moving at a cruising velocity (3 km per hour). They breathe once every 4 minutes when feeding, and twice when resting. With the first breath the lung is partially filled, and with the second one, in approximately 50 seconds, the lung is filled to capacity. This process is repeated at approximately 12-minute intervals (the author's personal observation).

Manatees have relatively scant layers of subcutaneous fat when compared with cetaceans and pinnipeds. The rectal temperature of the manatee ranges between 34.5° to 36°C; both free-ranging and captive animals are intolerant of cold temperatures and have been reported to develop anorexia and lethargy at water temperatures below 16°C.

Longevity of free-ranging animals has been estimated to exceed 50 years.[22] To estimate the age of a manatee, two techniques are applied: x-rays of its pectoral flippers and analyzing the ossification of calves' epiphysis, in which case better results are found with the thick ear bones.

REPRODUCTION

Sexual dimorphism is not evident in manatees, therefore, it is necessary to visualize the ventral region of a manatee to determine its gender. In females, a urogenital opening is close to the anus, whereas in the male the penile opening is located about 40–90 cm anterior to the anus. The penis and testes are intraabdominal for hydrodynamics.

Unlike the Amazonian manatee, the Antillean manatee does not show a seasonal reproduction pattern. This species seems to reproduce year-round,[29] pregnancy lasts about 13 months,[4, 14, 30] and they usually produce one calf every 2.5–5 years.[29] Although manatees tend to produce only one calf at a time, the birth of twins occurred in captivity at the National Center for the Conservation and Management of Sirenians in Itamaracá, Brazil.

Females have a single mammary teat situated under each flipper in the axilary region, and observations of calves born at the Miami Seaquarium suggest that young manatees continue to nurse until they are 18–24 months of age. These observations are in agreement with reports of free-ranging sirenians.[29]

NUTRITION

Manatees are herbivorous monogastric animals and are opportunistic feeders with a highly varied diet. The bulk of cellulose digestion occurs in the cecum and colon, and passage of foodstuffs through the upper digestive system is relatively rapid. The highly efficient microbial fermentation that takes place in the lower digestive tract has been compared to hindgut digestion in the horse, and allows for a relatively slow transit time, estimated at 146 hours.[29] For more details on the passage time of foodstuff see Itavo.[18] It is important to note that frequent flatulence has been observed as a normal occurrence in captive and released manatees.

It was previously believed that the Amazonian manatee could consume about 8% of its body weight per day,[3] but according to a more recent study by Cavallante,[9] food consumption is directly affected by the nutritive value of the diet, where factors such as the amount of protein, lipids, carbohydrates, fibers, and water content seem to act as moderators. Food consumption percentages varied from 2.2% to 23.7%, according to the nutritive content of the forage.[9]

A change in the diet of manatees at INPA is believed to have played a major role in the birth of the first Amazonian manatee in captivity,[16] which favors the theory on the relationship between diet and reproduction.

Formula milk used in Brazil for young manatees consists of:

- For the Amazonian manatees—2 tablespoons lactose-free milk , 1 cup filtered fresh lukewarm/tepid water, 1 tablespoon milk butter, 100 g wheat cereal (oat cereal caused constipation in Amazonians [F.A.P. Colares personal communication, 1995]), 1 egg yolk (every other day), and 1 mL multivitamin.
- For the Antillean manatees-–135 g lactose-free powdered milk, 1 L filtered and boiled fresh water, cooled to tepid, 4 tablespoons milk butter, 4 tablespoons quick oat cereal, and 5 mL multivitamin.

This formula should be offered three times a day in the first 4 months and four times a day when gradually increasing the amount of food. It is always important to offer soft vegetables, algae, and grasses (such as *Eichhornia crassipes, Gracilaria* sp., *Vidalia* sp., *Hipnaeae* sp., *Laurencia* sp., and *Halodule* sp.) to supplement the diet.

It is possible to improvise a bucket to hold milk to automatically nurse the pups, as is done with cattle calves, to avoid imprint with humans. To imitate their way of feeding a 3 m (10 ft) polyvinyl chloride (PVC) tube is cut longitudinally and pierced by round symmetrically distanced holes. The algae, lettuce, or sea grass is placed inside. The tube must have weights in both ends to sink it so the animals will have to dive to eat.

BEHAVIOR

Manatees are slow movers, often resting motionless either at the surface or near the bottom.[7] Such behavior may lead people to think that the animal is dead or in distress. Copulatory behavior during mating encounters can also be misinterpreted as aggressive, as several males try to mate with the same female. Real distress is produced by fresh propeller wounds or monofilament line wrapped around a flipper. Distress signs are weakness, emaciation, inability to submerge, pronounced listing to one side, reluctance to move or suckle, or labored breathing.[22]

Manatees may be regarded as semisocial, with the basic social unit represented by a female and her calf. Females and their offspring have been observed to remain together for up to 2 years. Sick or injured females may be stranded accompanied by a calf.

Calves vocalize at or soon after birth, which is important for mother–calf bonding. Vocalizations in manatees are best described as high-pitched squeals, chirp-squeals, and screams,[11] especially in young animals.

DEALING WITH MANATEES IN THE FIELD

It is of great importance that all necessary equipment for the capture of a manatee is arranged and in place before the process starts. The animal's condition should be evaluated before making further decisions. It is important to remember that usually the bigger the manatee, the calmer it is. On the other hand, healthy animals may injure handlers and even themselves when struggling to escape. Severely debilitated animals may offer little or no resistence.[26]

One method of capture, when using a boat, is to find a beach near the area of capture, then surround the manatee very fast while dropping a 100-m-long net 15 m deep around it. This net has buoys on one side and weights on the other, so it will be suspended in a column in the water like a wall around the animal. Then, the stern of the boat should be removed and people should start to pull the net toward the boat, moving the manatee slowly to the boat and then to the beach to be handled. This method requires about seven people for an adult manatee. Once pulled to a sandy beach, well above the water line, or into shallow waters, manatees usually become calm and may require little restraint. The net around it will make it feel restrained. It is always a good idea to cover its eyes, and the staff should be aware of sudden tail thrashings. Another capture method is described by Geraci.[13]

Calves may be restrained and supported in the water by a single person, but they must be laid on a stretcher after being removed from the water to avoid being dropped. They are difficult to hold and may be seriously injured if they fall.[13]

Open wounds or injured flippers should be protected from further injury during handling and transport.[13]

REHABILITATION IN CAPTIVITY

On arrival at a rehabilitation center, the animal should be measured and given a thorough physical examination. Blood and fecal samples should be collected. Once settled, the animal will soon need to eat and can be offered a variety of green plants, including lettuce, cabbage, spinach, celery, carrots, natural water grasses, and water hyacinth.[1] An animal that refuses food may first require fluids administered by stomach tube, followed by a mixture consisting of ground lettuce, apples, monkey biscuit, and water.[8] To intubate the animal one should insert a foalnasal tube through the esophagus to the first third of its body length, where the stomach is located.

Artificial formulas have been used successfully to nurse orphan calves back to health.[28]

HOUSING

In Brazil manatees have been maintained in captivity in a 2.5-m deep tank, approximately 10 m in diameter, with capacity for approximately 196,000 L (52,000 gal) of clean water that may be fresh or brackish. A tank of this size should support about six animals. It is a good idea to have a quarantine tank to keep orphaned calves that have recently arrived at the facility.

In an ideal situation, seawater can pass through a sand and gravel filter before entering the pool. The fresh water requirement of the manatee is not known; however, a source of fresh water should be available at all times to animals housed in saltwater.

The tank should have a holding area, which is a small pool separated from the main tank by a guillotine door. Once the animal is in this area, water can be drained and the necessary handling can take place. Two or more tanks may be linked by holding areas of about $3.5 \times 2.5 \times 1.5$ m with a capacity for 13,125 L each.[9] The tank should be cleaned every day, removing any feed present, and the water should be changed every week or filtered daily. Water analysis should be carried out once a week to monitor the amount of chlorine and the pH.

Care must be taken with tempered glass used as a viewing window for the public. It can support only a certain pressure, which must be respected. Increasing the amount of water or the number of animals can lead to great damage to the viewing window and to the animals. If the maximum pressure is exceeded, the glass window may break and the animals thrown out of the pool.

Algae growth may be an esthetic problem for facilities housing manatees outdoors. According to Zeiller[30] , successful biological control of algae on both manatees and concrete surfaces can be achieved by placing mullet (*Mugil cephalus*) in the manatee pool. Zeiller also reported that a substantial reduction of hours required to remove algae growth resulted when 0.05 mg/L of copper sulfate was added to the manatee pool before manual cleaning. Algae growth on the dorsal surface of the slow-moving manatee possibly provides needed protection from the harsh tropical sun. Therefore, algae control on the epidermis of captive manatees maintained in outdoor facilities should be governed by the degree of shade available.[28]

A temperature range of 16°–30°C has been suggested for manatees maintained in a captive environment.[28] In Brazil, the Antillean manatee is kept at a temperature range of 25–29°C, and the Amazonian manatee at a range of 20–25°C.

The amount of food given to captive manatees should be approximately 4% of their body weight and should consist of fresh vegetables such as carrots, lettuce, beet root, and cabbage. Fresh algae and sea grass (*Halodule* sp.) are recommended only if collected and fed on the same day. Monkey biscuit should not be given frequently to animals in captivity, because it may result in animals becoming overweight. Part of a conditioning program should include feeding manatees inside the holding area, which will help to avoid stress when handling is required.

Animals being kept for reproduction, or that cannot be reintroduced to the wild, should be allowed to develop a close contact with humans for ease of handling. On the other hand, animals destined for reintroduction should have little contact with researchers and staff.

PHYSICAL AND CHEMICAL RESTRAINT

Physical restraint is the most common means of handling manatees for physical examination and clinical procedures. The animals are tolerant of this type of handling, and no clinically evident adverse effects have been reported.[29] The animals can also be trained for blood sampling.

As the manatee begins to strand as the water level depth of the holding area drops, it may roll onto its back and calm itself. This represents an attempt by the animal to decrease pressure on its thorax and abdomen and may ease respiratory effort. Care should be taken that the nostrils are not submerged when the animal rolls.[29] A manatee can be rolled toward a stretcher or even lifted by about five people at once in a fast precise movement. Once on the stretcher, the manatee may be lifted to be weighed.

A stretcher or any special equipment is not necessary for routine blood sampling or body measurements. Alternatively, restraint of a large manatee for injection or simple procedures may also be accomplished by emptying the tank completely and holding the animal securely.[29] However, access to the ventral surface for urine collection or core body temperature measurements is easier if the animal is secured in a stretcher. The stretcher should be fitted with ventrally located slots to allow access to the urogenital area (i.e., for taking urine samples), with two holes for the pectoral flippers and two bars on both sides to make it firm to facilitate lifting the animal.

Chemical restraint has been successfully used in manatees by Walsh[28] to achieve relaxation and for local anesthesia (lidocaine, 2%) during minor surgical procedures. Diazepam (1 mg/55 kg body weight, intramuscularlly [IM]), midazolan hydrochloride (1 mg/55 kg body weight, IM), and meperidine (1.65 mg/kg body weight, IM) have been used.[29] Caution must be taken when administering the drug, because apnea may result.

Once the animal is restrained, blood samples may be collected by puncturing the subdivisions of the brachial arteries at the palmar side of the pectoral fin, between the radius and the ulna, using musculoskeletal landmarks. Firm restraint of the flipper while the animal is stranded is usually adequate. Before inserting the needle, the blood sampling area should be disinfected with a povidone iodine solution to minimize contamination of internal tissues with superficial bacteria and commensal organisms. Disposable syringes and 18/19/20-gauge, 4-cm-long needles, with or without an extension set, should be used to collect up to 20 mL of blood. Normal values for blood constituents have been reported (see Table 31.1).

The hematological parameters determined in wild manatees were assumed to represent normal standards of the species. According to Rosas et al.,[27] copper and sodium levels were similar between captive and wild manatees, but zinc, magnesium, iron, potassium, and calcium levels in wild manatees were significantly higher than those of captive animals. Reduced levels of hematocrit and hemoglobin reflect a long period of iron deprivation.[27] The hematocrit values increased in all six

TABLE 31.1. Manatees' blood parameters

		Amazonian Manatee		West Indian Manatee		
Glucose (mg/dL)	X	43.3	73.4	80.0	83.0	75.5
	S	7.5	11.9	35.8	29.0	26.6
Lipides (mg/dL)	X	543.6	—	—	—	—
	S	59.14	—	—	—	—
Urea (mg/dL)	X	29.3	34.2	17.8	30.4	28.7
	S	6.8	4.8	7.1	9.0	7.8
Protein (g/dL)	X	6.3	7.4	7.7	8.3	—
	S	0.4	0.7	0.1	0.4	—
Albumin (g/dL)	X	3.5	—	5.7	4.9	—
	S	0.2	—	0.2	0.4	
Globulin (g/dL)	X	2.8	—	2.8	3.4	—
	S	0.4	—	0.3	0.3	
References		10	29	17	23	25

X, average; S, standard deviation.

TABLE 31.2. Average and standard deviation values of hematocrit, hemoglobin, and mineral content of the serum of wild Amazonian manatees

	(x ± SD)	*n*	(x ± SD)	*n*
Ht (%)	36.0 ± 1.4	2	—	—
Hb (g/dL)	12.2 ± 0.8	3	11.2 ± 3.5	2
Zn (ppm)	3.4 ± 0.7[a]	3	1.1 ± 0.3[a]	2
Cu (ppm)	0.32 ± 0.2	3	0.26 ± 0.1	2
Mg (ppm)	41.8 ± 5.3[a,b]	3	26.8 ± 4.5[a]	2
Fe (ppm)	2.4 ± 0.1[b,c]	3	1.62 ± 0.1[b]	2
Na (ppm)	1869 ± 36.2	3	2084 ± 147.1	2
K (ppm)	287.3 ± 35.5[d]	3	326 ± 70.7	2
Ca (ppm)	125.5 ± 11.7[b,d]	3	73.8 ± 12.2[d]	2

Source: See reference 27.
[a] = $P < .05$.
[b] = $P < .01$.
[c] = $P < .01$.
[d] = $P < .02$.
SD, standard deviation; Ht, hematocrit; Hb, hemoglobin.

manatees with low levels after the proportions of lettuce and cabbage in their diet were increased toward the end of the study(Table 31.2).[27]

Intramuscular injections should be given in the musculature of the lumbar area. The selected site should be laterally located to permit drainage in the event of abscessation or similar complications.[29] Asepsia should be done on the injection area to avoid bacteria contamination. A 16-gauge, 2-inch disposable needle is adequate for intramuscular injection at this region.

TRANSLOCATION

Knowledge of the animals being prepared for release and the choice of the releasing area should also be carefully evaluated. It should be a calm, if possible, protected area, with plenty of food, warm water, and little or no human disturbance. It is strongly recommended that releases be done only after a period of at least 70 days of readaptation in a pen constructed at the exact location where the animals are to be released.

In Brazil, the translocation of adult manatees from captivity to semicaptivity in the wild is carried out inside a pool placed over a truck. A canvas is also placed over the pool to protect it from wind and the morning sun, in case of a long journey. The pool should contain only enough water to keep the animals moist, and a thick foam pad should be used under the manatees. A veterinarian is present during the entire trip, and he or she often travels with the animal. The translocation is done at night to avoid sunburn. Capture stress (myopathy) is not a concern, even after several hours of transport.[27] However, it is best to minimize pursuit, vigorous handling, and long journeys.[13] Stranded calves are transported the same way, but in a smaller pool inside a van.

DISEASES

Few reports exist on diseases of captive manatees. Morales et al.[24] reported systemic mycobacteriosis in a captive Amazonian manatee, *T. inunguis*. Sporadic behavioral signs of malaise, such as anorexia and lethargy, were first noted in 1980, 13 years after the capture of the animal.[24] Pruritic skin lesions, described as patchy gray areas distributed on the head and back, became apparent during the periods of behavioral change. Hair follicles in affected areas were white and raised.[24]

Tuberculosis (*Mycobacterium chelonei*) has been reported to produce pneumonia and dermatitis in the manatee.[5] Clinical signs included a pyoderma. Each vesicle was filled with purulent exudate. In this case no respiratory signs were evident; however, on necropsy six caseous tubercles were found in the lungs. Diagnosis may be made by biopsy, culturing the organism from skin lesions or tracheal swabs, by radiography, or possibly by intradermal tuberculosis testing. Treatment, if attempted, would be with isoniazid given orally.[6]

Pyoderma has been reported in a captive 1-year-old female Amazonian manatee, *T. inunguis*. Although initially the condition of the animal improved following therapy with lincomycin, it succumbed after 4 years, and *M. cheloniae* was isolated from lung abscesses.[5]

Epidermal mycosis, caused by *Epidermophytos floccosum*, was reported in a captive manatee by Dilbone.[29] The condition was manifested by lesions on the rostrum, face, flippers, and tail.[29] Diagnosis was made by skin scraping and treatment included Vaseline and Mycostatin ointment along with 20 mg/kg of griseofulvin wrapped in lettuce leaves and given orally.[6]

Ulcerative skin lesions in a captive bottlenose dolphin, *Tursiops truncatus*, and Florida manatee, *T. manatus*, were attributed to the copepod, *Harpacticus pulex*. Pathology caused by copepods have not been reported in free-ranging animals, which leads researchers to believe that such infestations are related to captive conditions.[29]

Observations of captive manatees at the Miami Seaquarium have revealed the sporadic occurrence of raised, circular, poxlike lesions, which are yellow in color and distributed on the backs of the animals. Etiology of these lesions has never been determined. Electron micrographs of biopsies do not reveal virus particles. Resolution of the lesions has been associated with increased dietary ascorbic acid.[29]

Numerous species of trematodes and cestodes have been described in sirenians, most of which have been diagnosed at necropsy, but the clinical significance is unknown. [6] Sirenia have been medicated orally using a balling gun.[6]

Dosage based on uptake and excretion rates for selected antibiotics are as follows: [6]

- Penicillin (IM)—8800 IU/kg/24 h;
- Gentamicin (IM)—5 mg/kg/12 h;
- Tetracyclines (oral)—88 mg/kg/4 h;
- Chloramphenicol (oral)—44 mg/kg, then 22 mg/kg/12 h;
- Doxycycline (oral)—same dose as chloramphenicol.

The most common causes of free-ranging manatee mortality are human-related trauma, cold exposure, and disease. Direct impact from boat collisions may be responsible for as much manatee mortality as propeller injuries.[29] Necropsies have been carried out on manatees with few or no propeller laceration damage, in which the force of the impact had fractured the cranium or caused death from exsanguination, pneumothorax, pleuritis, or a related sequel. One animal that died had ingested steel wool.

Lethal injections of barbiturate have been used effectively for euthanasia.[13]

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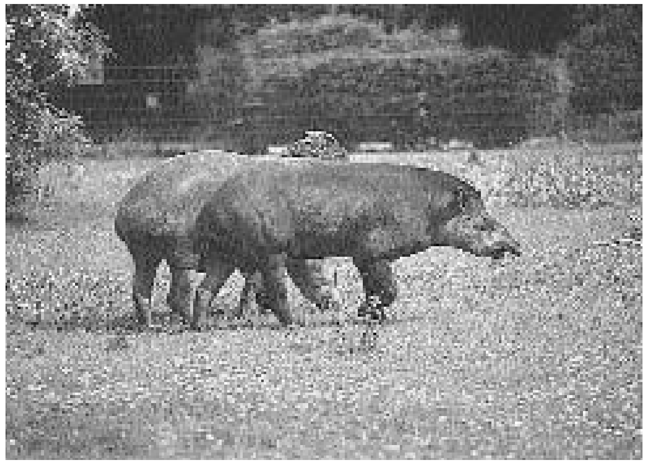
REFERENCES

1. Asper, E.D.; and Sarles, S.W. 1981. Husbandry of injured and orphaned manatees at Sea World of Florida.. In R.L. Brownell, Jr., and K. Ralls, eds., The West Indian Manatee in Florida. Tallahassee, Florida Department of Natural Resources, pp. 121–127
2. Best, R.C. 1981. A Salvação de uma Espécie; Novas Perspectivas para o Peixe-boi da Amazônia [The salvation of a species; new perspectives for the Amazonian manatee]. Manus, Division of Aquatic Mammal Biology at the National Institute of Amazonian Research, pp. 6–15.
3. Best, R.C. 1981. Foods and feeding habits of wild and captive Sirenia. Mammal Review 111(1):3–29.

4. Best, R.C. 1983. Apparent dry-season fasting in Amazonian manatees (Mammalia: Sirenia). Biotropica 15(1):61–64.
5. Boever, W.J.; Theon, C.O.; and Wallach, J.D. 1976. Systemic *Mycobacterium chelonei* infection in a Natterer manatee. Journal of the American Veterinary Medical Association 169:927.
6. Boever, W.J.; and Wallach, J.D. 1978. Diseases of Exotic Animals: Medical and Surgical Management. Philadelphia, W.B. Saunders, pp. 686–725.
7. Bonde, R.K. 1992. Pers. commun. Cited in Geraci, J.R.; and Lounsbury, V.J. 1993. Marine Mammals Ashore: A Field Guide for Strandings. Galveston, Texas, Texas A & M University, pp. 145–158.
8. Bossart, G. 1991. Pers. commun. Cited in Geraci, J.R.; and Lounsbury, V.J. 1993. Marine Mammals Ashore: A Field Guide for Strandings. Galveston, Texas, Texas A & M University, pp. 145–158.
9. Cavallante, A.P. 1995. Taxa de consumo alimentar do peixe-boi da Amazônia (*Trichechus inunguis*) (Natterer, 1883), em cativeiro. Bachelor of Science thesis, State University of Londrina.
10. Colares, I.G.; Colares, E.P.; and do Amaral, A.D.P. 1992. Parâmetros bioquímicos do sangue do peixe-boi da Amazônia (*Trichechus inunguis;* Mammalia: Sirênia). Revista Peixe-boi 1:26–31.
11. Evans, W.E; and Herald, E.S. 1970. Underwater calls of a captive Amazon manatee, *Trichechus inunguis*. Journal of Mammalogy 51(4):820–823.
12. Gallivan, G.J.; and Best, R.C. 1980. Metabolism and respiration of the Amazonian manatee (*Trichechus inunguis*). Physiological Zoology 53(3):245–253.
13. Geraci, J.R.; and Lounsbury, V.J. 1993. Marine Mammals Ashore. A Field Guide for Strandings. Galveston, Texas, Texas A & M University, pp. 145–158.
14. Harrison, R.J.; and King, J.E. 1980. Marine Mammals, 2nd Ed. London, Hutchinson & Co.
15. Howard-Williams, D.K.; and Junk, W.J. 1977. The chemical composition of Central Amazonian aquatic macrophytes with special reference to their role in the ecosystem. Archives of Hydrobiology 79(4):446–464.
16. 1998. Isto é Brazilian Journal. 1998. Peixe-boi no berço. Editora Tês, Sâo Paulo, 25(1497):1–3.
17. Irvine, A.B.; Neal, F.C.; Cardeilhac, P.T.; Popp, J.A.; White, F.H.; and Jenkins, R.L. 1980. Clinical observations on captive and free-ranging manatees, *Trichechus manatus,* in Florida. Aquatic Mammals, 8:2–10.
18. Itavo, R.V. 1995. Tempo de passagem do alimento no trato digestivo do peixe-boi da Amazônia (*Trichechus inunguis*) em cativeiro [Food transit time in the captive Amazonian manatee (*Trichechus inunguis*) digestive tract]. Masters thesis, State University of Londrina.
19. Junk, W.J. 1970. Investigations on the ecology and production biology of the floating meadows (Paspalo: Echinochloetum) of the Middle Amazon, 2. The aquatic fauna in the root zone of floating vegetation. Amazonian 4(1):9–102.
20. Marmontel, M.; Odell, D.K.; and Reynolds, J.E., III. 1992. Reproductive biology of South American manatees. In W.C. Hamlett, ed., Reproductive Biology of South American Vertebrates. New York , Springer-Verlag, pp. 295–312.
21. Marmontel, M.; and Rosas, F.C.W. 1996. Plano de manejo para preservação e uso sustentado do peixe-boi da Amazônia (*Trichechus inunguis*) na Estação Ecológica Mamirauá [Management plan for the conservation and sustainable use of the Amazonian manatee in the Mamirauá Reserve]. Manuscript, p. 22.
22. Marsh, H.; and Anderson, P.K. 1983. Probable susceptibility of dugong to capture stress. Biological Conservation 25:1–3.
23. Medway, W.; Bruss, M.L.; Bengtson, J.L.; and Black, D.J. 1982. Blood chemistry of the West Indian manatee (*Trichechus manatus*). Journal of Wildlife Diseases. 18:229–234.
24. Morales, P.; Madin, S.H.; and Hunter, A. 1985. Systemic *Mycobacterium marinum* infection in an Amazon manatee. Journal of the American Veterinary Medical Association 187:1230.
25. O'Shea, T.J., Rathbun, G.B.; Asper, E.D.; and Searless, S.W. 1985. Tolerance of the West Indian Manatee to capture and handling. Biological Conservation 33:335–349.
26. O'Shea, T.J.; Rathbun, G.B.; Bonde, R.K.; Buergelt, C.D.; and Odell, D.K. 1991. An epizootic of Florida manatees associated with a dinoflagellate bloom. Marine Mammal Science 7:165–179.
27. Rosas, F.C.W.; Lehti, K.K.; and Marmontel, M. 1999. Hematological indices and mineral content of serum in captive and wild Amazonian manatees, *Trichechus inunguis*. Arquivos de Ciençias Veterinarias E Zoologia Da Unipár 2(1):37–42.
28. Walsh, M. 1992. Pers. commun. Cited in Geraci, J.R.; and Lounsbury, V.J. 1993. Marine Mammals Ashore: A Field Guide for Strandings. Galveston, Texas, Texas A&M University, pp. 145–158.
29. White, J.R.; and Francis-Floyd, R. 1990. Manatee biology and medicine. In L. Dierauf, ed., Handbook of Marine Mammal Medicine: Health, Disease, and Rehabilitation. Boca Raton, Florida, CRC Press, pp. 601–623.
30. Zeiller, W. 1978. The management of West Indian manatees (*Trichechus manatus*) at the Miami Seaquarium. In R.L. Brownell and K. Ralls, eds., Proceedings of the West Indian Manatee in Florida. Maitland, Florida, Florida Audubon Society, p. 103.



32 Order Perissodactyla, Family Tapiridae (Tapirs)

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BIOLOGY

Emília Patrícia Medici

INTRODUCTION

The family Tapiridae as a taxonomic entity first appeared in the Eocene of North America, nearly 50 million years ago. Crossing the intermittent connections between North America and Asia via the Bering Straits, the tapirs soon appeared in Euro-Asia. With the completion of the land bridge between North America and South America during the Pliocene (7–2 million years ago), tapirs entered South America.

Although tapirs have died out over much of their former range, they persist in southeast Asia, Central America, and South America. There are four surviving species: one in Asia (Malayan tapir, *Tapirus indicus*), one in Central America (Baird's tapir, *Tapirus bairdii*), and two in South America (lowland tapir, *Tapirus terrestris,* and mountain tapir*, Tapirus pinchaque*); all are threatened to some extent, if not endangered (see Table 32.1).

ANATOMY

Tapirs are about the size of a donkey with a body that is rounded in back and tapered in front, making it well suited for rapid movement through thick underbrush. In the forest a tapir can run as fast as a human. They are also excellent swimmers and are fond of splashing in water and wallowing in mud. The tail is short and seems to have no significant purpose other than providing a cover for the anus. Tapirs have bristly hairs scattered all over the body, and an inconspicuous mane is present on the two South American species. All the South American tapirs are a uniform dark brown or gray color, whereas the Malayan tapir is black on its hind legs and the entire front of its body and creamy white through its midsection. All tapirs have a short, fleshy proboscis made of the snout and upper lips. This proboscis is more elongated in the South American species. The tapir's eyes are small and set flush with the side of the head; the ears are oval, erect, and not very mobile.

Skeletal features include short, slender legs with the radius and ulna separate and equally developed. The fibula is also complete. The feet are mesaxonic. The forefoot has three primary digits, and a smaller one (the fifth),

TABLE 32.1. Biological data for South American tapirs (order Perissodactyla, family Tapiridae)

Scientific Name	Name (English)	Name (Spanish)	Name (Portuguese)	Identification	Distribution	Weight (kg)
Tapirus terrestis	Brazilian tapir, common tapir, lowland tapir	Tapir, anta, danta, gran bestias, sacha vaca	Tapir, anta, marebis	All tapirs have a rounded topline in the back and are tapered in front. Color is a dark brown to reddish above and lighter below. Possesses a short fleshy proboscis. Eyes small; ears oval, erect and have a thin white margin. The legs are short and slender. Three main toes in front with a smaller fifth digit. Three toes on the hind foot. The tail is short and thick. One pair of mammary glands. Prominent nuchal crest. Concave head profile.	Amazon basin, northern countries of South America to southern Brazil, eastern Peru, Bolivia and Paraguay and northern Argentina.	150–200
Tapirus pinchaque	Mountain tapir, wooly tapir	Tapir, anta, danta	Tapir, anta	Has wiry black hair with prominent white ear and lip fringes. Convex head profile. No nuchal crest or mane.	Andes Mountains, 2000 to 4000 m, Colombia, Venezuela, Equador, Peru	100–145
Tapirus bairdii	Central American tapir, Baird's tapir	Tapir, danta, anta, anteburro	Tapir, anta	Similar to *T. terrestris* without a prominent nuchal crest, but with stiff mane. Head profile is convex.	Southern Mexico to Colombia, Venezuela, and Equador, west of the Andes.	110–145

which is used only when the tapir is walking on soft ground. The hind feet have three digits. All the toes are hoofed. The splayed feet, with four toes on each front foot and three on each back foot, help them walk in soft or muddy ground. Tapirs have relatively long, laterally compressed skulls with a high brain case and convex profile. The nasal bones are short, arched, and freely projecting. The nasal opening is large.

The dental formula of tapirs is similar to that of the equids: 3/3, 1/1, 4/3–4, and 3/3 for a total of 42-44 teeth. The incisors are chisel shaped and canines are conical. All cheek teeth lack cement. They are low crowned and strongly lophodont.

REPRODUCTION

Tapirs have a low reproductive rate. Most information about reproduction comes from the lowland tapir, but some generalizations may be advanced. A single young is born after a 13-month gestation. Although a female tapir may conceive within a month after giving birth, this is not an invariable rule. It is safe to say that under the best circumstances an offspring may be born every 14 months in habitats exhibiting little seasonality in food availability. In seasonally arid habits, the interval between births may be longer. A female does not become sexually mature until she is nearly 2 years of age under the best of conditions, but may remain reproductively active into her 10th year of life and beyond. In South America tapir productivity is lower than that exhibited by the deer or peccaries that may share their habitat.[15]

Young of all four species have striped markings that are lost at 6 months old. Most young tapirs weigh approximately 7–9 kg at birth. The young are weaned at 10–12 months. Tapirs live for approximately 30 years.

BEHAVIOR

Tapirs are shy, silent, and rarely seen. When they are alarmed, they run for the nearest water, dive in, and swim beneath the surface. They possess keen senses of hearing and smell. Although primarily solitary, females with dependent young, adults with juveniles, or feeding groups are not unusual.[14,37,41]

FEEDING

Tapirs are exclusively herbivorous, sheltering in thickets by day and emerging at night to feed in bordering grassy or shrubby areas. They eat the leaves, buds, twigs, and fruits of low-growing, terrestrial plants and also consume aquatic vegetation. The extensible proboscis is used to strip leaves and pluck fruits. Their diet includes a bewildering array of plant species and many different plant parts. Fruit may be taken from low shrubs or as fallen fruit on the ground. Podlike fruits with small seeds may be chewed, with considerable damage to seeds, but fleshy fruits with large seeds may be consumed whole and the seeds passed through the gut with minimal damage and enhanced ability to germinate.

MISCELLANEOUS

The spatial requirements of tapirs vary with the carrying capacity of the habitats. Mountain tapir may exhibit an exclusive home-range use pattern. Densities of tapirs tend to be low, with estimates ranging from a high of 1/km^2 to fewer than 0.3/km^2. The individualistic life style and relatively low density precludes achieving a high local abundance, and with high hunting pressure local populations may easily become extinct. Fragmentation of preferred habitat increases the vulnerability for extirpation. Mortality in tapirs may be heavy during the first year of life since the larger predators (jaguars and pumas in Central and South America) can and do take younger animals.

Although tapirs do not share the glamour of elephants, pandas, and the large cats, they certainly deserve higher visibility. What we are looking at today is a remnant of a very successful taxon, with a distinguished and ancient lineage. Although relatives of the horse and rhinoceros, these shy, cryptic animals are often overlooked.

THE SOUTH AMERICAN TAPIRS

Baird's Tapir (*Tapirus bairdii*)

LIFE HISTORY Captive males may be reproductively mature at 24 months; one captive female was 22 months at first mating. Interestrous intervals in captive individuals range from 25 to 38 days, with estrous periods lasting 1 to 4 days.[3,8] Although females resume cycling 14–18 days after parturition, the interbirth interval is rarely less than 18 months for captive females. A single young is born after 390–410 days for captive tapirs.[1,3,8,14] During the first week after birth, the young is tucked in a secluded spot where the female periodically returns from feeding to nurse it. By day 10, the young actively follow the mother.[14] The spotted calves grow rapidly and are capable of swimming at 3 weeks old.[4]

In Central America, minimum density is 0.05 animals/km^2. It is interesting to note that there is a general decline at Barro Colorado Island (BCI), Panama. This may be a relict of different sampling regimes.

In northwestern Costa Rica, an adult male's nocturnal range (1.8 km^2) was 12 times greater than his diurnal range (0.15 km^2), and a juvenile male's nocturnal range (1.61 km^2) was 6 times greater than his diurnal range (0.27 km^2).[41] Home ranges may be used in a rotational manner; a portion of the annual home range may be used intensively before moving to another portion.[41]

Wooly Mountain Tapir (*Tapirus pinchaque*)

LIFE HISTORY Estrus lasts 3–4 days and is on a lunar cycle. The mountain tapir has a gestation period of around 393 days,[6] and usually gives birth to a single young, and rarely twins.[14,39] Sexual maturity is reached at about 3 years of age. Males are reported to engage in violent confrontations over females.

Similar to the Malayan tapir, home ranges of adults overlap by as much as one-third, with a core territory belonging to the male, his mate, and their offspring.[40] Marking by depositing dung piles and rubbing on trees seems to be part of a male's territorial behavior; marking is done by females who share the same territory, as well.[12,13]

The core home range of a mountain tapir averages 8.8 km^2. The steep terrain mountain tapirs inhabit actually provides it with a much greater surface habitat to exploit. Males show greater fidelity to their more circular territory and have a greater facility of defense than females. The mountain tapir is equally active during the day and night, with strong crepuscular behavior.[12,13] Increased nocturnal activity may be witnessed in areas with a greater presence of humans and livestock invasion.

Lowland Tapir (*Tapirus terrestris*)

LIFE HISTORY Captive females are polyestrous.[9] The gestation period is approximately 13 months.[42] In some regions, such as El Rey National Park, Argentine tapirs are typically diurnal, perhaps because of the lack of human disturbance.[19] Range use changes seasonally in this species. Mutualistic symbiotic relationships develop between tapirs and birds such as anús (*Crotophaga* spp.) and black caracaras (*Dapirius ater*), where the birds glean ectoparasites such as ticks from the tapirs skin.[32]

Lowland tapirs are threatened with local extinction in many areas in South America, because of overhunting and selective destruction of preferred tapir habitat. Throughout their range, tapirs are highly susceptible to overhunting, and populations show rapid decline when harvested. In many South American countries subsistence laws allow hunting of tapir, because the susceptibility of tapir to overhunting was not understood when the laws were initially enacted. Tapirs are also threatened by the destruction of palm forests and other preferred tapir habitat, which are being cut down at alarming rates in some areas by workers in development projects and by local residents.

REFERENCES

1. Alvarez del Toro, M. 1966. A note on the breeding of Baird's tapir at Tuxtla Gutiérrez Zoo. International Zoological Yearbook 6:196–197.
2. Arita, H.T.; Robinson, J.G.; and Redford, K.H. 1990. Rarity in Neotropical forest mammals and its ecological correlates. Conservation Biology 4:181–192.
3. Barongi, R.A. 1986. Tapirs in captivity and their management at Miami Metrozoo. AAZPA Annual Proceedings 22:96–103.
4. Barongi, R.A. 1993. Husbandry and conservation of tapirs. International Zoological Yearbook 32:7–15.
5. Bodmer, R.E. 1990. Fruit patch size and frugivory in the lowland tapir (*Tapirus terrestris*). J. Zool. 222:121–128.
6. Bonney, S.; and Crotty, M.J. 1978. Breeding the mountain tapir at the L.A. Zoo. International Zoological Yearbook 18:198–200.
7. Brooks, D.M.; Bodmer, R.E.; and Matola, S. Eds. 1997. Tapirs - Status Survey and Conservation Action Plan. (English, Spanish, Portuguese.) IUCN/SSC Tapir Specialist Group. IUCN, Gland, Switzerland and Cambridge, UK. pp. viii, 164 pp.
8. Brown, J.L.; Citino, S.B.; Shaw, J.; and Miller, C. 1994. Endocrine profiles during the estrous cycle and pregnancy in the Baird's tapir (*Tapirus bairdii*). Zoo Biology 13:107–117.
9. Carter, D.C. 1984. Perissodactyls. In S. Anderson and J.K. Jones, Jr., eds, Orders and Families of Recent Mammals of the World. New York, John Wiley and Sons, pp. 549–562
10. Dirzo, R.; and Miranda, A. 1991. Altered patterns of herbivory and diversity in the forest understory: a case study of the possible consequences of contemporary defaunation. In P.W. Price, T.M. Lweinsohn, G. Wilson, and W.W. Benson, eds., Plant-Animal Interactions: Evolutionary Ecology in Tropical and Temperate Regions. New York, John Wiley and Sons, pp. 273–287.
11. Downer, C.C. 1991. Semiannual report NYZS-WCS. Bronx, New York.
12. Downer, C.C. 1995. The gentle botanist. Wildlife Conservation. New York Zoological Society. August, pp. 30–35.
13. Downer, C.C. 1996. The mountain tapir, endangered flagship of the high Andes. Oryx 30:45–58.
14. Eisenberg, J.F. 1989. Mammals of the Neotropics, Vol. 1. The Northern Neotropics. Chicago, University of Chicago Press.
15. Eisenberg, J.F. 1997. Introduction. Tapirs-Status Survey and Conservation Action Plan. IUCN/SSC Tapir Specialist Group. IUCN, Gland, Switzerland and Cambridge, UK, pp.1–2.
16. Emmons, L.H.; and Feer, F. 1990. Neotropical Rainforest Mammals: A Field Guide. Chicago, University of Chicago Press.
17. Enders, R.K. 1935. Mammalian life histories from Barro Colorado Island, Panamá. Bulletin of the Museum of Comparative Zoology 73:383–502.
18. Enders, R.K. 1939. Changes observed in the mammal fauna of Barro Colorado Island, 1929-1937. Ecology 20:104–106.
19. Fragoso, J.M. 1991. The effect of selective logging on Baird's tapir. In M.A. Mares and D.J. Schmidly, eds., Latin American Mammalogy: History, Biodiversity, and Conservation. Norman and London, University of Oklahoma Press, pp. 295–304.
20. Hershkovitz, P. 1954. Mammals of Northern Colombia, Preliminary Report No. 7: Tapirs (Genus *Tapirus*), with a Systematic Review of American Species. Proceedings of the U.S. National Museum 103:465–496.
21. IUCN. 1996. IUCN Red List of Threatened Animals. IUCN, Gland, Switzerland and Cambridge, UK.
22. Janzen, D.H. 1983a. The dispersal of small seeds by big herbivores: foliage is the fruit. American Naturalist 123:338–353.
23. Janzen, D.H. 1983b. *Tapirus bairdii* (Danto, Danta, Baird's tapir). In D.H. Janzen, ed., Costa Rican Natural History. Chicago, University of Chicago Press, pp. 496–497.
24. Janzen, D.H.; and Wilson, D.E. 1983. Mammals. In D.H. Janzen, ed., Costa Rican Natural History. Chicago, University of Chicago Press, pp. 426–442.
25. MacKinnon, K. 1985. Tapirs. In D. MacDonald, ed., The Encyclopedia of Mammals. New York, Facts on File Publications, pp. 488–489.
26. March, I.J. 1992. The situation of *T. bairdii* in México. Tapir Conservation 3:3–6.
27. March, I.J. 1994. Situacion actual del tapir en Mexico. Centro de Investigaciones Ecologicas del Sureste, Serie Monogr. No. 1. Chiapas, México, San Cristobal de las Casas, 37 pp.
28. Naranjo-P., E.J. 1995a. Abundancia y uso de hábitat del tapir (*Tapirus bairdii*) en un bosque tropical humedo de Costa Rica. Vida Silvestre Neotropical 4:20–31.
29. Naranjo-P., E.J. 1995b. Hábitos de alimentacion del tapir (*Tapirus bairdii*) en un bosque tropical humedo de Costa Rica. Vida Silvestre Neotropical 4:32–39.

30. Olrog, C.C. 1979. Los mamíferos de la selva humeda, Cerro Calilegua, Jujuy. Acta Zoologica Lilloana 33:9–14.
31. Patzelt, E. 1989. Fauna del Ecuador. Quito, Banco Central del Ecuador.
32. Peres, C. 1996. Ungulate ectoparasite removal by black caracaras and pale-winged trumpeters in Amazonian forests. Wilson Bulletin 108:170–175.
33. Ramsay, P.M. 1992. The Paramo Vegetation of Ecuador: The Community Dynamics and Productivity of Tropical Grassland in the Andes. Ph.D. Thesis. University College of North Wales, Bangor, UK.
34. Roulin, X. 1829. Memoire pour servir a l'histoire du Tapir; et description d'une espece nouvelle appartenant aux hautes regiones de la Cordillere des Andes. Ann. Sc. Nat. Paris (Ire Serie)17:26–56.
35. Schauenberg, P. 1969. Contribution a létude du tapir pinchaque, *Tapirus pinchaque*. Revue Suisse de Zoologie 76:211–256.
36. Stummer, M. 1971. The wooly tapir, *Tapirus pinchaque* (Roulin) in Ecuador. Zoological Garten N.F. Leipzig 40.
37. Terwillinger, V.J. 1978. Natural history of Baird's tapir on Barro Colorado Island, Panamá Canal Zone. Biotropica 10:211–220.
38. Thornback, J.; and Jenkins, M. 1982. The IUCN Mammal Red Data Book. Part 1. IUCN, Gland, Switzerland.
39. Walker, E.P. 1964. Mammals of the World. Vol. II. Baltimore, Maryland, The John Hopkins Press,
40. Williams, K.D. 1979. Radio-tracking tapirs in the rainforest of west Malaysia. Malay Natural Journal 32:253–258.
41. Williams, K.D. 1984. The Central American tapir (*Tapirus bairdii Gill*) in Northwestern Costa Rica. Ph.D. Dissertation, Michigan State University.
42. Wilson, R.; and Wilson, S. 1973. Tapirs in captivity. Claremont, California, Tapir Research Institute.

CAPTURE METHODOLOGY AND MEDICINE

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IN NATURAL HABITAT

The capture and study of tapirs in their natural environments is required in order to improve knowledge of the biology and conservation of this group of animals. The methods used are anesthetic darting, box trapping, capture pens, and pitfalls.

A capture technique should be carefully planned in detail to minimize stress and injury to the animals and safety for the handlers. Moreover, it should be adequate to obtain the samples or data desired. Whichever method is selected, the best results are achieved by placing baits to attract the animals. A major factor is the choice of the anesthetic protocol, essential for safe manipulation of a wild tapir. A wide safety margin is of major importance since it is impossible to determine the exact body mass of the animals to be captured. The calculation of predetermined doses for body mass estimates at 50–kg intervals is usually safe for adult tapirs. Chemical restraint should be performed during the coolest time of day, and the animal must be monitored until it has fully recovered. After the intervention, the tapir should be fully capable of performing its ecological functions. It is also necessary to address contingencies for possible emergencies, as well as the disposition of animals that may become disabled during the capture process.

Anesthetic Dart-Shooting Technique

A platform should be built near a spot where bait may be placed. Compressed air or carbon dioxide guns should be used to propel the darts. The bait should be placed approximately 10 m from the platform. Avoid weapons that make a noise, as this will startle the animal. Long waiting periods should be expected. Tapirs are often active during the early morning, when poor lighting lowers the precision of both the shot and body mass estimation. Supplemental lighting may be needed. Additionally, anesthetic drugs usually require up to 15 minutes for induction. During this period, an animal may suffer trauma as a result of the beginning effects of the anesthetic, or may escape and not be found. The advantages of this method are the possibility of repeat capture of the individual, the fact that only a few field assistants are required and with minimal logistic complications.

Immobilization was used with great success during a study on *Tapirus bardii* in Central America. Bananas were used to lure the animals, which tended to remain interested in the food after being darted. Using a butorphanol/xylazine combination, sternal recumbency was achieved within 4–24 minutes (S.H. Foerster, personal communication, 1999). Problems were encountered when the same method was tried with *T. terrestris* in Brazil. None of the capture attempts were successful.[32]

Pitfall Technique

A pitfall for capturing tapirs consists of a hole 2.4 m deep, 1.5 m wide, and 2.3 m long, which is covered and camouflaged with forest debris. Holes less than 2.0 m deep may allow the animals to escape.[33] It is important to emphasize that the holes should be dug in frequently visited paths or near bait stations.

Use of this technique is controversial. The tapir may fracture a limb, more than one animal may be caught at a time, manipulation of the captured animal inside the hole is difficult, and habitat disturbance and local geologic conditions must be considered. Advantages are that the traps are unnoticeable and the same animal can be repeatedly captured. Also, after the animal is caught, there is time enough for the animal to be manipulated at the most suitable moment. The animals usually remain calm. It is easier to estimate the body mass and shoot anesthetic darts precisely. The impossibility of escape after the first shot allows the design of safer protocols, with correct administration of preanesthetic drugs and ability to hold the animal until recovery is complete.

This method has proved successful and safe for the capture of eight tapirs, to which radio collars have been attached. The anesthetic agents were injected by using a blowgun to propel a syringe dart with a 0.40- × 0.12-cm needle. The best site for darting a tapir in the pitfall is the dorsal portion of the neck (E.P. Medici and P.R. Mangini, personal communication, 1999).

In a capture project in the Atlantic rain forest, a female tapir and her 2- or 3-month-old calf were captured in a pitfall. Both tapirs remained calm. Before starting to handle the female, the calf was removed from the pitfall. After standard procedures were completed, the animals were reunited and released with no physical disturbance, demonstrating the efficacy of the method even under increased risk conditions (P.R. Mangini, personal communication, 1999).

To release a tapir captured by this method, a slope must be produced at one of the pitfall walls to allow the animal to walk out of the pitfall as soon as it has completely recovered from the immobilization.

Box Trapping

Wooden or metal boxes with two doors located on opposite sides are used in this technique. As the tapir attempts to pass through the open box, a trigger is pulled, and the doors fall simultaneously, with the animal inside. The traps are placed on natural tapir tracks with food items inside to attract the animals. The main advantages of such a technique is that the animal is close enough to be easily reached, manipulated, or injected with anesthetic drugs. It prevents escapes and is a practical method for capturing animals to be relocated. However, it is ineffective if the box is too small (2.2 × 1.1 × 1.1 cm). In addition, some tapirs are reluctant to enter a box, even one with both doors held open.[32]

Capture Pens

These are excellent traps for lone or grouped ungulates. For tapirs, they must be built with posts more than 10 cm (4 in.) in diameter, with boards thicker than 2.5 cm (1 in.). The walls, as for the pitfalls, should be at least 2.4 m high to prevent escapes. The lateral dimensions may be about 2 × 3 m, preventing captured individuals from excessive movement. Automatic-action guillotine doors improve efficacy. Captured tapirs may be easily darted, preferably in the dorsal portion of the neck or the thigh muscles. The accidental capture of tapirs has been reported, with corncob baits in pens built for peccaries. The pens were placed in a semideciduous forest in southern Brazil (T.C.C. Margarido, personal communication, 1997).

MANAGEMENT IN CAPTIVITY

Tapirs are relatively easy to maintain in captivity, but care and handling of these animals requires basic knowledge of its biology, physiology, and behavior. Ignorance of these characteristics is the primary cause for most of the medical problems. Tapirs are hardy animals that may be found in many zoos in South America and elsewhere throughout the world. Table 32.2 shows occurrence of tapirs in the zoos of Brazil.

Behavior and Social Groups

Tapirs are predominantly nocturnal, but also may be active during the day, mainly during early morning and evening hours, remaining in shaded areas during the hottest hours of the day. Tapirs have been grouped for exhibit with many different animals, including the guanaco (*Lama guanacoe*), llama (*Lama glama*), Asian elephant (*Elephas maximus*), agouti (*Dasyprocta* spp.), capybara (*Hydrochaeris hydrochaeris*), peccary (*Tayassu* spp.), Patagonian cavy (*Dolichotis patagonun*), emu (*Dromaius novaehollandiae*), and rhea (*Rhea americana*).[2,5,8,24,28] The capacity to live in harmony with other animals varies with an animal's temperament,

TABLE 32.2. Distribution of tapir *Tapirus terrestris* in Brazilian zoos: population, birth rate, and mortality in the years 1996–1998

Year	Zoos	Population	Birth Rate	Mortality
1996	39	110	11 (10.0%)	8 (7.27%)
1997	28	81	13 (16.0%)	8 (9.88%)
1998	31	80	6 (7.5%)	13 (16.25%)

Source: Census of the Brazilian Zoo Society 1996/1997/1998.

food availability, and size, site and design of the enclosure. Usually docile, at times tapirs may exhibit great aggressiveness, both intra- and interspecific. Some will not accept a partner, whereas others live harmoniously in groups as large as 10 individuals.[3] Tapirs defend themselves by biting in a rapid lateral movement. When biting, they grasp and drag the teeth off, causing severe contusions, as may be attested to by many handlers who have been injured, even to the extent of losing fingers.[3] The authors saw a tapir cause a wound 20 cm in length in the neck of a rhea that insisted on harassing it. An adult male tapir fought with a male white-lipped peccary from a neighboring enclosure, resulting in serious wounds to both animals.

Tapirs appreciate being scratched, the more intensely better. The neck, jaw, belly, and the inner sides of the legs are the preferential areas. Some remain immobile, others lie down, stretching out the legs in utter ecstasy. Some veterinary procedures may be carried out while scratching.[24]

Females should be isolated in a quiet place before parturition, because sometimes the male kills the newborn. However, a male licking a newborn while the female recovered from parturition has been observed (R.R. Mangini, personal communication).

Enclosures

Tapirs are active animals and should be provided with space to move about. Legal regulations for zoos in Brazil require an area of 500 m^2 for a pair,[19] whereas Kuehn[24] suggests at least 200 m^2 for each animal. The most appropriate space will depend on individuals' compatibility. Enclosures should be large enough to permit an animal to escape aggression or during courtship.

In general, in South America it is not necessary to have a heated shelter, as is necessary in cold-climate countries. Many locals offer just a covered area large enough to protect the animal and its food from the sun and the rain. Brazilian law requires a shelter 20 m^2 for a pair,[19] whereas Barongi[4] indicates an area of 9.3 m^2 for each individual, increased by 50% if a calf is present. Larger groups need two or more feeding points, equally protected.[25] Strong correlation exists among corneal opacity and ulcers and enclosures with little protection from direct sunshine.[24] Pens appended to main enclosures are important for handling aggressive animals, separation of pregnant females, and loading into transport boxes. The internal walls of such a pen should be 1.8 m (6 ft) in height.

Pool

A pool for bathing is essential for tapirs. They play, refresh themselves, defecate, and copulate in water. Lack of a pool is correlated with skin and hoof problems and rectal prolapse caused by persistent constipation.[2,3,24,37] The pool should be deep enough to allow total submersion of an adult, which means it must be 1–2 m (6.5 ft) deep.[3] The surface area should be 33 m^2 or 50 m^2, according to Brazilian regulations.[2,19] The ramp for entrance and exit should be safe, without corners, to prevent slipping and wounds. More than one access ramp is desirable, to allow escape from aggression.[25] The pool should be drained and cleaned at least once a week. Besides the bathing pool, fresh drinking water should be constantly available. A concrete drinking water container is adequate.

Fences and Moats

Tapirs are unable to jump, but they can climb fences or walls of less than 1.5 m (5 ft). If the fence is not strong, a tapir may collapse it. Wire fences should be at least 2 m (6.5 ft) high.[3,24] The public should be kept 1 m (5 ft) distant from the fence. Moats should be at least 1.5 m (5 ft) wide and 1.8 m (6 ft) deep. A moat may be dry or filled with water.

Substrate

The floor of shelters may be of smooth concrete. The surface of the outside enclosure should be sand or grass. Tapirs should never be kept on hard floors for a long time. Hard or stony soil has been correlated with chronic lameness.[2,3,24,42]

Feeding

South American tapir species are basically herbivorous, and a variety of food items may be offered to captive animals. Tapirs may be fed with commercial feed for equids, which contains about 12-14% protein. An adult animal consumes about 1–1.5 kg of dry commercial food in a day. Wheat bran may be fed to prevent rectal prolapse (M.L. Javorouski, personal communication, 1999).

Fruits and vegetables are important feed items, including bananas, apples, carrots, green corn, cabbage, kale, melons, pumpkins, spinach, sweet potatoes, and celery. These items may compose 5–12% of the total diet. Bananas are much appreciated and may be good for attraction, amusement, or as a vehicle for medications. Good quality hay (alfalfa, grass) should be offered ad libitum. Grain may also be offered. In South America, sugarcane is offered in the winter (C. Giacomini, J.A.B. Bastos, and C.E.P. Saad, personal communication, 1999).

Horse or cattle mineral mixes may be offered ad libitum (J.A.B. Bastos and C.E.P. Saad, personal communication, 1999). A powdered vitamin mix should be added once a week.

Transportation

The transport box should be large enough to allow the animal to lie down and stand up, but it should not be able to turn around. Tapirs may be driven into transport boxes with wooden panels or opaque plastic sheeting. The ideal is to put the box with two doors at opposite ends inside the enclosure, in a place where the animals pass frequently, open the doors, and place food inside. Alternatively, feces may be placed inside of the box, making the environment more familiar. In that way the animal will become habituated to the box and enter it without resistance. Tapirs should always be transported individually. The box should be placed obliquely in the transport vehicle, to avoid trauma to the head or back when the vehicle brakes. The animal must be transported in the coolest hours of the day. On long trips, fresh grass and drinking water should be offered and the animal sprayed with water periodically.

MEDICAL MANAGEMENT

Adult body weight of American species of tapirs varies from 160 to 317 kg (353–700 lbs).[4,24] The internal anatomy is similar to that of the domestic horse, including a guttural pouch, located in the pharyngeal region; absence of a gall bladder; a small stomach; large cecum and colon; and nonlobular kidneys. They have four toes on the front feet and three on the rear feet.

Clinical Examination

Abdominal and thoracic auscultation are difficult because of the thickness of the skin, but they can be accomplished. Rectal palpation may be accomplished under sedation or it may require complete immobilization. Rectal temperature may be taken while scratching or offering a tasty food. Table 32.3 lists physiologic parameters of *Tapirus terrestris* at rest.

Blood samples may be collected from the medial saphenous or cephalic veins. At these sites, the skin is not as thick as in other areas of the body. Access to the jugular vein is more difficult, but it is available for sampling if large volumes of blood are required.[20] Some docile animals permit venipuncture during lateral recumbency induced by scratching the back, neck, or belly, without physical or chemical restraint.

Intramuscular injections may be given in the cervical or gluteal area and subcutaneous injections in the skin behind the ear. Oral medications should be given in a favorite food. Some physiologic parameters and serum biochemical values of Brazilian tapirs before and after sedation are available in Tables 32.4, 32.5, 32.6, and 32.7.

Physical Restraint[24,26,36]

Forced physical restraint is impracticable. Scratching behavior was described previously. Wooden panels can be used to press animals against a wall to facilitate examination.

Immobilization and Anesthesia

Determination of an individual's exact body weight is an obstacle to chemical restraint of wild or captive tapirs. Most institutions in Latin America lack scales with the capacity to weigh tapirs. When scales are not available, reasonable estimates may be made. In adult American species of tapirs, the corporal mass may vary from 160 to 317 kg.[3,24]

Chemical restraint is usually indicated in order to perform clinical or surgical procedures, transport animals, or conduct research. Sedative and anesthetic protocols are listed in Table 32.8.

Miscellaneous Conditions and Preventive Medicine

Most of the information available regarding diseases in tapirs was obtained from specimens in captivity in Europe and North America. Generally, Tapiridae are affected by the same pathogens as other Perissodactyla (Equidae and Rinocerontidae). Most of the diagnostics and therapeutics applied to tapirs are based on those developed for domestic equids.[35]

During the past 10 years, 101 medical problems were observed in a tapir-breeding facility in southern Brazil. Problems included cutaneous lesions (50.49%), parasitic observations (36.63%), musculoskeletal disturbances (6.94%), fatal diseases (3.96%), and other alterations in health (1.98%).[29] These data were different

TABLE 32.3. Physiologic parameters of the Brazilian tapir (*Tapirus terrestris*)

Respiratory Rate (/min.)	Heart Rate (/min.)	Rectal Temperature (°C)
20.5 ± 6.24	54.3 ± 1.88	37.1 ± 0.60

Source: Data from A.L.V. Nunes, unpublished data.

TABLE 32.4. Mean blood values of Brazilian tapir

RBC ($\times 10^6$)	Hb (gm/dL)	PCV (%)	MCV (μm^3)	MCH	MCHC	WBC ($\times 10^3/\mu L$)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Basophils (%)	Eosinophils (%)
						($n = 15$)					
4.6 ± 1.1	9.4 ± 2.6	27.4 ± 6.7	59.4 ± 3.9	20.3 ± 2.2	34.3 ± 3.3	6.2 ± 2.0	52.9 ± 17.0	29.7 ± 15.0	2.4 ± 1.9	0.4 ± 0.5	13.8 ± 8.3

Source: J.R.V. Ferreira, M.L. Cruz, A.L.V. Nunes (unpublished data). RBC, red blood cells; Hb, hemoglobin; PCV, packed cell volume; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; WBC, white blood cells.

TABLE 32.5. Venous blood gases, pH, hematocrit, hemoglobin, electrolytes, plasma glucose, and cortisol concentrations in conscious *Tapirus terrestris*

Parameters	Average ± SD	*n*	Parameters	Average ± SD	*n*
PvO_2 mm Hg	71 ± 7	4	HCO_3 mmol/L	22 ± 1	4
$PvCO_2$ mm Hg	35 ± 2	4	Calcium mg/dL	4 ± 2	4
Total CO_2 mmol/L	24 ± 0	2	Potassium mmol/L	3 ± 0	3
O_2 saturation %	93 ± 3	4	Sodium mmol/L	131 ± 2	4
pH	7.41 ± 0.03	4	Glucose mg/dL	71 ± 6	3
Hematocrit %	34 ± 1	3	Cortisol nmol/L	41 ± 15	4
Hemoglobin g/dL	11 ± 0	3			

Source: See reference 10.
SD, standard deviation; *n*, number of animals.

TABLE 32.6. Heart and respiratory rates and temperature (average ± SD) of Brazilian tapir before and after detomidine sedation

	Before Sedation	20 Min. After Sedative Administration	40 Min. After Sedative Administration
Heart rate (beats/min)	60 ± 15.4	63 ± 14	62 ± 11
Respiratory rate (breaths/min)	31 ± 9	30 ± 12	29 ± 10
Rectal temperature °C	36.8 ± 0.6	36.9 ± 0.7	36.9 ± 0.7

Source: See reference 28.
Average ± standard deviation; $n = 28$.

TABLE 32.7. Serum biochemical values in Brazilian tapirs before and after detomidine sedation

Parameter	Before Sedation	*n*	10 Min. After Detomidine Administration	*n*	20 Min. After Detomidine Administration	*n*	40 Min. After Detomidine Administration	*n*
Bilirubin, mg/dL[a]	0.66	07	0.53	15	0.61	15	0.56	13
Glucose, mg/dL[a]	95.76 ± 9.83	07	87.13 ± 22.1	15	93.73 ± 22.2	15	101.02 ± 29.4	13
Urea, mg/dL[a]	30.3 ± 2.62	07	28.05 ± 3.01	15	27.77 ± 2.73	15	2873 ± 3.33	13
Creatinine, mg/dL[a]	1.16 ± 0.15	07	1.06 ± 0.15	15	1.09 ± 0.23	15	1.09 ± 0.16	13
AST, U/L[a]	37.24 ± 11.4	07	46.43 ± 28.1	15	43.88 ± 28.4	15	45.53 ± 27.4	13
ALT, U/L[a]	6.5 ± 3.84	07	7.9 ± 7.32	15	7.52 ± 6.98	15	8.45 ± 9.21	13
Total protein, g/dL[b]	7.55 ± 1.91	10	7.45 ± 2.70	26	8.32 ± 2.30	27	8.14 ± 1.87	24
Albumin, g/dL[b]	2 ± 1.59	10	1.86 ± 0.75	26	1.98 ± 0.83	27	1.88 ± 0.77	24
Phosphorus, mg/dL[b]	4.28 ± 2.69	10	4.51 ± 2.69	26	4.44 ± 2.18	26	4.12 ± 2.35[c]	24
Calcium, mg/dL[b]	11 ± 4.74	10	11.82 ± 3.52	26	11.34 ± 3.21	26	10.94 ± 3.56	24
Magnesium, mg/dL[b]	0.87 ± 0.57	10	0.19 ± 0.78	26	0.82 ± 0.74	27	0.73 ± 0.69	24
Phosphatase alkaline, U/L[b]	7.26 ± 5.45	10	11.02 ± 10.8	26	11.07 ± 9.62[c]	26	12.05 ± 10.4[c]	24

Source: See reference 13.
[a]Reflotron method.
[b]Labtest method.
[c]Difference when compared with before sedation ($P < 0.05$).
Average ± standard deviation; *n*, number of sampled animals.

than the most common diseases observed in North American institutions.[20] The most common medical problems in tapirs kept in zoos in Brazil are cutaneous wounds, constipation, rectal prolapse, lameness from trauma or hoof lesions, and corneal opacity (J.A.B. Bastos, C. Giacomini, M.L. Javorousky, and A.L.V. Nunes, personal communication, 1999). All of the above may have high correlation with environmental problems in the facilities.

Diseases caused by enteric bacteria, tuberculosis, ecto- and endoparasitosis, tetanus, and cutaneous vesicles are often suggested as being associated with lack of sanitation.[6,9,31,39] Unfortunately, because of diagnostic

TABLE 32.8. Anesthetic protocols used in Tapirus spp.

Protocols	Parameters	Comments	*n*	Reference
Etorphine (10 µg/kg) IM	Chemical restraint	Traditionally used.	—	20
Carfentanil (20 µg/kg) IM	Chemical restraint	Attention to synergistic effects of the combination with xylazine.	—	20
Buthorphanol (0.15 mg/kg) IM Xylazine (0.3 mg/kg), IM or Detomidine (0.05 mg/kg), IM Further restraint: Ketamine (0.5 mg/kg) IV	Sedation Heart rate: 30–55/min. Oxygen saturation 90–95%	Good relaxation in 10 min. Reversal: Yohimbine (0.2–0.3 mg/kg) IV plus narcotic antagonist. Rapid, smooth, and complete recovery.	19	20
Azaperone (1.0 mg/kg) IM	Sedation	Minor standing procedures	—	21
Xylazine (1.0 mg/kg) IM	Sedation	Minor standing procedures, less effective	—	21
Xylazine (3.6–4.5 mg/kg) IM After sedation: Ketamine (3.6–4.5 mg/kg) IV	Sedation for anesthesia	Surgical plane, with good relaxation and analgesia to premolar extraction.	1	26
Immobilization: Detomidine (0.13 mg/kg) IM Butorphanol (0.2 mg/kg) IM Ketamine (2.2 mg/kg) IV After 20 minutes, add ketamine (1.5 mg/kg) IV, perform endotracheal intubation, maintain anesthesia with isoflurane	Anesthesia	Rapid and smooth recovery. Hypoventilation. Sufficient arterial pressure.	1	43
Induction: Atropine (1 mg) IM Etorphine (1–2 mg) IM Maintenance: supplementary doses of etorphine or gas such as halothane or methoxyflurane	Induction: 15–30 min.	Immobilization in standing position or lateral recumbency. Reversal agent: diprenorphine IV. Should regain standing position within 1–30 min.	—	24

n, number of sampled animals.

challenges or incomplete knowledge about etiopathological processes in tapirs, it is impossible to determine the exact etiology of a large number of clinical diseases and death.

Vaccination has not been regularly used in tapirs; it has been limited to a few high-risk situations. Vaccination protocols for tapirs have not been evaluated for effectiveness, but inactivated vaccines are appropriate for use against tetanus, infectious equine encephalitis, clostridial diseases, rabies, and leptospirosis. It is particularly important to immunize tapirs and other wild ungulates against tetanus, especially those living close to livestock.

A new arrival should be quarantined for at least 30 days, given a thorough physical examination and blood and feces collected for laboratory analysis. These samples then serve as a reference point should the tapir become ill later.

Serum should be tested for bluetongue, infectious bovine rhinotracheitis, equine encephalomyelitis, equine infectious anemia, foot and mouth disease, brucellosis, and leptospirosis. Intradermal tuberculin testing should be performed.

Infectious Diseases

Tapirs have no unique infectious diseases. Ubiquitous organisms that have been isolated from disease processes include *Pasteurella* spp., *Corynebacterium pyogenes, Actinomyces* spp., *Necrobacillus* spp., and *Escherichia coli*. Positive serologic responses to poxvirus, foot and mouth disease, bluetongue, infectious bovine rhinotracheitis, and eastern equine encephalomyelitis have been reported.

Parasitic Diseases

As in other species, tapirs are host to numerous parasites, but clinical parasitism is not a common problem. Nematodes include *Strongyloides* spp., *Strongylus* spp., *Ascaris* spp., and *Capillaria* spp.[15,24] Protozoa reported include *Ballantidium, Giardia, Babesia,* and *Trypanasoma* spp.[15,31,37,44] External parasites include

Sarcoptes spp. (sarcoptic mange) and ticks (*Amblyomma* spp.).[22,23,30,31,35,37]

Surgical Problems

Common surgical problems include lacerations, abscesses and trauma. These are managed as in the domestic horse.

Rectal prolapse has been a common cause of death. Predisposing factors may include absence of bathing, feeding whole fruits and vegetables instead of diced portions, and feeding indigestible hay. It may also be caused by ingestion of sand and any other factor that causes diarrhea or constipation.

When a prolapse occurs, the exposed tissue must be cleaned, replaced, and sutured, using a temporary purse string suture. Previously, it was recommended that granulated sugar be applied to reduce edema, but recent studies have shown that the sugar crystals may lacerate and damage the mucosa. In case of severe damage to the mucosa, the necrotic tissue may be amputated, the edges sutured, and then replaced. Sometimes it may be necessary to suture the terminal portion of the colon to the ventral body wall to prevent reprolapse.[24] Even if the surgery is successful, the animal may die because of toxemia.[37]

At Curitiba Zoo, recurrent vaginal prolapse was observed following estrus, probably caused by hormonal imbalance. Surgical prolapse reduction and an ovariohysterectomy were satisfactory, without reoccurrence (M.L. Javorousky, personal communication, 1999).

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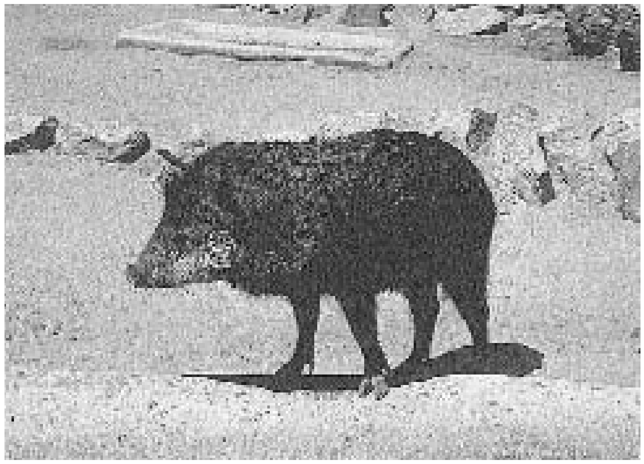
REFERENCES

1. Alexander, I.D. 1978. Actinomyces infection in a Tapir *Tapirus terrestris*. Journal of Zoo Animal Medicine 9(4):124–126.
2. Tapir Research Institute. 1971. Results of a Survey of Captive Tapirs Taken by the Tapir Research Institute Between July of 1970 and March of 1971. California, Tapir Research Institute, pp. 1–22.
3. Barongi, R. 1986. Tapirs in captivity and their management at Miami Metrozoo. In Proceedings of the American Association of Zoological Parks and Aquariums. Minneapolis, pp. 96–108.
4. Barongi, R. 1999. AZA Zoo Standards for Keeping Tapirs in Captivity. Manuscript.
5. Bonney, S.; and Crotty, M.J. 1979. Breeding the mountain tapir *Tapirus pinchaque* at the Los Angeles Zoo. International Zoo Yearbook 19(1):198–200.
6. Buschinelli, M.C.P. 1990. Quimioprofilaxia e quimioterapia em antas *Tapirus terrestris* (LINNé, 1758) acometidas por tuberculose. In Proceedings of the 14° Congresso da SZB. Belo Horizone, Brazil, Sociedade de Zoológicos do Brasil, p. 21.
7. Sociedade de Zoológicos do Brasil. 1996–1998. Censo de Animais em Cativeiro. Bauru, Brazil, Sociedade de Zoológicos do Brasil.
8. Crotty, M.J. 1977. The year of the tapir. Zoo News 12(1):10.
9. Cubas, Z.S. 1996. Special challenges of maintaining wild animals in captivity in South America. In M.E. Fowler, ed., Wildlife Husbandry and Disease: Scientific and Technical Review. Paris, Office International des Epizooties, pp. 267–282.
10. Ferreira, J.R.V. 1997. Anestesia Intravenosa Contínua em *Tapirus terrestris* (Anta Brasileira) com Associação de Detomidina ou Xilazina com Midazolam e Ketamina [Continuous intravenous anesthesia in Brazilian tapir with combination of detomidine or xylazine, with midazolam and ketamine]. Masters thesis, Universidade Federal do Paraná.
11. Ferreira, J.R.V.; and Luna, P.L. In press. Anestesia em *Tapirus terrestris* (anta brasileira). Muriqui, ABRAVAS.
12. Ferreira, J.R.V.; Cruz, M.L.; Nunes, A.L.V.; and Adauto, L.V. 1994. Sedation of *Tapirus terrestris* (Brazilian tapir) using detomidine and antagonism by yohimbine. In Proceedings of the 1st Veterinary Anesthesia Meeting. Foz do Iguacu, Brazil, Socidade de Zoologicos do Brasil, p. 80.
13. Ferreira, J.R.V.; Cruz, M.L.; and Nunes, A.L.V. 1995. Valores Bioquímicos séricos de *Tapirus terrestris* (anta brasileira), antes e após sedação pela detomidina (Biochemistry values before and after detomidine sedation in *Tapirus terrestris* (Brazilian tapir)). In Proceedings of the 1° Encontro Internacional da SZB e 2° Congresso Brasileiro da Sociedade de Zoológicos do Brasil. Goiania, Brazil, Socidade de Zoológicos do Brasil, p. 19.
14. Finnegan, M.; Munson, L.; Barret, S.; and Calle, P.P. 1993. Vesicular skin disease of tapirs. In Proceedings of

the American Association of Zoo Veterinarians. Saint Louis, pp. 416-417.
15. Gale, N.B.; and Sedgwick, C.J. 1968. A note on the wooly tapir (*Tapirus pinchaque*) at Los Angeles Zoo. International Zoo Yearbook 8(1):211–212.
16. Hertzog, R.E. 1975. Xylazine in exotic animal practice. In Proceedings of the American Association Zoo Veterinarians. pp. 40–43.
17. Horan, A. 1983. An outline of tapir management. Proceedings of the Symposium of the Association of British Wild Animal Keepers 7:24–29
18. Hugues, F.; Leclerc-Cassan, M.; and Marc, J.P. 1986. Anesthésie des animaux non domestiques: Essai d'un nouvel anesthésique: l'association tilétamine-zolazepam (Zoletil N.D.). Recerche en Médicine Vétérinaire 162(3):427–431.
19. IBAMA. 1989. Instrução normativa No. 001/89-P de 19 de Outubro de 1989. In Legislação sobre Zoológicos. Brasilia, IBAMA, pp. 9–27.
20. Janssen, D.L.; Rideout, B.A.; and Edwards, M.S. 1996. Medical management of captive tapirs (*Tapirus* sp.). In Proceedings of the American Association of Zoo Veterinarians. Puerto Vallarta, Mexico, pp. 1–11.
21. Janssen, D.L.; Rideout, B.A.; and Edwards, M.S. 1998. Tapir medicine. In M.E. Fowler and R.E. Miller, eds. Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 562–568.
22. Jones, E.K.; Clifford, C.M.; Keirans, J.E.; and Kohls, G.M. 1972. The ticks of Venezuela (Acarina: Ixodidoidea) with a key to the species of *Amblyomma* in the Western Hemisphere. Brigham Young University Science Bulletin (Biological ser.) 17(4):40.
23. Knight, J.C. 1992. Observations on potential tick vectors of human disease in the cerrado region of central Brazil. Revista da Sociedade Brasileira de Medicina Tropical 25(2):145–146.
24. Kuehn, G. 1986. Tapiridae. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 931–934.
25. Lee, A.R. 1993. Management Guidelines for Welfare of Zoo Animals: Tapirs (*Tapirus* sp). London, The Federation of. Zoological Gardens of Great Britain and Ireland, p. 51.
26. Lopez, N.; Tome, F.; and Ciccio, M. 1998. Extración de un premolar de um tapir. Isondú, Associacion Argentina de Veterinários de Animales Silvestres 3(5):6–9.
27. Lozano-Alarcon, F.; Bradley, G.A.; Houser, B.S.; and Visvesvara, G.S. 1997. Primary amebic meningoencephalitis due to *Naegleria fowleri* in a South American tapir. Veterinary Pathology 34(3):239–243.
28. Mahler, A.E. 1984. Activity budgets and use of exhibit space by South American tapir (*Tapirus terestris*) in a zoological park setting. Zoo Biology 3(1):35–46.
29. Mangini, P.R.; and Medici, E.M. 1998. Utilização de associação de cloridrato de medetomidina com cloridrato de tiletamina e zolazepam na contenção de *Tapirus terrestris* em vida livre(Relato de dois casos [Chemical restraint of wild Brazilian tapir with medetomidine, tiletamine/zolazepam combination(Two case reports]. In Proceedings of the 22° Congresso Brasileiro e 4° Encontro Internacional da SZB. Salvador, Brazil, Sociedade de Zoológicos do Brasil, p. 78.
30. Mangini, P.R.; Sinkoc, A.L.; and Medici, E.P. 1998. Relato da ocorrência de *Amblyomma cajannense* em antas (*Tapirus terrestris*) de vida livre, no parque estadual do Morro do diabo, SP [Occurrence of *A. cajannense* in wild tapirs in State Park of Morro do Diabo, SP]. In Proceedings of the 22° Congresso Brasileiro de Zoologicos e 4° Encontro Internacional de Zoológicos. Salvador, Brazil, Sociedade de Zoológicos do Brasil, p. 59.
31. Mangini, P.R.; Moraes, W.; and Santos, L.C. 1999. Enfermidades observadas em *Tapirus terrestris* (anta brasileira) mantidas em cativeiro em Foz do Iguaçu, Paraná [Diseases in captivity *Tapirus terrestris,* in Foz do Iguaçu, Paraná, Brazil]. Arquivos de Ciências Veterinárias e Zoologia da Unipar-Universidade Paranaense, Brazil (in press).
32. Medici, E.P. 1999. Biologia da Conservação da Anta (*Tapirus terrestris*) no Parque Estadual do Morro do Diabo, Instituto Florestal de São Paulo [Conservation biology of Brazilian tapir in Morro do Diabo, Brazil]. Project Report. Instituto de Pesquisas Ecológicas. São Paulo, Nazaré Paulista.
33. Medici, E.P.; and Mangini, P.R. 1998. Avaliação da utilização da metodologia de Trincheiras para captura de *Tapirus terrestris* em vida livre [Evaluation of pitfall technique to capture wild *Tapirus terrestris*]. In Proceedings of the 22° Congresso Brasileiro de Zoológicos e 4° Encontro Internacional de Zoológicos. Salvador, Socidade de Zoológicos do Brasil, p. 180.
34. Miller-Edge, M.; Amsel, S.; and Junge, R.E. 1994. Carfentanil, ketamine, xylazine combination (CKX) for immobilization of exotic ungulates: clinical experiences in bongo (*Tragelaphus euryceros*) and mountain tapir (*Tapirus pinchaque*). In Proceedings of the American Association of Zoo Veterinarians and Association of Reptilian and Amphibian Veterinarians. Pittsburg, Pennsylvania, pp. 192–196.
35. Ramsay, E.C.; and Zainuddin, Z.Z. 1993. Infectious diseases of the rhinocerus and tapir. In M.E Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 459–466.
36. Read, B. 1986. Breeding and management of the Malayan tapir *Tapirus indicus* at St. Louis Zoo. International Zoo Yearbook 24–25:294–297.
37. Reichel, K. 1982. Tapirs. In H.G. Klos and E.M. Lang, eds., Handbook of Zoo Medicine. New York, Van Nostrand Reinhold, pp. 186–193.
38. Robinson, L.E. 1926. Ticks, A Monograph of the Ixodoidea, Pt. 4. The Genus *Amblyomma*. London, Cambridge University Press, p. 302.
39. Salles, A.R.C. 1995. Viabilidade do tratamento da tuberculose em antas (*Tapirus terrestris*) [Tuberculosis treatment of tapir]. In Proceedings of the 17° Congresso Brasileiro e 1° Encontro Internacional da SZB. Goiânia, Brazil Sociedade de Zoológicos do Brasil, p. 44.

40. Santos, J.A.; and Costa, J.E. Tuberculose em anta, *Tapirus terrestris*. [Tuberculosis in tapirs]. In Proceedings of the 3° Congresso da SZB. Sapucaia do Sul, Brazil, p. 21.
41. Schonborn, C.; et al. 1971. Untersuchungen zur Pathophysiologie der Schilddruse des Wellensitichs, Vols. 1–5. Mitteilung, XIV. VISZ, pp. 123–144.
42. Stummer, M. 1971. Wolltapire, *Tapirus pinchaque* (Roulin) in Ecuador. Der Zoologishe Garten (Leipzig) 40(3):148–159.
43. Trim, C.M.; Lamberski, N.; Kissel, D.I.; and Quandt, J.E. 1998. Anesthesia in a Baird's tapir (*Tapirus bairdii*). Journal of Zoo and Wildlife Medicine 29(2):195–198.
44. Vroege, C.; and Zwart, P. 1972. Babesiosis in Malayan tapir (*Tapirus indicus* Desmarest, 1819). Z. Parasitenkde. 40(1):177–179.
45. Yamini, B.; and Veen, T.W.S. 1988. Schistosomiasis and nutritional myopathy in a Brazilian tapir (*Tapirus terrestris*). Journal of Wildlife Diseases 24(4):703–707.



33
Order Artiodactyla, Family Tayassuidae (Peccaries)

Teresa Cristina Castellano Margarido
Paulo Rogerio Mangini

BIOLOGY AND MEDICINE

NATURAL HISTORY

Taxonomy and Evolution

The South American "wild pigs" belong to the family Tayassuidae, order Artiodactyla. The family, Tayassuidae, known since the 17th century, has created controversy as far as nomenclature is concerned.[70] Generally, white-lipped and collared peccaries are still included in the genus *Tayassu,* [72] however, according to Wilson and Reeder[83] the family Tayassuidae is presently represented by three genera and three species; *Catagonus wagneri* (giant Chaco peccary), *Tayassu pecari* (white-lipped peccary), and *Pecari tajacu* (collared peccary). *C. wagneri* was first described by Rusconi in 1930,[70] who then believed that the species had been extinct in the beginning of the Holocene. However, in 1975, Wetzel reported the species still lived in the Gran Chaco in Paraguay.[77,78]

Geographical Distribution

The species in this family are distributed from Texas, in North America, to northern Argentina. *P. tajacu* occupies the widest range, occurring in the southern United States, Central America, Colombia, Venezuela, Ecuador, Guyana, Brazil, and Northern Argentina. *T. pecari* occurs in southern Mexico, Venezuela, Ecuador, Guyana, Brazil, Paraguay, and northern Argentina.[11] *C. wagneri* is endemic in the xerophytic forests of the Gran Chaco in Paraguay, Argentina, and Bolivia. Although first described as a fossil, it was rediscovered as a living form by Wetzel, in 1974.[82, 21]

Social Structure

Catagonus wagneri lives in small family units and, except for the size of the groups, resembles the other peccaries in behavior.[58] *P. tajacu* is a highly sociable species, usually forming groups from 6 to 12 individuals,[19] although groups of up to 50 individuals have been observed in Venezuela.[13] White-lipped peccaries prefer dense and humid forests, and they may travel long distances.[19] They are mostly gregarious and active during the day, and the size of the groups is much larger than in *P. tajacu,* generally more than 40, but even up to 300 individuals.[22] There seems to be a linear hierarchical dominance involving both sexes. The highest positions in a group are mostly occupied by males, but dominant females were observed in several captive groups. There is no couple or harem formation. Instead, dominance hierarchy gives reproductive priority to the dominant males.[75]

Habitat and Feeding

The tayassuids live in a great variety of habitats, including desert vegetation and arid and humid forests, where the animals are more active at night or during cooler times of day.[75] Although usually considered to be humid tropical forest dwellers, white-lipped peccaries are also found in seasonally dry areas in southern South America, but with smaller density. This species has been reported to exist at a gross biomass of 44 kg per square kilometer in the Brazilian pantanal swamplands.[71] The collared peccaries occur in a wide variety of habitats, from the humid tropical forest to the Paraguayan Gran Chaco, a xeric environment.[68] The giant peccary is found in the Chaco Seco in southern South America, in areas with high temperatures and low rainfall, more frequently in forests with short vegetation in Paraguayan Central Chaco.[78] *P. tajacu* is plainly sedentary and does not seem to leave its birthplace, whereas *T. pecari* travels such long distances that it is often considered as a nomad or migrating species.[75,63]

Tayassuids feed mainly on roots, leaves, tubercles, fruits, seeds, cactuses, and, occasionally, small vertebrates. These animals play an important role in floral dispersion. The white-lipped peccary is often described as being an omnivore, but also a frugivore, hence its diet may consist 60% of fruits in which the seeds are generally consumed. Collared peccaries in the humid tropical forest consume mostly the seeds and fruits of palm trees and diverse vegetable material such as leaves and culms. The diet is complemented with insects.[7,8,45] In Caatinga, a Brazilian xeric environment, *P. tajacu,* the generalist among tayassuid species, feeds equally on roots and seeds.[65] In Mato Grosso's Pantanal, Brazil, a subarea of Nhecolândia, direct observation and data supplied by inhabitants and local researchers indicate that peccaries are important annelid predators.[72] *T. pecari* diet is composed of seeds, fruits and roots, invertebrates, small vertebrates, carrion, and fungi, which makes the animal a major seed dispersor,[8] although it destroys many by breaking the embryo. This capacity to break seeds and chestnuts harder than those consumed by *P. tajacu* is regarded as one of the primary factors for the division of ecological niches in areas where white-lipped and collared peccaries overlap.[8,46,65]

Relevant Aspects of Tayassuidae Conservation

All the members of this family are hunted, and they are disappearing in certain areas because of hunting and habitat destruction. The cougar (*Puma concolor*) and the jaguar (*Panthera onca*)are the major predators of wild peccaries. However, humans are the only predator that can adversely impact on peccaries and their habitat. Large-scale hunts are characterized by absence of selection concerning classes of age and sex, injury of animals that cannot recover, and separation of litters of small piglets from nursing females. These practices often eliminate large numbers of the group, interrupt the social organization, and adversely affect herd survival, occasionally contributing to local extinction of the species.

C. wagneri is hunted for food and for its skin. The fact that these animals visit cattle salt licks and the tendency of the group members to stay close together has contributed to massive deaths of this species, and its interrupted distribution seems to reflect the intensive hunting.[68] Until the mid-1970s, the giant Chacon peccary was abundant within its distribution limits, but its number had declined by the end of the 1980s, due to a combination of overhunting, habitat destruction, and, possibly, disease. The species is now classified as vulnerable by the International Union for the Conservation of Nature, with a population probably below 7000, and it is also listed in Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) Appendix I.[77,78]

METHODS FOR CAPTURE OF FREE-RANGING PECCARIES

The capture of peccary herds in natural environments is often required so that studies of population ecology and management may be performed. The most common techniques are capture pens, box trapping, and ambush and chasing.

Capture Pens

First, the presence of tayassuids must be ascertained, evidenced by smell and marks on the ground from grubbing or footprints. Locations where signs are stronger and more recent are good spots to place baits. Salt licks are efficient and may be used in the beginning. A bag of mineralized salt with small holes in it, hanging from a tree branch about 2 m high should allow the salt to fall gradually to the ground. These attract several animal species, particularly white-lipped peccaries. Once attendance at the bait is confirmed, corncobs or seeds should be added. After some time, capture pens measuring 12 m long, 6.3 m wide, and 2.2 m high should be built with resistant wooden boards. Pervious attempts to build the upper part of the walls with wire fence resulted in injured animals, leading to severe lesions and even death. A covered food trough should be installed inside the pen and constantly refilled with corn. The animals enter the pen through a drop gate held open by a stick. A cord connects both the stick and the bait so that when an animal touches the lure the stick falls and the gate drops, confining the animals inside the pen. Proper positioning of the bait behind the hod allows a large group of peccaries to enter the pen. A field assistant, using a wooden shield for protection, encourages the animals to move into restraint boxes. The boxes are closed and taken to the small handling chute beside the pen. Using the chute, animals may be handled

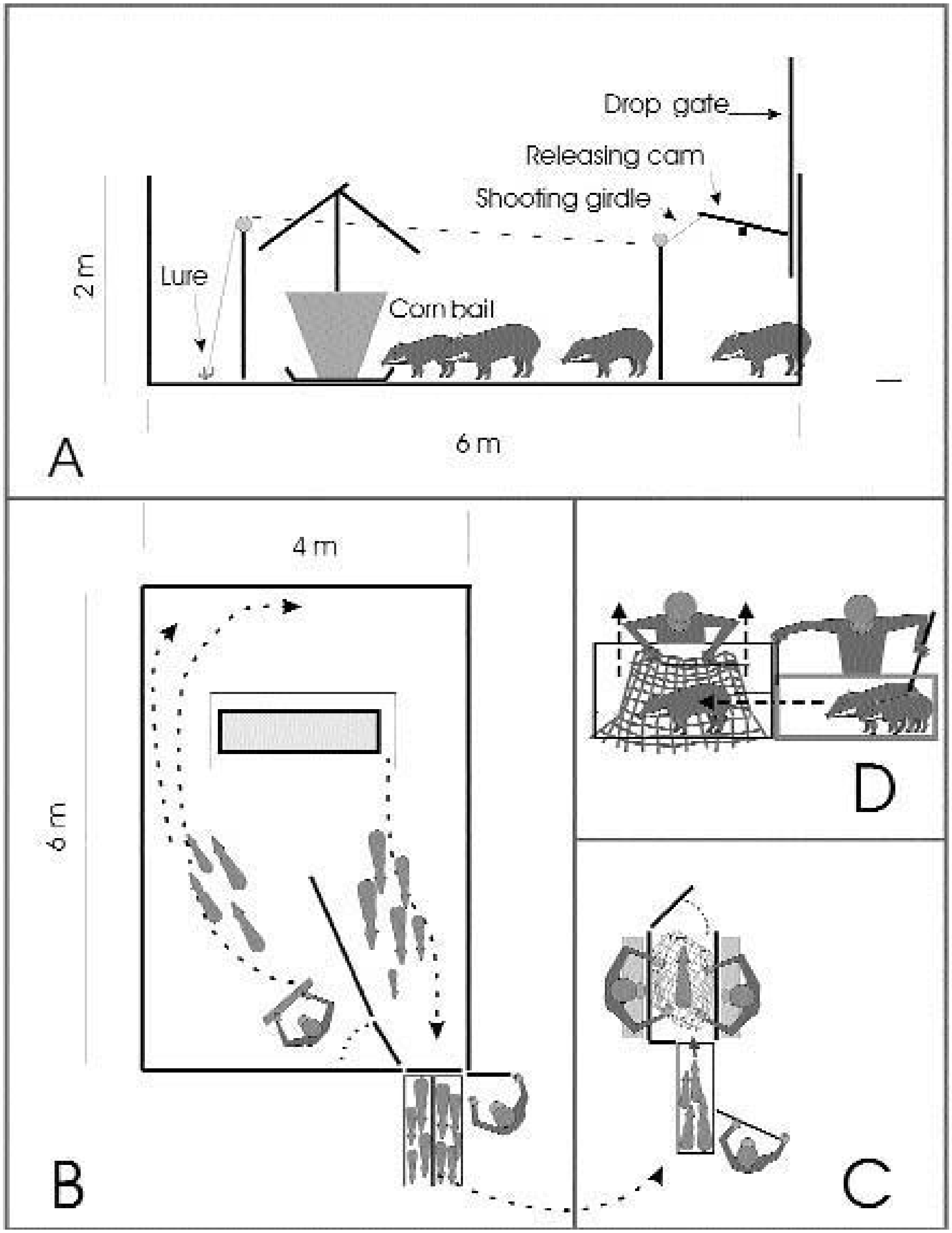


FIGURE 33.1. Wild or captive peccary management, schematic representation. **A.** Capture pen for white-lipped peccaries; lateral view showing disarming system. **B.** Management of wild peccaries inside capture pen, directing animals to transport box. **C.** Manipulation of wild or captive peccaries in handling chute, using net to restrain the animals. **D.** Peccaries inside handling chute, hemi-lateral view.

under field conditions as described in the physical restraint section of this chapter (Figure 33.1).

The authors found the method described here particularly efficient in capturing white-lipped peccaries in the seasonally humid semideciduous forest in southern Brazil. It allowed more than 240 manipulations in a group of white-lipped peccaries, without accidents or deaths. Some advantages are the number of animals captured at each turn, which ranged from 3 to 35, and the possibility of recapturing animals that returned to the area on multiple occasions. The easiness of manipulation of the animals using the chute allows such procedures as pregnancy diagnosis; ear tagging; and ectoparasite, fecal, and blood sampling to be carried out without chemical restraint.

The incidental capture of other species such as the tapir and mazama's deer may occur. On a few occasions, capuchin monkeys (*Cebus apela*) and coatimundi (*Nasua nasua*) disarmed the doors, preventing the capture of peccaries. Capture pens were also used to collect white-lipped peccaries by Karesh in Bolivia.[44] The authors built a large 55-yard pen with two drop gates activated by assistants behind observation blinds. The method allowed capture and chemical restraint of 40 individuals. However, the method is not satisfactory for the capture of collared peccaries.

Box Trapping

Box traps are commonly used for capturing white-lipped and collared peccaries. A box to capture collared peccaries should be 1 m long, 40 cm wide, and 60 cm high, built of wooden boards. A pedal located on the bottom of the trap may work as a trigger. The floor should be covered with straw. The bait (preferably corncobs) should be placed on the straw inside the box. Two or more boxes may be placed in the same area, occasionally yielding double or triple captures. Box traps proved more efficient for collared peccaries than capture pens.

Ambush and Chasing

This capture method resembles some commonly used hunting techniques. Trained dogs are used to chase and encircle peccary herds. When the animals locate in certain spots to avoid the dogs, they are darted with anesthetic solutions. Some variations without dogs may be performed. The methodology requires plenty of field experience from the researchers, field assistants, and trained dogs. It is possible to capture peccaries by waiting on a platform, up to 10 m away from a bait spot, and dart them with a special weapon, essentially a carbon dioxide or compressed air system. Darting trajectory errors, long waiting periods, depending on the activity of the herd, and the ability to manipulate only a few animals at each successful capture are disadvantages of both methods.

HANDLING AND MANAGEMENT OF CAPTIVE ANIMALS

Enclosures and Handling in Captivity

Throughout their geographical distribution, peccaries are raised in captivity. Most facilities are semi–free-ranging. The installation of enclosures in native vegetation–covered areas with running water facilitates obtaining supplemental feed sources for the diet. If captive conditions resemble natural habitats, stress is lessened. However, this type of enclosure hinders individual control, as well as requiring greater effort to capture the animals.

In a seasonal semideciduous forest, groups as large as 75 white-lipped and up to 50 collared peccaries may be maintained in approximately 1-ha enclosures (50 m × 196 m). Enclosures should be surrounded by wire mesh (12 mm^2) 1.20 m high.

In commercial facilities, smaller enclosures approximately 1000 m^2 may be used to raise piglets or for formation of new groups. About 30 young, 60–90 days of age, may be easily maintained in such areas. In the described conditions it is advisable to rotate enclosures every year, allowing previously used enclosures to remain empty for a year. Such measures may become costly, but preserves vegetation in the enclosure, contributing to the feeding of the animals.

To maximize such a breeding operation, each enclosure should contain an area for feeding that gives access to the handling chute. Having these areas of the enclosures in the handling area provides more opportunities to observe the animals. The gate to the entrance of the handling area may be equipped with a trap system, with armament linked to the food station. At the moment animals enter the handling area to feed, it is possible to close the gate minimizing the stress of the manipulation. This system can also be worked manually at a distance. The animals contained in the feeding area may be driven easily by keepers with protection shields. Also, portions of the captive group may be captured with help of three keepers, who move inside the enclosure and produce noises, driving the animals to a wall that leads to the handling area. Keepers should wear rigid shin protectors and helmets as safety equipment while manipulating peccaries, to avoid accidents inside the enclosures or aggression by animals that are being driven. Both systems, when operated by experienced and cautious people, were shown to be safe and accident free (Figure 33.2).

In Proyecto Tagua, Allen[1] reports that 45 *C. wagneri* were maintained in enclosures surrounded by fences of 1.35 m in height. The group was divided into three family groups of 20 individuals, each housed in enclosures of 0.5, 2, and 2 ha, respectively. The enclosures contained native vegetation composed of bushes, shade trees, and open grass areas, as well as artificial reservoirs of water. Two holding pens facilitated handling.

Proportion of Males and Females in Captivity

To form a captive herd, it is advisable to keep animals captured on the same occasion together in the same enclo-

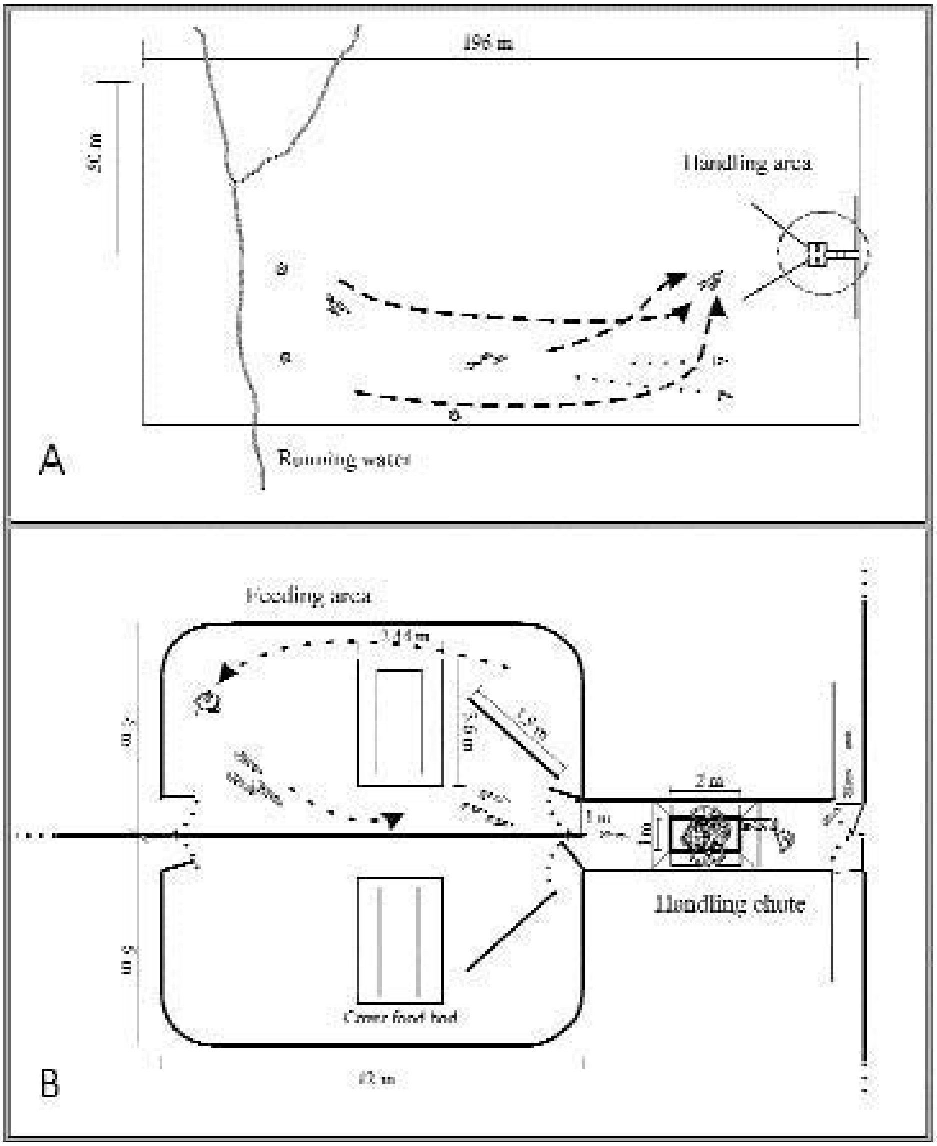


FIGURE 33.2. **A.** General distribution of enclosures and handling areas in commercial breeding of collared and white-lipped peccaries. **B.** Feeding area, handling chutes, and management of peccaries inside handling area.

sure. This minimizes aggression and facilitates acceptance in the group. The proportion of one male to four females in reproductive groups was shown to be satisfactory. The hierarchic dominant male has breeding priority and having equal numbers of males and females provokes an increase of aggressive interactions among the males. Studies in natural habitats shows sexual proportion of 1:1 for *P. tajacu* and for *T. pecari;* this proportion was also maintained in the analysis of fetuses.[34] In the state of Parana, Brazil, 160 individuals were captured and marked; of this total, 95 were female and 65 were male. Among the adults there was a proportion of 56 females for 28 males (2:1), whereas in the other age classes the proportion was 39 females to 37 males (close to 1:1) (T.C.C. Margarido, personal observation, 1997; unpublished data) The observations seem to indicate a tendency in the modification of this proportion in the adults, what was also observed by Fragoso[28].

Diet in Captivity

Usually diets used in captivity do not include most items that peccaries ingest in natural conditions. In zoos it is common to use corn, cassava, commercial rations for swine, fruits, roots, ensilages, ground sugarcane, and other sources of fibers. The high adaptability of the peccary's digestive system lessens the likelihood of a nutritional deficiency disease. Even so, the basic needs of the three species of Tayassuidae are still not completely defined.

TABLE 33.1. Weight of *Tayassu pecari* and *Pecari tajacu* according to sex

Species	Weight, Male (kg)	Variation (kg)	Weight, Female (kg)	Variation (kg)	Reference
Tayassu pecari	30.78	24–38.5	32.67	23.5–40	80
	39.30	34–45	38.40	36–42	6
	34		33		71
Pecari tajacu	19.20	14–23	18.78	16–22	80
	26.80	21–35	26.70	21–33	6
	19.90		20.70		71

TABLE 33.2. Birth weight of *Catagonus wagneri,* according to sex and age class

Species	Age Class*	Sex	Weight (kg)	Minor Mass (kg)	Major Mass (kg)	*n*	Reference
Catagonus wagneri	1	M	0.82	0.67	0.96	2	58
		F	0.70	—	—	1	
	2	M	16.2	12.5	18	4	
	3	M	23.6	23.5	24	3	
		F	23.8	23	24.5	3	
	4	M	34.8	29.5	40	18	
		F	34.6	30.5	38.5	10	
	3–4	F+	38.4	32	43.5	8	

*Determinates for dental eruption patterns.
F+, pregnant female; *n*, sampled animal number.

MORPHOLOGIC ADAPTATIONS

Peccaries possess a proportionally large head in relation to the extremities. Similar to the suids, the snout is elongated, with the nostrils in the rostral extremity. That structure is sustained by the union of the rostral bone with the nasal bones and by the nasal cartilage, forming a cartilaginous disk covered by a mucous membrane. The legs are slender, with two toes and two dew claws in the fore foot and two toes in the hind foot, each covered by small hoofs. The stomach of peccaries and swine differ. Peccaries have a stomach with four compartments, whereas suids are monogastric.[4] The gastric characteristic of Tayassuidae allows more efficient digestion of fiber, similar to ruminants.[61] In the peccaries, the stomach also produces volatile fat acids.[63] Peccaries have six to nine coccygeal vertebras, whereas suids have 20 to 23 coccygeal vertebrae. A gland is located approximately 20 cm ahead of the tail, which produces an oleaginous secretion of strong scent used for territory marking and also to promote cohesion of the members of the group.[63,75] The dental formula of the three species of tayassuids is I 2/3; C 2/2; PM 3/3, and M 3/3, in a total of 38 teeth.[76] Peccaries have large canine teeth, having little function in feeding, but valuable as defense mechanisms against predators.[21, 76] Each canine is sharpened in acute angles by abrasion against the opposite teeth.[24]

Tables 33.1 and 33.2 present bibliographical data on the weight of the three Tayassuidae species. Table 33.3 presents weights of *T. pecari* obtained in captivity and free ranging populations in south Brazil.

REPRODUCTIVE CHARACTERISTICS

The majority of available data about the reproduction of Tayassuidae was collected from studies of *P. tajacu* maintained in captivity or free ranging in southwestern North America. Studies of captive and wild peccaries indicate that they lack strong reproductive seasonality. In the wild, frequency of birth seems to be related to the amount of rainfall and food availability.[6,23,33,47,61,74] However, reproductive seasonality may be a possibility for wild populations of *T. pecari.*[80]

Anatomy

Most descriptions of the reproductive tract of Tayassuidae are restricted to a few studies based on *P. tajacu*[33,47,52,76] and *T. pecari.*[53,33] The anatomy of the reproductive tracts of female and male peccaries is similar to that of domestic suids.

In captivity, peccaries reach sexual maturity between 8 and 24 months.[26,53,61,74,76] For collared peccaries, the

TABLE 33.3. Weight of captive and wild *Tayassu pecari,* according to age class

Age Class	*n*	Corporal Mass (kg)	Minor Mass (kg)	Major Mass (kg)	Population
Young[a]	41	9.74 (±3.43)	3	15	Wild
Subadult[a]	58	20.15 (±2.79)	15.5	24.5	Wild
Adult[a]	147	33.87 (±5.76)	24.5	51	Wild
1[b]	10	1.59 (±0.47)	0.9	2.5	Captive
2[b]	24	4.89 (±2.25)	2	9.5	Captive
3[b]	51	10.45 (±3.04)	5	18	Captive
4[b]	47	17.54 (±4.15)	8.5	28	Captive
5[b]	34	22.98 (±5.30)	12	33.5	Captive
6[b]	9	37.27 (±5.32)	32	49	Captive

Source: Margarido, T.C.C, Personal observation, unpublished data, obtained in south of Brazil (52°54′ W; 25°26′ S).
[a]Preliminary determination according to body mass and phenotypic patterns.
[b]Determination by dental eruption patterns.
n, sampled animal number.

estrous cycle varies between 23 and 34 days, with a 4-day estrus. For white-lipped peccaries, the cycle varies between 25 and 32 days with a 6-day estrus. In both species silent heat occurs in more than 58% of the cycles.[56] The progesterone values, for nonpregnant female collared peccaries, varies between 1 and 27 ng/mL. In the white-lipped peccaries, this value varies between 1 and 25 ng/mL.[53,56] In this species, females in estrus have estradiol levels above 130 pg/mL and values below 60 pg/mL in other phases of the cycle.[53]

The gestation period for the collared peccary is 145 days,[74] and for white-lipped peccary it is approximately 159 days.[26] Females of both species can be impregnated during the nursing period, reducing the interval between parturitions.[62] The average concentration of progesterone between day 11 and 140 of gestation of the collared peccary is 36-48 ng/mL. Until the day 90 of gestation, the estradiol concentration varies between 5 and 10 pg/mL. After that period the concentrations of this hormone in the plasma reaches 49.07 pg/mL.[38] Pregnancy diagnosis is easily performed by ultrasonography. *T. pecari* have one to three young, and *P. tajacu* and *C. wagneri* have one to four young. For these three species the average litter size is two.[33,58,61,76] The nursing period may vary between 6 and 8 weeks.[74] Captive-born collared or white-lipped peccaries may be separated from the group within 2 months of age.

PHYSICAL RESTRAINT

Physical restraint may be used for simple procedures, such as ear-tagging, physical examination, medication, and administration of anesthetic agents. In peccaries, physical restraint is usually accomplished with the aid of nets and snares. Tayassuids do not tolerate restraint with a snout snare. More satisfactory methods are permanent chutes or narrow runway systems that restrict forward and backward movement. In Brazil, collared peccaries are directed into a small enclosure, then a nylon mesh net hoop is placed over the exit, capturing peccaries as they leave the enclosure.[24] Young and baby *Catagonus wagneire* may be restrained manually; adult animals may be restrained in squeeze chutes or by push boards.[1]

These animals may be manipulated in a handling chute 1.5 m long, 1.0 m wide, and 1.2 m high. It should possess two doors and a superior bar of wood, where a suspended scale can be positioned. A net of resistant nylon, made with thread 6 mm in diameter and an 80–100 mm mesh, may be placed on the floor of the chute for mechanical restraint.[3] Peccaries captured in a restraint box may be positioned in front of one of the chute doors. When the box's door is opened, the animal enters the chute, positioned over the net. Two keepers, one on each side of the chute, can suspend the peccary in the net. To determine the body weight, the net is hung on the scale (Figures 33.3 and 33.4). Other necessary procedures may be accomplished by pressing the captive animal against the side of the handling chute. After the manipulation, the net is returned to the floor, and the animal is released from the chute by the opposite door.

Restraint and manipulation of peccaries in a squeeze chute are recommended for such simple procedures as blood sampling, verification of sex, pregnancy examination, rectal and vaginal swabs, ear tagging, physical examinations, parasitic prophylaxis, or injection of anesthetic drugs. The collection of blood may be made, preferably from the saphenous or cephalic veins. When suspended in the net the distal portion of the limbs can be pulled out to expose the veins. The compression of the veins by the mesh may facilitate venipuncture.

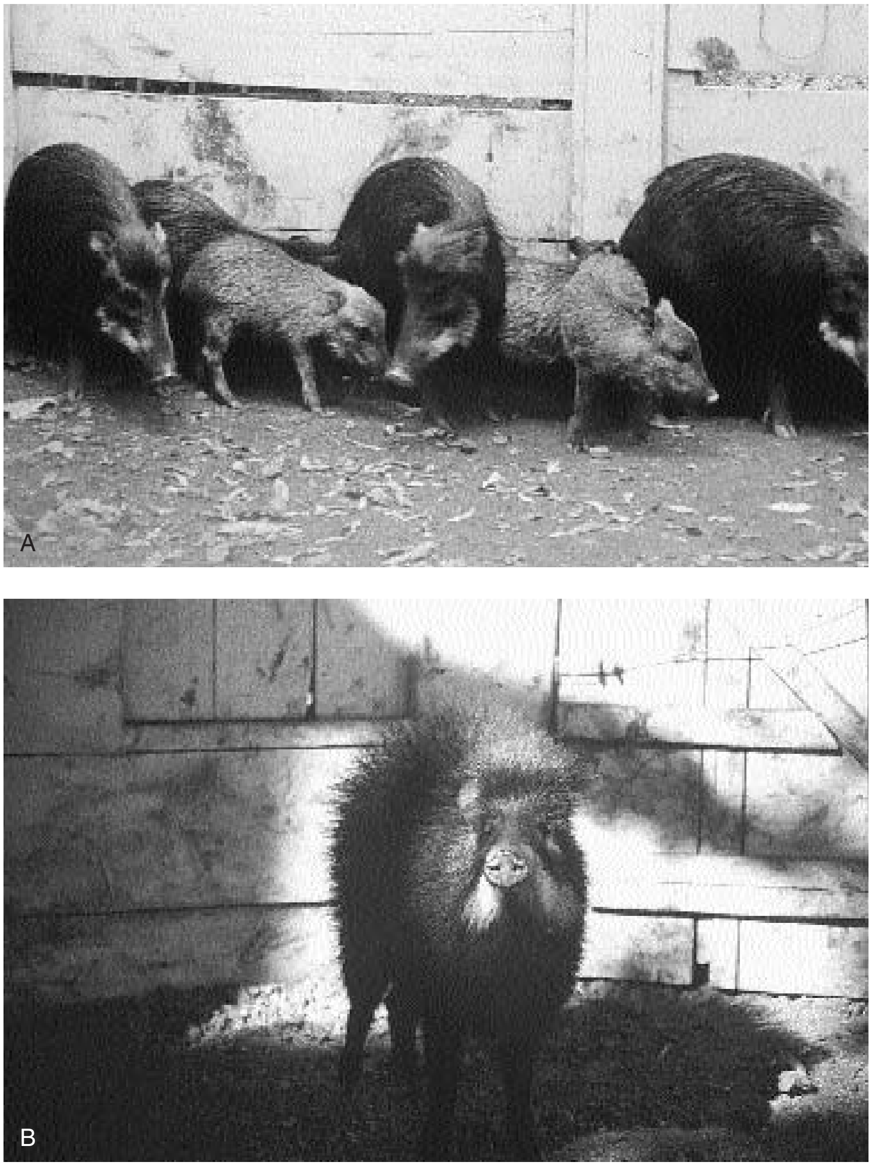


FIGURE 33.3. A and B. White-lipped peccaries in holding pen waiting for manipulation on handling chute.

CHEMICAL RESTRAINT AND ANESTHESIA

Chemical immobilization or anesthesia is necessary for such procedures as suturing wounds or surgeries for repair of fractures. The anesthetic agents frequently used in wild or domestic ungulates can be used with success in peccaries. Fowler[24] and Calle and Morris[12] describe anesthesia in tayassuids.

The combination of tiletamine hydrochloride and zolazepam hydrochloride has been satisfactory for such procedures as biometry, collecting blood samples, biopsies, ear tagging, and small surgeries.[2,12,43, 44] The doses used in peccaries vary from 1.5–5.0 mg/kg. The higher dosage will produce a prolonged recovery period. Halothane and isoflurane inhalation anesthetic agents are excellent for prolonged surgery, but they must be administered through a precision vaporizer. Induction may be made with an injectable drug such as ketamine hydrochloride or tiletamine/zolazepam. Tracheal intubation is difficult because of the narrow jaws and inability to open the mouth widely,[24] but a nasal mask is satisfactory for maintaining anesthesia. Previous diseases, difficult to detect in field situations, may complicate anesthesia. Death during anesthetic procedures has been reported, caused by hyperthermia when environmental temperatures were high.

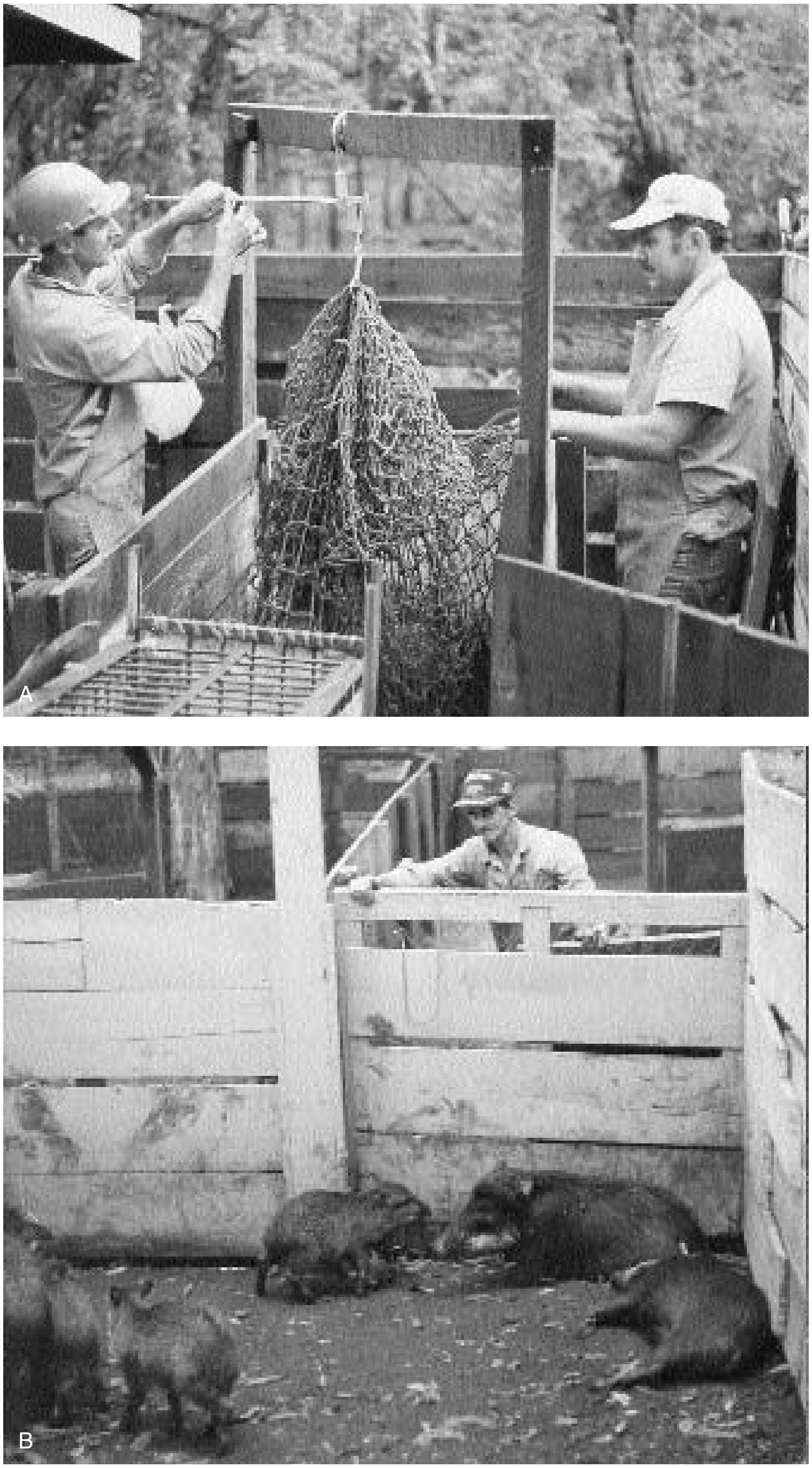


FIGURE 33.4. **A.** Body mass measurement of peccary in the handling chute, using net for physical restraint. **B.** White-lipped peccaries in the holding pen and keeper preparing to release them back to the enclosure.

TABLE 33.4. Infectious diseases evidences, excluding macroparasitic diseases, in free-ranging peccaries (*T. pecari*–white-lipped peccary and *P. tajacu*–collared peccary) in the Southwestern U.S. and Central and South America

Disease	Species	Location	Evidence	Reference
Canine distemper	cp	U.S.A.: Arizona	ta/em/sa	5
Venezuelan equine encephalitis	cp	U.S.A.: Texas	sa	73
Western equine encephalitis	cp	U.S.A.: Texas	sa	73
Pseudorabies	wlp	Brazil: Parana	sa	54
	wlp	Bolivia	sa	44
	cp	U.S.A.: Arizona	sa	16
Rabies	cp	Arizona	if	42
Vesicular exanthema of swine	wlp	Bolivia	sa	44
San Miguel sea lion virus	wlp	Bolivia	sa	44
Vesicular stomatitis	wlp	Bolivia	sa	44
	cp	U.S.A.: Arizona	sa	16
Brucellosis	wlp	Brazil: Parana	sa	54
	cp	Venezuela	sa/ai	51
Leptospirosis	wlp	Bolivia	sa	44
	cp	U.S.A.: Arizona	sa	16
Mycoplasma hyorhinus	wlp	Bolivia	sa	44
Streptococcus suis	wlp	Bolivia	sa	44
Yersinia pestis	cp	U.S.A.: Texas	—	36
Toxoplasma sp.	cp	Panama	sa	29
Borellia burgdorferi	cp	U.S.A.: Texas	—	36

cp, collared peccary; wlp, white-lipped peccary; ta, tissue antibody; em, electronic microscopy; sa, serum antibody; if, direct immunofluorescent antibody; ai, agent isolation.

DISEASES

Infectious Diseases

Infections diseases are presented in Table 33.4.

Bacterial and Fungal Diseases

Bacterial and fungal infections in peccaries primarily cause enteric and respiratory diseases. Coccidioidomycosis, candidiasis, and cryptococcosis in captive peccaries have been reported.[20,41, 48] Serologic evidence of *Mycoplasma hyorhinus* and *Streptococcus suis* were observed in wild white-lipped peccaries in Bolivia.[44] Diarrheas in captive *P. tajacu* can be caused by *Salmonella muenchen* and *Escherichia coli*.[76] In *T. pecari,* diarrheas have been caused by *Klebsiella* sp., *Salmonella* sp,. and *Shigella flexneri*. Purulent arthritis may result from *Proteus vulgaris* infection. Subcutaneous abscesses and edema in white-lipped and collared peccaries have been associated with *E. coli* or *Alcaligenes* sp.[55]

Hemorrhagic or nodular caseous pneumonia has been seen in rejected newborn and young individuals, possibly the result of stress. Severe diarrheas are also common causes of mortality among rejected animals. In many of these cases, necropsy has failed to determine the etiologic agents.

Serologic evidence of the presence of pathogenic agents have been found in wild and captive peccaries. Research to determine presence of antibodies against *Brucella* spp. has been negative in wild populations of white-lipped and collared peccaries,[16,44] but in southern Brazil, 6.5% of captive and wild *T. pecari* showed positive serologic results for *Brucella abortus*.[5] In Venezuela 87% of *P. tajacu* had positive serums for *Brucella* sp. The authors isolated *B. suis* from 43 spleen and lymph node samples.[51] In one breeding facility in southern Brazil (CASIB-Itaipu Binational) such problems as metritis, cervicitis, and abortions accounted for 1.2% of the clinical problems observed in *T. pecari*.[55] Clinical signs of brucellosis are not frequently observed in tayassuids, however, problems of fertility, common in captivity, may be due to the presence of that etiologic agent in the herd. In certain areas, the dissemination of *Brucella* sp. among wild populations seems to be one of the factors of slow growth or instability of peccary populations. The presence of antibodies against *Leptospira interrogans* has also been investigated in some wild populations of Tayassuidae. Positive results were observed in 65% of the white-lipped and 23% of the collared peccaries evaluated.[16,44] Few signs of disease were observed in the population.[44]

Viral Diseases

Free-ranging tayassuids are susceptible to many of diseases common to domestic ungulates. There is evidence of the occurrence of vesicular stomatitis, San Miguel sea lion virus, vesicular exanthema of swine, pseudorabies, rabies, western and Venezuelan equine encephalitis, and canine distemper.[5,16,42,44,54, 3] Neurological signs such as lethargy, depression, ataxia, and head and limb tremors are related to infection by morbillivirus. Serological studies suggest that canine distemper virus infection may be common in free-ranging peccaries in the southern United States.[5] Experimentally, *P. tajacu* was susceptible to foot-and-mouth disease, rinderpest, and hog cholera, but was resistant to the African swine fever. In wild populations of *T. pecari,* no evidence was found of the occurrence of these diseases or bluetongue, porcine parvovirus, swine influenza virus, and transmissible gastroenteritis.[44,54] The susceptibility of peccaries to those viruses is doubtful; even so, the possibility should not be discarded. Samples from an outbreak of nonsevere diarrhea, of yellowish coloration, in a captive breeding herd of *T. pecari* (CIZBAS-USP-Brazil), were analyzed by electron microscopy. Large numbers of particles compatible with coronavirus were found.[14] The results are still preliminary and indicate the susceptibility of the species to transmissible gastroenteritis. In that herd, the infection was probably transmitted from domestic suids. Contact between peccaries and domestic swine should be restricted in commercial farms, scientific breeding herds, and zoo collections. Outbreaks of contagious and endemic diseases in the area should be monitored. Outbreaks of disease caused by viruses or other infectious diseases have been suspected in wild populations of the three species of Tayassuidae.[27,76,77]

Parasitic Diseases

Many authors have described parasites in wild or captive peccaries.[10,55,60,76] The interrelationships of parasites and hosts in Tayassuidae are not completely established. The great majority of reports on parasitism in this group refer to sporadic observations. Evaluations of the influence of parasites on population dynamics are rare.[17]

Several species of internal parasites have been ascribed to Tayassuidae.[76] Lung lesions associated with nematodes have been observed in wild *T. pecari.* Infestations of *Capillaria* spp. may result in a moderate and chronic active eosinophilic hepatitis. Acanthocephalic parasitism may be negative in a fecal examination; however, fibrous and granulomatosis nodules may be found in the intestinal wall of the infested animals.[44] Animals affected by liver and lung lesions are less resistant to environmental stress or manipulation.

Large numbers of ectoparasites have frequently been observed in wild peccaries. Tick infestation is associated with multifocal dermatitis, but in general, such infestations are not associated with more severe signs of organic weakness. The tick most frequently found on all three species of Tayassuidae is *Amblyoma* spp.[32,76] Sarcoptic mange may cause disease in wild or captive collared peccaries.[24,59] In captivity, infestations by ticks, fleas, and lice frequently cause health problems. Ivermectin in a dose of 0.02–0.04 mg/kg, carbamate powder, or dusting with Fipronil may give good results. Hematic parasites such as *Eperythrozoon* sp. and *Trypanosoma pecarii* were observed in collared peccaries.[37,67] Antibodies against *Toxoplasma* spp. were observed in one of two wild *P. tajacu* in Panama.[29] Wild collared peccaries may have a high prevalence (71%) of infestation with *Ballantidium coli.*[10] Cysts and trophozoites of *Ballantidium* sp. were also observed in captive peccaries.[55] Generally considered a commensal organism, it may cause episodes of diarrhea in captive animals. A diet high in carbohydrates may stimulate increased growth of protozoan populations. Ballantidiosis is a zoonosis; the close contact between captive peccaries and people may facilitate the infection. Six percent of Amazonian indigenous people who keep *P. tajacu* as pets and as a source of animal protein, had infections with *B. coli.* This fact indicates the possible existence of a wild cycle of disease involving indigenous humans and collared peccaries.[10]

Miscellaneous Conditions

In captivity, cutaneous lesions from intraspecific aggression comprise about 20% of the medical problems. Injuries of the musculoskeletal system, e.g., limb and canine teeth fractures and lameness, represent 12% of the cases. Other medical problems of captive peccaries are parasitic lesions, bacterial diarrhea, rectum prolapses, arthritis, puerperal paralysis, pneumonia, hydrothorax, miocarditis, abortion, metritis, or cervicitis.[55]

Multiple lesions may be observed in free-ranging peccaries. Chronic active eosinophilic interstitial pneumonia, bronchitis, tracheitis, pleuritis, degenerating nematodes in the lungs, moderate and chronic active eosinophilic hepatitis associated with *Capillaria* sp., fibrous and granulomatous nodules associated with acanthocephalid attachment in the intestinal wall, severe diffuse lymphoplasmatic epicarditis and multifocal lymphoplasmatic myocarditis, eosinophilic inflammation in the tunica adventita of aorta, splenic capsulitis and interstitial nephritis were observed in free-living white-lipped peccaries.[44] Ingestion of foreign bodies may represent one of the causes of death among collared peccaries in the wild, mainly when in poor habitat. Death of a subadult individual was caused by gastric obstruction, resulting from ingestion of a plastic bag (P.R. Mangini, unpublished data). Congenital alterations such as cyclopia and limb deformity have been described in collared peccaries.[40] In southern Brazil, a case of albinism was observed in a young wild collared peccary.[81] In captivity most deaths of *P. tajacu* and *T. pecari* are of 1-week-old individuals, in general, resulting from adult predation or following maternal rejection. Environmental conditions of high population density, little social stability in family groups, or poor health conditions of the offspring are related to rejections. Even if given intensive care, most rejected newborns die from hemorrhagic lesions in the lungs and heart. Bacterial diarrheas caused by *Klebsiella* spp., may also been responsible for death of rejected young.[55]

PREVENTIVE MEDICINE

No specific prophylactic procedures have been developed for peccaries. The prophylactic protocol most used in captive peccaries is parasite control, which should be repeated regularly. However, risk situations to animal health are sometimes ignored. Special attention should be paid to the prevention of diseases when stressful activities are undertaken, such as introducing new individuals into an established herd, dividing populations into new groupings, transport, introducing animals into new enclosures, and reintroduction of animals to the wild.

The possibility and risk of zoonotic transmission and infection by diseases common to domestic animals should be constantly evaluated. For most diseases there are no available killed vaccines. In addition, vaccine protocols and immunologic responses have not been tested in peccaries. In high-risk situations vaccination may be necessary.

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REFERENCES

1. Allen, J.L. 1992. Veterinary work with the chacoan peccary (*Catagonus wagneri*) in Paraguay. In Proceedings of the Joint Meeting of the AAZV and AAWV. pp. 72–74.
2. Allen, J.L. 1992. Immobilization of giant chacocoan peccaries (*Catagonus wagneri*) with a tiletamine hydrochloride/zolazepam hydrochloride combination. Journal of Wildlife Diseases 28(3):499–501.
3. Almeida, A.F.; Bertolani, F.; and Nicolielo, N. 1979. Estudo de uma população de catetos, *Tayassu tajacu,* em uma floresta implantada de *Pinus* spp. [Study of collared peccaries population in *Pinus* spp. plantation forest]. Publicaço Semestral do instituto de Pesquisas e Estudos Florestais-Universidade de São Paulo (19):21–35.
4. Anderson, S.; and Jones, J.K., Jr. 1984. Orders and Families of Recent Mammals of the World. New York, Wiley Interscience, p. 686.
5. Appel, M.J.; Reggiardo, C.; Summers, B.A;. Pearce-Kelling, S.; Máre, C.J.; Noon, T.H.; Reed, R.E.; Shively, J.M.; and Orvell, C. 1991. Canine distemper virus infection and encephalitis in javelinas (collared peccaries). Archives of Virology 119(1):147–152.
6. Bodmer, R.E.; Aquino, R.; Puertas, P.; Reyes, C.; Fang, T.; and Gottedenker, N.L. 1996. Evaluando el uso sostenible de pecaries en el Nor-Oriente del Peru: Evaluacion poblacional y manejo de *Tayassu pecari* y *Tayassu tajacu* en la Amazonía Peruana. Iquitos, Peru, University of Florida, Tropical Conservation & Development Program, p. 121.
7. Bodmer, R.E.; and Sowls, L.K. 1996. *Tayassu tajacu*. In W.L.R Oliver, ed., Plan de Acción y Evaluación de la Condición Actual de los Peccaríes. Quito, Ecuador, IUCN/CSE, pp. 5–15
8. Bodmer, R.E. 1991. Influence of digestive morphology on resource partitioning in Amazonian ungulates. Ecologia 85(1):361–365.
9. Boever, W.J. 1986. Restraint, handling, and anesthesia. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 942–952.
10. Brites-Neto, J.; and Thatcher, V.E. 1986. Estudos parasitologicos preliminares em tayassuídeos (*Tayassu tajacu*) na Amazônia Central [Preliminar study of parasitosis of *Tayassu tajacu* in Central Amazonia]. Revista Brasileira de Medicina Veterinária 8(6):175–184.
11. Cabrera, A.; and Yepes, J. 1960. Mamíferos Sudamericanos: Vida, Costumbres y Descripción. Buenos Aires, Companhia Argentina de Editores, p. 370.
12. Calle, P.P.; and Morris, P.J. 1998. Anesthesia for nondomestic suids. In M.E. Fowler and R.E. Miller, eds., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 639–646.
13. Castellanos, H.G. 1983. Aspectos de la organizacion social del baquiro de collar *Tayassu tajcu* L. en el Estado Guarico-Venezuela. Acta Biologica Venezuelana 11(4):127–143.
14. Catroxo, M.H.B.; Miranda, M.L.; Andrade, P.C.M.; Cappelaro, C.E.M.P.D.M.; and Lavorenti, A. 1996. Detecção ao microscópio eletrônico de transmisso de partículas semelhantes a coronavírus em fezes de queixada (*Tayassu pecari*). In Proceedings of the 15° Congresso Panamericano de Ciência Veterinárias. Campo Grande, Sociedade Brasileira de Medicina Veterinária p. 73.
15. Colbert, E.F.; and Morales, M. 1991. Evolution of the Vertebrates: A History of the Back-boned Animals through Time, 4th Ed. New York: Willey-Liss.

16. Corn, J.L.; Lee, R.M.; Erickson, G.A.; and Murphy, C.D. 1987. Serologic survey for evidence of exposure to vesicular stomatitis virus, pseudorabies virus, brucellosis and leptospirosis, in collared peccaries from Arizona. Journal of Wildlife Diseases 23(1):551–557.
17. Corn, J.L; Pence, D.B.; and Warren, R.J. 1985. Factors affecting the helminth community structure of adult collared peccaries in Southern Texas. Journal of Wildlife Diseases 21(3):254–263.
18. Coscarelli, K.P. 1985. Ovulation in the collared peccary as documented by laparoscopy. Masters thesis, Texas A&M University, p. 53.
19. Crespo, J.A. 1982. Ecologia de la comunidad de mamíferos del Parque Nacional Iguazu, Misiones. Ecologia 3(2):45–162.
20. Culjak, K. 1973. Candidose beim sudamerikanischen Halsbandpekari (*Tayassu tajacu*) [Candida infection in a South American peccary (*Tayassu tajacu*)]. In Proceedings of the 15° Internationalen Symposiums, Erkrankungen der Zootiere. pp. 305–306.
21. Eisenberg, J.F. 1989. Mammals of the Neotropics, Vol. 2. The Southern Neotropics. Chicago, University of Chicago Press, pp. 229–234.
22. Emmons, L.H. 1990. Neotropical Rainforest Mammals: A Field Guide. Chicago, University of Chicago Press, p. 281.
23. Esbérard, C.E.L.; Honório, A.S.; and Bezzera, A.M. 1995. Sazonalidade Reprodutiva de Mamíferos Silvestres na Fundação Riozoo. Rio de Janeiro, Fundação RIOZOO.
24. Fowler, M.E. 1993. Wild swine and peccaries. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, Vol. 3. Current Therapy. Philadelphia, W.B. Saunders, pp. 513–522.
25. Fowler, M.E.; and Boever, W.J. 1986. Superfamily suidoidae. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 964–967.
26. Frädrich, H. 1995. Breeding the white-lipped peccary (*Tayassu pecari*) at Berlin Zoological Garden. International Zoo Yearbook 34:217–221.
27. Fragoso, J.M.V. 1997. Desapariciones locales del baquiro labiado (*Tayassu pecari*) en la Amazonia: migracion, sobre-cosecha, o epidemia? In T.G. Fang, R.E. Bodmer, R. Aquino, and M.H Valqui, eds., Manejo de Fauna Silvestre en la Amazonia. La Paz, OFAVIM, pp. 309–312.
28. Fragoso, J.M.V. 1994. Large Mammals and the Community Dynamics of an Amazonian Rain Forest. Doctoral thesis, University of Florida, p. 210.
29. Frenkel J.K.; and Sousa, O.E. 1983. Antibodies to toxoplasma in Panamanian mammals. Journal of Parasitology. 69(1):244–245.
30. Gabor, T.M.; Hellgren, E.C.; and Silvy, N.J. 1997. Immobilization of collared peccaries (*Tayassu tajacu*) and feral hogs (*Sus scrofa*) with telazol and xylazine. Journal of Wildlife Diseases 33(1):161–164.
31. Gallager, J.F.; Lochmiller, M.S.; and Grant, W.E. 1985. Immobilization of collared peccaries with ketamine hydrochloride. Journal of Wildlife Management 49(2):356–357.
32. Gazêta, G.S.; Amorim, M.; Teixeira, R.H.F.; Teixeira, P.S.; and Serra-Freire, N.M. 1999. *Tayassu pecari* (Artiodactila: Tayassuidae) novo hospedeio para *Amblyoma cooperi* Nutatall & Warburton, 1908 (Acari:Ixiodidae). In Proceedings of the 23° Congresso Brasileiro de Zoológicos na Sociedade de Zoológicos do Brasil. Goiânia, Brasil, Sociedade de Zoológicos do Brasil, p. 24.
33. Gottdenker, N.L. 1996. Reproductive Ecology and Harvest Evaluation of Peccaries in the Northeastern Peruvian Amazon. Masters thesis, University of Florida, p. 106.
34. Gottdenker, N.L.; and Bodmer, R.E. 1998. Reproduction and productivity of white-lipped and collared peccaries in the Peruvian Amazon. Journal of Zoology [London] 245(1):423–430.
35. Grubb, P.; and Groves, C.P. 1993. The neotropical tayassuids *Tayassu* and *Catagonus*. In W.L.R. Oliver, ed., Status Survey and Conservation Action Plan: Pigs, Peccaries and Hippos. Gland, Switzerland, International Union for Conservation of Nature and Natural Resources, pp. 5–7.
36. Gruver, K.S.; and Guthrie, J.W. 1996. Parasites and selected diseases of collared peccaries (*Tayasu tajacu*) in the Trans-Pecos region of Texas. Journal of Wildlife Disease 32(3):560–562.
37. Hannon, P.G.; Lochmiller, R.L.; Mellen, J.W.; Craig, T.M.; and Grant, W.E. 1985 Eperythrozoon in captive juvenile collared peccaries in Texas. Journal of Wildlife Diseases 21(4):439–440.
38. Hellgren, E.C.; Lochmiller, R.L.; Amoss, J.R.; and Grant, W.E. 1985. Serum progesterone, estradiol-17B, and glucocorticoids in the collared peccary during gestation and lactation as influenced by dietary protein and energy. General and Comparative Endocrinology 59(1):358–368.
39. Hellgren, E.C.; Lochmiller, R.L.; Amoss, J.R.; and Grant, W.E. 1985. Endocrine and metabolic responses of the collared peccary (*Tayassu tajacu*) to immobilization with ketamine hydrochloride. Journal of Wildlife Diseases 21(4):417–425.
40. Hellgren, E.C.; Lochmiller, R.L.; Thomas, M.W.; and Grant, W.E. 1984. Cyclopia, congenital limb deformity, and osteomyelitis in the collared peccary. Journal of Wildlife Diseases 20(1):354–357.
41. Hendrickson, R.V. 1972, Cryptococcosis in man and captive exotic animals, Journal of Zoo Animal Medicine 3(1):27.
42. Hetrick, M.; Goodman, H.; and Wright, M. 1986. Rabies in a javelina, Arizona. Morbidity and Mortality Weekly Report 35(35):555–556.
43. Karesh, W.B.; and Stetter, M.D. 1997. Pers. commun. Cited in Calle, P.P.; and Morris, P.J. 1998. Anesthesia for nondomestic suids. In M.E. Fowler and R.E. Miller, eds., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 639–646.
44. Karesh, W.B.; Uhart, M.M.; Paiter, R.L.E.; Wallace, R.B.; Braselton, W.E.; Thomas, L.A.; House, C.; McNamara, T.S.; and Gottdenker, N.L. 1998. Health evaluation of white-lipped peccary populations in Bolivia. In

Proceedings of the Joint Conference of the AAZV and AAWV. pp. 445–449.
45. Kiltie, R.A. 1981. Stomach contents of rain forest peccaries (*Tayassu tajacu* and *T. pecari*). Biotropica 13(3):234–236.
46. Kiltie, R.A. 1982. Bite force as a basis for niche differentiation between rain forest peccaries (*Tayassu tajacu* and *Tayassu pecari*). Biotropica 14(3):188–195.
47. Lochmiller, R.L.; and Hellgren, E.C. 1992. Reproduction in collared peccaries. In W.C Hamlett, ed., Reproductive Biology of South American Vertebrates. New York, Springer-Verlag, pp. 313–322.
48. Lochmiller, R.L.; Hellgren, E.C.; Hannon, P.G.; Grant, R.E.; and Robinson, R.M. 1985. Coccidioidomycosis in the collared peccary in Texas. Journal of Wildlife Diseases 21(1):305–309.
49. Lochmiller, R.L.; Hellgren, E.C.; and Grant, W.E. 1987. Influence of moderate nutritional stress during gestation on reproduction of collared peccaries (*Tayassu tajacu*). Journal of Zoology [London] 221(1):321–328.
50. Lochmiller, R.L.; Hellgren, E.C.; Vaner, L.W.; Grrene, L.W.; Amoss, M.S.; Seager, S.W.J.; and Grant, W.E. 1985. Physiological responses of the adult male collared peccary, *Tayassu tajacu* (Tayassuidae) to severe dietary restriction. Comparative Biochemistry Physiology 82(1):49–58.
51. Lord, V.R.; and Lord, R.D. 1991. *Brucella suis* infection in collared peccaries in Venezuela. Journal of Wildlife Diseases 27(1):477–481.
52. MacDonald, A.A. 1985. Notes em placentation in the suina. Placenta 6(1):83–91.
53. Mangini, P.R. 1999. Estudo dos Níveis Séricos de Progesterona e Estradiol e da Estrutura do Trato Genital Feminino de Queixada (*Tayassu pecari* Link, 1795). Masters thesis, Universidade Federal do Paraná, p. 76.
54. Mangini, P.R.; Gasino-Joineau, M.E.; Carvalho-Patricio, M.A.; Fortes, M.; Gonçalves, M.; Margarido, T.C.C.; and Klemz, C. 1998. Avaliação da incidência de doenças infecto contagiosas em populações selvagens e cativas de *Tayassu pecari,* na região de quedas do iguaçu-Paraná. In Proceedings of the 7° Congresso Associação Brasileira de Veterinários de Animais Selvagens. Foz do Iguaçu, Brasil, Associação Brasileira de Veterinários de Animais Selvagens, pp. 24–25.
55. Mangini, P.R; Moraes, W.; and Santos, L.C. 1999. Enfermidades observadas em *Tayassu pecari* (queixada) e *Tayassu tajacu* (cateto) mantidos em cativeiro em Foz do Iguaçu, Paraná [Diseases in captivity *Tayassu pecari* e *Tayassu tajacu,* in Foz do Iguaçu, Paraná, Brazil]. Arquivo de Ciências Veterinárias e Zoologia da Unipar – Universidade Paranaense (in press).
56. Mauget, R.; Feer, F.; Henry, O.; and Dubost, G. 1997. Hormonal and behavioral monitoring of ovarian cycles in peccaries. In Proceedings of the 1st International Symposium on Physiology and Ethology of Wild and Zoo Animals, Suppl. 2. Berlin, Alemanha, pp. 145–149.
57. Mayer, J.J.; and Wetzel, R.M. 1987. *Tayassu pecari.* Mammalian Species 293(1):1–7.
58. Mayer, J.J.; and Brandt, P.N. 1982. Identity, distribution, and natural history of the peccaries, Tayassuidae. In M.A. Mares and H.H. Genoways, eds., Mammalian Biology in South America. Special Publication Pymatuning Laboratory of Ecology 6(1):85–93.
59. Meierhenry, E.F.; and Clausen, L.W. 1977. Sarcoptic mange in collared peccaries. Journal of the American Veterinary Medical Association 171(9):983–984.
60. Nascimento, A.A.; Bonuti, M.R.; Mapeli, E.B.; Arantes, M.D.G.; Tebaldi, J.H.; and Arantes, I.G. 1996. Helmintos parasitos de suínos (*Sus scrofa domesticus*), catetos (*Tayassu tajacu*) e veado catingueiro (*Mazama gouazoubira*) provenientes do Pantanal do Mato Grosso do Sul, Brasil. In Proceedings of the 15° Congresso Panamericanao de Ciência Veterinárias. Campo Grande, Brasil, Sociedade Brasileira de Medicina Veterinária, p. 79.
61. Nogueira-Filho, S.L.G.; and Lavorenti, A. 1997. O manejo do caitetu (*Tayassu tajacu*) e do queixada (*Tayassu pecari*) em cativeiro. In C. Valladares-Padua and R.E. Bodmer, eds., Manejo e Conservação de Vida Silvestre no Brasil. Belém, Sociedade Civil Mamirauá, pp. 106–115.
62. Nogueira-Filho, S.L.G.; and Lavorenti, A. 1995. Criação do caititu e do queixada em cativeiro [Captive breeding of collared and white-lipped peccary]. Ciência Hoje 9(114):6–9.
63. Novak, R.M. 1991. Walker's Mammals of the World, Vol. 2, 5th Ed. Baltimore, Johns Hopkins University Press, p. 1629.
64. Oliver, W.L.R. 1993. Status Survey and Conservation Action Plan: Pigs, Peccaries and Hippos. Gland, Switzerland, International Union for Conservation of Nature and Natural Resources, p. 202.
65. Olmos, F. 1993. Diet of sympatric Brazilian caatinga peccaries (*Tayassu tajacu* and *T. pecari*). Journal of Tropical Ecology 9(1):255–258.
66. Peres, C.A. 1996. Population status of white-lipped *Tayassu pecari* and collared peccaries *T. tajacu* in hunted and unhunted Amazonian forests. Biological Conservation 77(1):115–123.
67. Pessoa, S.N.; and Biasi, P. 1972. Sobre uma nova especie de tripanosoma, *Trypanosoma pecarii* sp. n., parasita do porco-do-mato: *Tayassu tajacu* (Linneu). Actas da Sociedade Biologia do Rio de Janeiro 15(2):109–111.
68. Redford, K.H.; Eisenberg, J.F. 1992. Mammals of the Neotropics. Chicago, University of Chicago, p. 430.
69. Robinson, J.G., and Redford, K.H. 1991. Neotropical Wildlife Use and Conservation. Chicago, University of Chicago Press, p. 520.
70. Rusconi, C. 1930. Las especies fosiles argentinas de Pecaríes ("TAYASSUIDAE") y sus relaciones con las del Brasil y Norte América. Anales del Museo Nacional de Historia Natural Bernardino Rivadavia 36(1):121–241.
71. Schaller, G.B. 1983. Mammals and their biomass on a Brazilian ranch. Arquivos de Zoologia, São Paulo 31(1):1–36.
72. Sicuro, F.L. 1996. Inferências Acerca da Coexistência de Taiassuídeos e Suídeos Ferais (Mammalia, artiodactyla), no Pantanal da Nhecolandia (MS): Um Modelo Ecomorfologico. Rio de Janeiro, Universidade Federal do Rio de Janeiro, p. 162.

73. Smart, D.L.; Trainer, D.O.; and Yuill, T.M. 1975. Serologic evidence of Venezuelan equine encephalitis in some wild and domestic populations of southern Texas. Journal of Wildlife Diseases 11(1):195–200.
74. Sowls, L.K. 1966. Reproduction in the collared peccary (*Tayassu tajacu*). In I.W. Rowlands, ed., Comparative Biology of Reproduction in Mammals. London, Zoological Society of London, pp. 155–172.
75. Sowls, L.K. 1984. The Peccaries. Tucson, Arizona, University of Arizona Press.
76. Sowls, L.K. 1997. Javelinas and Other Peccaries: Their Biology, Management and Use, 2nd Ed. Texas A & M University Press.
77. Taber, A.B. 1991. The status and conservation of the Chacoan peccary in Paraguay. Oryx 25(3):147–155.
78. Taber, A.B.; Doncaster, C.P.; Neris, N.N.; and Colman, F.H. 1993. Ranging behavior and population dynamics of the Chacoan peccary, *Catagonus wagneri*. Journal of Mammalogy 74(2):443–454.
79. Taber, A.B.; and Oliver, W.R.: 1993. Review of priorities for conservation action and future research on neotropical peccaries. In W.L. Oliver, ed., Status Survey and Conservation Action Plan: Pigs, Pecaris, and Hippos. Gland, Switzerland, Unio Mundial para Conservação de Natureza, pp. 37–40.
80. Townsend, W.R. 1996. Nyao Itõ: Caza y pesca de los Sirionó. Instituto de Ecologia, Universidade Mayor de San Andrés, Santa Cruz de La Sierra, Bolívia, pp. 79–130.
81. Veiga, L.A. 1994. Um caso de albinismo em *Tayassu tajacu* Linnaeus (Arctiodactila, Tayassuidae) na Serra do Mar, São José dos Pinais, Paraná [A case of albinism in *Tayassu tajacu* in Paraná, Brazil]. Revista Brasileira de Zoologia 11(2):341–343.
82. Wetzel, R.M. 1977. Chacoan peccary, *Catagonus wagneri* (Ruscuni). Bulletin of the Carnegie Museum of Natural History 3(1):36.
83. Grubb, P. 1992. Order Artiodactyla. In D.E. Wilson and D.M. Reeder, eds., Mammal Species of the World: A Taxonomic and Geographical Reference, 2nd Ed. Smithsonian Institution Press, Washington, D.C., p. 1206.

34 Order Artiodactyla, Family Camelidae (Guanacos, Vicuñas)

BIOLOGY AND MEDICINE

P. Walter Bravo, and Murray E. Fowler

BIOLOGY

South American camelids (lamoids) evolved in North America, then migrated south when the Caribbean land bridge was formed, approximately 3 million years ago.[4,6] The karyotype of all camelids is 2n = 74. All South American camelids have produced fertile hybrids. Dromedary and Bactrian camels also produce fertile crosses. A cross between a camel male and a guanaco female by artificial insemination produced a living full-term offspring in the United Arab Emirates. The male offspring is only 1 year of age, thus it is not known whether or not he is fertile. All South American camelids have similar behavioral patterns.

Taxonomy

The classification of South American camelids has been controversial, but the system used in this chapter is order Artiodactyla, suborder Tylopoda, family Camelidae, genus *Lama* (guanaco, llama, alpaca), and genus *Vicugna* (vicuña) (Table 34.1).[5]

Distribution

The guanaco has the broadest distribution, both historically and currently, of the four South American camelids (Table 34.1). Four geographic subspecies of guanaco have been described, ranging from sea level in Tierra del Fuego at the southernmost tip of South America to 4600 m in the Andes. The northernmost populations exist at latitude 8° south in Peru. Guanacos live in both migratory and sedentary groups. All guanaco subspecies share uniform coloration (Table 34.1).

Vicuña distribution is limited to the puna (Quechua for highland) life zone of the Andes (elevation 4200–4800 m). The vicuña is the smallest of the South American camelids and has the finest fiber coat (Table 34.1). There are two geographic subspecies of vicuña: the Peruvian, with long white hairs on the bib, and the Argentine, with shorter hair on the bib. The vicuña was considered the property of the Inca kings, and only royalty were allowed to wear garments made from the fiber.[5]

Anatomy and Physiology

Despite size differences, the anatomy of all species of camelids is similar. Camelids have a complex, three-compartmented stomach, with glandular mucosa in all

TABLE 34.1. South American camelids—biological data

Scientific Name	Common Name (English)	Common Name (Spanish)	Common Name (Portuguese)	Identification	Distribution	Weight (kg)
Lama guanacoe	Guanaco	Guanaco	Guanaco	Basic body color is light to dark reddish brown above, whitish hair below. White extends up behind the fore legs and in the front of the rear leg, around the perineum, inside of legs, and up the bottom of the neck. Head, face, and ears are dark gray to black	Peru, Bolivia south to Tierra del Fuego	100–120
Vicugna vicugna	Vicuna	Vicuña	Vicuna	Color is similar to that of guanaco, but the basic body color is a yellowish light brown. The white in front of the rear limbs may extend to the top of the back. White bib on the chest. No sexual dimorphism in color or size.	High altiplano of southern Peru, Bolivia, Chile, and Argentina	38–42; height at withers 85–90 cm, at rump 90–94 cm
Lama glama	Llama—domestic animal	Llama	Lhama	Numerous solid colors from white to black. Multicolors (pinto, appaloosa) also seen.	Throughout Peru, Bolivia, northern Chile and Argentina	113–250
Lama pacos	Alpaca—domestic animal	Alpaca	Alpaca	22 solid colors recognized, ranging from white to black. Multicolored fleece also produced.	Similar to llama	55–90

compartments. Gastric digestion is similar to, but not analogous with, ruminant digestion. The two suborders separated from each other 30–40 million years ago when primordial species were simple stomached. Both groups used fibrous forage and developed similar fore gut fermentation systems by parallel evolution. Camelids regurgitate and rechew ingested forage, as do ruminants, but are more efficient than ruminants in extracting protein and energy from poor-quality forages.

The camelid placenta is epitheliochorial. The most unique characteristic of the camelid fetal membranes is the development of an extra membrane of fetal epidermal origin. This membrane was described in the dromedary camel 15 years ago, but was not described in South American camelids until 1988.[5]

The epidermal membrane (EM) is an opaque whitish membrane, approximately 1–2 mm thick, covering the surface of the fetal body, head, neck, and limbs at near full term. The EM is attached to the neonate at the mucocutaneous junctions such as at the lips, anus, vulva, and prepuce. The EM is also attached at the junction of the skin and foot-slipper, coronet of the nail, and at the umbilicus.

The EM is friable and easily torn or brushed from the surface of the neonate with only slight friction. Even with little movement or abrasion, it dries out and withers away soon after parturition. Histologically, the EM consists of a layer of stratified squamous epithelial cells lying next to the fetus and an outer keratinized layer with indistinct cell outlines and no nuclei. The fetus is usually delivered with the EM intact. However, if parturition is prolonged, with erratic or intense labor, or if manual assistance is necessary, the EM will split and disintegrate. The precise function of the EM is unknown.[5]

Field Data

Vicuña social structure consists of family groups of two to five females and their crias, which are dominated by a single breeding male. Both male and female offspring are driven from the family group by the dominant male (males at 4–9 months of age, females at 10–12 months). Females rarely conceive before they are 2 years old, but a few become pregnant at 12–14 months.[5]

Males driven from the family group form temporary associations with other males (bachelor herds of 2–100 animals), and do not attempt to establish their own territories until 4 years of age. Juvenile females that are driven from the family group may attempt to enter another family group, but they may be repulsed by the male so as to maintain the optimum size of the group. Unattached females will ultimately become part of a new male's family group.

The social organization of guanacos is similar to that of the vicuña. However, guanacos have a broader distribution in more diverse habitats, from sea level to 3000 m. Guanacos are more flexible than vicuñas, some populations being sedentary, whereas others are migratory. Migratory behavior necessitates some modifications of family group and male group patterns. In addition to these, aggregates of females may form during the winter, whereas each breeding male remains alone in the family group territory. There may even be mixed groups of males and females that come together during the winter.

Diets and Foraging Behavior

All the South American camelids have incisor teeth that are firmly fixed in the mandible, similar to the dentition of sheep and goats, but in contrast to the loose attachment of bovine teeth. These teeth, pressing against the upper dental pad, shear vegetation. These animals are able to graze short plants and cut them close to the ground, but their general behavior is to move about a feeding area, picking a mouthful here and there, not mowing any one spot clean, unless they are tethered.[4]

The lips of camelids are unique. The upper lip is split by a labial cleft. Each side of the lip can be manipulated independently to investigate a potential food item and draw it to the teeth.[5]

The high Andes habitat of the vicuña is a harsh environment of semiarid grassland, and barren pampas, with cold temperatures and sparse vegetation. The vicuña is a grazer of forbs and grasses. The guanaco is both a grazer and a browser, inhabiting desert grassland, savanna, and shrub land; it may even be seen in forests. It is found in one of the driest deserts of the world, the Atacama in Chile, and also in the wet archipelago of Tierra del Fuego, where rain falls year-round.[5]

Reproductive Strategies in the Wild

Vicuñas are nonmigratory. The family groups are highly territorial. The dominant male defends a family feeding territory against intrusion by strangers, either male or female. The borders of the territory are strictly delineated. The family group also maintains a sleeping territory, to which the family retreats in the evening. Corridors between sleeping and feeding territories may be neutral territory for several family groups.

Vicuñas are seasonal breeders in Peru, with 90% of all births occurring between mid-February and early April. Though females will be bred within 2 weeks of parturition, only 60-80% of the females will give birth the following year. In the Andes, over 90% of vicuña births occur in the morning hours. This is probably an evolutionary adaptation to the weather patterns of that area. The frequent afternoon storms could interfere with adequate drying of the cria before the nightly drop to near-freezing temperatures occurs.[5]

Guanaco reproduction takes place only within the family group. Juvenile males and females are evicted from the family group, as in the vicuña strategy, but the age differs at which dispersal occurs. In guanacos, juvenile males and females are weaned at approximately 7 months but will begin nursing again when the next cria is born, continuing until evicted at 13–15 months of age. Thus both juveniles and the crias of the year are present in a family group for a few months. It is thought that enhanced social maturity is fostered by this biologic strategy. Guanaco males do not become territorial until they are 4–6 years of age. This does not mean that male guanacos are physically incapable of breeding earlier than this, but social and behavioral restrictions prevent earlier breeding.[5]

Conservation

The following estimates of guanaco populations were made in 1992: Argentina (550,000), Chile (20,000), and Peru (1,400). There are fewer than 350 guanacos in zoos in the United States, Europe, and Australia. The vicuña population in South America was down to 500 individuals prior to an embargo being placed on exportation of either the animal or its fiber by the countries to which they are native. The numbers have climbed steadily, and now the following estimates are made: Argentina (23,000), Bolivia (12,000), Chile (28,000), and Peru (98,000). Fewer than 200 vicuñas are exhibited in zoos. Vicuñas are listed in Convention on International trade in Endangered Species of Wild Fauna and Flora (CITES) Appendix II .

MANAGEMENT IN CAPTIVITY

Housing

South American camelids require minimal housing. They should be provided with protection from inclement weather, particularly harsh winds in extremely cold climates; however, the animals are often seen standing or recumbent in rain or snowstorms, even though shelter is available.

Guanacos and vicuñas are social animals and generally tolerate close association with others of their own kind. Adult breeding males should not be kept in the same enclosure, unless the manager is prepared for the fighting that may ensue.

Environment

For optimal ambient temperatures, see discussion on heat stress.

Feeding

South American camelids may be maintained on a good-quality grass hay or mixed grass and legume hay. Supplemental feeding with concentrates is not necessary except for growing juveniles and lactating females.

Reproduction in Captivity[8,9,10]

FEMALE The female camelid is an induced ovulator. There is no estrous period; females are generally receptive to a male unless they have been recently bred or are pregnant. The follicular cycle of guanacos and vicuñas has not been studied in detail, but is assumed to be similar to that of llamas and alpacas. Follicles grow and regress in an average time of 10–12 days, with ovaries alternating in 85% of the cases for presence of mature follicles. Studies of ovarian follicle dynamics using laparoscopy and ultrasonography, correlated with hormone concentrations, revealed that follicles grow, mature, and regress in an overlapping wavelike pattern (follicular wave).

Following is a succinct discussion of the anatomy of the reproductive organs and the main reproductive events of the female vicuna gained from endocrine work done in captured animals and maintained year-round in a natural environment, but in fenced pastures.[6,8-10]

The reproductive organs are located within the pelvic cavity with the ovaries at the level of the first sacral vertebrae. They are irregular in shape as a result of the presence of follicles of different diameters. The ovaries are $1.2 \times 1.0 \times 0.7$ cm. The oviducts are convoluted and are 11 cm in length and 0.3 cm in diameter. The left uterine horn is longer (6.1 cm) than the right uterine horn (5.3 cm). The body of the uterus is about 2.3 cm in length, and the cervix has 3 annular rings. The vagina is 8.1 cm in length and 1.9 cm in diameter, with longitudinal rugae.

Open female vicuñas maintained with no male contact show only the follicular phase of the ovarian cycle. Receptivity and fecundity of vicuñas is not correlated with the size of the follicle, and estradiol-17 levels are always low.[6]

Females ovulate after being bred to vasectomized or intact males. When bred to a vasectomized male, the luteal phase of the ovarian cycle is manifested by elevated progesterone concentrations. At 5 days after mating, the progesterone level is 4 nmol/L, (1.2 ng/mL), reaching a peak at day 8 of 12 nmol/L (3.8 ng/mL). Levels regress by day 9 or 10 to 0.6 ng/mL. Progesterone remains elevated throughout pregnancy 10 nmol/L, (3.2 ng/mL) in females bred to intact males. Progesterone concentrations fall to basal values immediately before parturition and some females were bred even before parturition.[4]

Sexual behavior of the female may be expressed by either acceptance or refusal of the male. A receptive female will usually adopt sternal recumbency, but sometimes the male will chase and try to mate the female. If the female is receptive, she will become recumbent after a short time, not more than 2–3 minutes. Refusal of the male is indicated by running away and kicking and spitting at the male. Refusal indicates that the female is under the influence of progesterone or pregnant.

The male is taken to the enclosure of the female for copulation. If the female refuses him, they should be separated for a week before repeating. Copulation lasts 20–25 minutes and should not be repeated because the penis of the male penetrates into the uterine horns and traumatizes the uterine mucosa by mechanical rubbing. Following copulation the female should rest for a week and then be exposed to the male again to ascertain ovulation. A refusal of the female is a sign that ovulation has occurred. If the female is receptive, she should be bred again. A refusal at 21 or 30 days after breeding is a sign of pregnancy which may be confirmed using ultrasonography. The presence of the embryonic vesicle within the uterine horn is the ultimate diagnosis of pregnancy. If an ultrasound is not available, the combination of sexual behavior (refusal of the male) and elevated progesterone levels are good indicators of pregnancy.

The occurrence of dystocia is rare in SAC. Records from thousands of births in the natural setting of llamas and alpacas indicate that only 2% of births may be considered as dystocia. The signs of dystocia and obstetrical management are the same as in other livestock species.

Assessment of fertility is rarely done in wild guanacos and vicuñas. Assessing fertility is complicated, especially in guanacos, due to dispersal of males after the breeding season. The number of crias in a family group may represent an accurate evaluation of fertility, especially of the male. Sixty to eighty percent of the females should produce crias in a given year.

MALE Fertility evaluation of captive animals is done as in llamas and alpacas. The male vicuna has similar reproductive anatomy and physiology to llamas and alpacas, but has different sexual behavior. Changes in testicular size by age is given in Table 34.2.[10]

Testosterone concentrations are seasonal. During the breeding months testosterone is 6.6 nmol/L (19.1 ng/mL) and during August, a month of sexual quiescence, 4.5 nmol/L (12.9 ng/mL). In addition, testicular size is also influenced by season. The length of the testicle is greater in summer (3.3 cm) than in winter (2.6 cm).[10]

TABLE 34.2. Testicle size of male vicuñas in Peru

Age	Left Testicle	Right Testicle
12 months	1.1 × 0.7	1.0 × 0.6
18 months	1.5 × 0.8	1.4 × 0.8
24 months	2.1 × 1.3	2.1 × 1.2
36 months	2.6 × 1.5	2.6 × 1.4
Adults	3.2 × 2.2	3.3 × 2.1

Length × transverse axis.

Semen may be collected from the female vagina following breeding. Normally a small volume of semen remains in the vicinity of the cervix after breeding. A clean and dry vaginal speculum is used to view the cervix. Semen can then be retrieved, aspirating it with a syringe attached to an insemination rod. The retrieved liquid, if semen, is viscous and at microscopic examination reveals abundant red blood cells (normal) and clusters of spermatozoa entrapped in the seminal plasma. Normal heads of spermatozoa are opaque in contrast to colored heads, which are an indication of dead spermatozoa. Semen characteristics are assumed to be similar between vicuñas, guanacos, and their domesticated counterparts. No reports exist on the seminal characteristics of vicuñas; however, the author (P.W. Bravo) has been able to recover semen from the ductus deferens after castration and slaughter. In both cases, 0.1–0.3 mL of semen was retrieved using phosphate saline as rinse solution. Semen collected was opaque and live spermatozoa showed oscillatory movement of the flagellum, but there was no progressive motility.

Neonatology

Neonate camelids must consume colostrum within the first 24 hours of postnatal life or there will be a failure of passive transfer of maternal immunoglobulins. Without such protection, the offspring is most likely to die of neonatal septicemia. Considerable experience has been gained in hand-rearing orphaned llamas and alpacas. The same principles apply to guanacos and vicuña.

Llamas and alpacas have numerous congenital/hereditary defects, but none have been reported in guanacos and vicuñas.

RESTRAINT, ANESTHESIA, SURGERY

Restraint

Guanacos may be tamed if reared in association with humans and handled during maturation. Such animals may be handled in a manner similar to llamas or alpacas, but it should be recognized that guanacos may be more easily frightened or excited. Free-ranging newborn guanacos may be hand captured within the first few hours of birth, but thereafter they can run as fast as their dams. Vicunas also may be reared in association with humans. Sometimes, during capture of vicunas, nursing neonates are caught, orphaned, and fed cow's milk. These animals are imprinted on humans and used on especial occasions such as parades.

Vigorous manual restraint is needed to control wild guanacos or vicuñas. Two or more persons may be needed to catch an animal within a restricted space such as a small stall. Most of these animals will not tolerate being tied with a halter and will fight if placed in one of the chutes designed for their domestic cousins.

Restraint of vicuñas initiates an elevation of cortisol levels. Basal concentrations of this hormone in tamed vicunas is 23.5 and 17.5 ng/mL in male and female juveniles, respectively. Restraint causes an increase of cortisol to 49.7 and 68.5 ng/mL in adult and juvenile males, respectively. Shearing produces a similar cortisol response; 56.5 and 72.1 ng/mL in adult and juvenile males, respectively, and 36.5 and 60.9 ng/mL in adult and juvenile females, respectively. Thus, it appears that juvenile vicunas are more stress prone than the adults. In addition, the presence of generalized muscular tremors and opisthotonus may be observed in free-ranging vicuñas the day after being restricted to a fenced enclosure. Extreme care should be taken in handling vicunas, as the neck may be traumatized. One of the authors (P.W. Bravo) diagnosed a fractured cervical vertebra in a female vicuna as a result of excessive force during handling .

A less traumatic and stressful method of restraint is intramuscular chemical sedation via a blow gun or stick pole syringe. Neither the guanaco nor the vicuña has a heavy fleece to obstruct insertion of the needle into the muscle mass of the upper rear limb or triceps. Numerous drugs and drug combinations have been successfully used on camelids (Table 34.3).[4]

Anesthesia

Optimum general anesthesia uses isoflurane inhalation and endotracheal intubation. South American camelids may also be anesthetized with halothane. Anesthesia induction may be accomplished by intravenous administration of 10% guaifenesin (120–180 mL), followed by a bolus of ketamine at 3.5 mg/kg body weight.[5]

The immobilizing agents may be used for injectable anesthesia. Any supplemental doses required should be administered intravenously to avoid prolonged recovery. Elective surgery and anesthesia should be preceded by 24–36 hours of fasting and water withheld for 12 hours to minimize regurgitation.[5]

Surgery

Surgery for castration, hernia repair, fracture repair, and laceration suturing is performed as in livestock and horses. The closure of a ventral midline laparotomy incision requires special mention. The anatomical associations on the midline are different than in other species. Closure should consist of a continuous suture to close the peritoneum, then a series of tension sutures should be placed in the muscle, being careful to include the internal and external aponeuroses (cruciate [a figure 8] or vertical mattress suture), then skin sutures.[5]

DIAGNOSIS

Clinical Examination

Vital signs for both free-ranging and captive vicuñas are: respiratory rate 30–34 breaths per minute, heart rate 78–100 beats per minute, and body temperature 38.5–39.7°C (101–103.5°F).[2] Guanacos are probably similar. Vicuña and guanaco hematology and blood chemistry values are similar to those of llamas and alpacas.

DISEASES

Nutritional Diseases

In North America, soil selenium deficiency results in diminished intake of selenium and hence a selenium deficiency. Nutritional myopathy of both skeletal and cardiac muscles causes stiff joints, lameness, paresis, and/or paralysis. Other signs include abortion and sudden death.

A syndrome first described as hypophosphatemic rickets appeared in llamas and alpacas in the Pacific Northwest of the United States. It was associated with winter-born crias that were subjected to cloudy conditions, obscuring the sun for days to weeks. Ultimately it was found that the underlying cause was a lack of vitamin D. The signs and radiographic lesions are those of classical rickets seen in many other species of mammals and birds.[5]

Infectious Diseases

South American camelids have only one unique infectious disease, actinomycosis (caused by a recently described species, *Actinomyces lamae),* which causes abscesses in soft tissue and osteitis in bone. There are no

TABLE 34.3. Sedatives, immobilizing agents, and anesthetics agents used in camelids

Generic Name	Commercial Name	Dosage (mg/kg body weight)	Reversal Agent (Commercial Name) [dose, mg/kg body weight]	Comments
Xylazine HCl	Rompun	0.1–0.4, IM, IV	Yohimbine (Anatagonil) [0.125] Tolazoline (Tolazine) [0.4–1]	Low dose IV, Higher dose IM
Ketamine HCl	Vetalar, Ketalar	2–5, IM, IV	None	Rarely used alone
Butorphanol tartrate	Torbugesic	0.05–0.1, IM	Naloxone (Narcan) Naltrexone (Trexan)	Good sedative, usually used in combination for immobilization
Xylazine/ketamine	Rompun/Vetalar	X. 0.25–0.5 K. 2–5	Yohimbine, Tolazoline, for xylazine only	
Detomidine/ketamine	Dormosedan/Vetalar	D. 0.02–0.04 K. 2–3	Yohimbine Tolazoline	
Meditomidine/ketamine	Domitor/Vetalar	M. 0.03–0.08 K. 2–3	Atipamesole (antisedan) [4.1–5.0 times M. dose]	Excellent combination
Diazepam/ketamine	Valium/Vetalar	Di. 0.1–0.3 K. 3–8	Flumazenil (Maxicon) [0.03–0.04]	
Tiletamine/zolazepam	Telazol (Zoletil)	4.7–6.0, IM		Dosage based on combined drugs. Long recovery, especially at higher dosages

IM, intramuscularly; IV, intravenously; X, xylazine; K, ketamine; D, detomidine; Di, diazepam.

common viral diseases. The following bacterial diseases may be regionally prevalent: enterotoxemia (*C. perfringens,* type A, type C, and type D.), colibacillosis (*Escherichia coli*), and actinomycosis. Common fungal infections include dermatophytosis (ringworm, *Trichophyton verrucosum, Microsporum* spp.) and coccidioidomycosis (*Coccidioides immitus*).

Uncommon bacterial diseases include botulism, tetanus, malignant edema (*Clostridium septicum*), brucellosis (*Brucella melitensis*), necrobacillosis (*Fusobacterium necrophorum*), corynebacteriosis (*Corynebacterium pseudotuberculosis*), staphylococcosis, eperythrozoonosis (*Eperthyrozoon* spp.) and the anaerobe *Bacteroides fragilis*. Cryptococcosis has been reported in a vicuña. Opportunistic Gram negative organisms such as *Pseudomonas* spp. and *Klebsiella* spp. may also cause infection. Uncommon viral diseases include rabies, vesicular stomatitis, contagious ecthyma, equine rhinopneumonitis, and Borna disease.

Diseases that are rarely reported and to which the SAC have a high level of resistance include foot and mouth disease, bovine virus diarrhea, Johne's disease (*Mycobacterium paratuberculosis),* and tuberculosis (*Mycobacterium bovis* and *Mycobacterium* spp.).[4]

No clinical disease has been reported, but South American camelids may develop a serologic response to the viral agents causing the following diseases: blue tongue, infectious bovine rhinotracheitis, influenza B, rotavirus, and parainfluenza III. Camel pox, which is a scourge of camels, has been produced experimentally in guanacos. Brucellosis caused by *Brucella abortus* is not a clinical disease of SAC, but the disease has been produced experimentally and serologic responses are reported.[5]

Parasitic Diseases

All free-ranging vicuñas are infested with parasites of one or another species. Ectoparasites include the mange mite *Sarcoptes scabiei* var. Auchenia and the tick *Amblyomma parvitarsum*. Parasites in the third compartment of the stomach are *Trichostrongylus axei, Ostertagia circumcinta, Ostertagia ostertagi, Ostertagia lyrata,* and *Haemonchus contortus*. Parasites found in the small intestine include *Nematodirus spathiger, Nematodirus fillicollis, Nematodirus battus, Lamanema chavezi, Cooperia oncophora, Cooperia mcmasteri, Trichostrongylus colubriformis, Capillaria* spp., *Moniezia expansa, Moniezia benedeni, Eimeria macusaniensis, Eimeria alpacae,* and *Eimeria punoensis*. Parasites found in the large intestine include *Trichuris globulosa, Trichuris ovis, Oesophagostomum venulosum,* and *Chabertia ovina*. A common parasite in the muscles is *Sarcocystis*. Selected parasitic diseases are listed in Table 34.4. More details may be found in the references provided.[1,5]

SAC may share numerous parasites with cattle, sheep, and goats, but it is not known definitively whether the species are the same or if there are strain differences. In addition to the previously mentioned parasites, the following species may be regionally important in captive South American camelids: *Damalinia* spp. and *Microthoracis* spp. (lice), and *Cephenemyia* spp. (deer nasal bots). Uncommon parasites include *Giardia* spp., *Toxoplasma gondii, Sarcocytis* spp., *Dictyocaulus* spp., *Thelazia californiensis, Bunostomum* spp., and *Echinococcus granulosus.*[1,3]

Three species of gastrointestinal nematodes deserve special mention. They are found only in South America and may represent a threat when animals are exported to other countries. They include *Graphinema auchenia, Lamanema chavezi,* and *Spiculopteragia peruvianus.*[3]

Noninfectious Diseases

South American camelids are cool-weather tolerant. In free-ranging populations they live in areas for which they are adapted. In captivity, guanacos and vicuñas should be protected from extremely high and low ambient temperatures. Hypothermia is a problem for neonates when parturition takes place during winter months in northern climates with subzero temperatures and chilling winds.

Hyperthermia is a constant concern in climates characterized by prolonged daytime ambient temperatures greater than 29°C (85°F) and 50% relative humidity. Guanacos have been subjected to elevated ambient temperatures followed by restricted water intake. They are able to withstand elevated body temperatures and significant dehydration, but not nearly as effectively as dromedary camels. Llamas in hot, humid climates in the United States may routinely have an afternoon body temperature of 40°C (104°F), normal is 37.5–38.6°C (99–101.5°F). Under such circumstances males will become temporarily sterile because of hyperthermic effects on spermatogenesis.

Hyperthermia affects every organ system, but produces pronounced damage to the reproductive, digestive, and cardiovascular systems. Clinical signs include an elevated body temperature (41.1–42.2°C [106–108°F]), accelerated heart rate (>90 beats per minute), dyspnea, sweating, convulsions, teratogenesis in pregnant females, scrotal edema in males, hypotensive shock, and disseminated intravascular clotting.

Management requires rapid cooling with a cold water enema. Hyperthermia is prevented by providing shade and water in which the animal may immerse itself.

TABLE 34.4. Selected parasitic diseases of South American camelids

Disease (English)	Disease (Spanish)	Disease (Portuguese)	Etiology	Location in or on Body	Clinical Signs	Diagnosis	Management
Deer nasal bots	Larva de la mariz del venado	Berne do nariz de ovinos	*Cephenemyia* spp.	Nasopharynx	Sneezing, coughing, noisy breathing, exercise intolerance	Radiography, endoscopy	Double dose of ivermectin
Spinose ear tick	Garrapata del oido	Carrapato de orelha	*Otobius megnini*	External ear canal	Head shaking, scratching, exudation from the ear	Observe the ticks in the ear canal	Acaricide, removal of ticks
Sacrcoptic mange	Sarna	Sarna	*Sarcoptes scabiei*	Skin	Crusty lesions on skin, scratching	Deep skin scraping	Ivomectin
Coccidiosis	Coccidiosis	Coccidiose	*Eimeria* spp.	Small intestine	Catarrhal to hemorrhagic diarrhea	Fecal flotation	Coccidiostats
Strongylosis	Estrongilosis	Veminose	*Nematodirus battus*	Small intestine	Diarrhea	Fecal flotation, ova are large	Anthelmintics
Meningeal worm	Gusano	Parelafostrongilose	*Parelaphostrongylus tenuis*	Spinal cord, brain	Incoordination, paresis, paralysis	Signs, history of exposure to white-tailed deer. Eosinophils in cerebrospinal fluid	Anthelmintics monthly during the snail season
			Lamanema chavezi	Small intestine, liver, lung	Hepatic and respiratory failure	Fecal flotation	Anthelmintics
Whipworms		Tricuríase	*Trichuris* spp.	Colon, cecum	Weight loss, diarrhea	Fecal flotation	Anthelmintics, high doses

Preventive Medicine

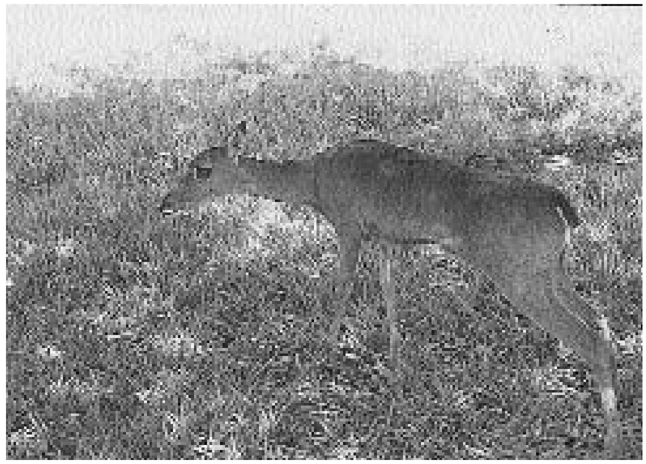
In the free-ranging state, herd size may be managed to avoid overpopulation and social stress. In captivity, vaccination protocols are similar to those of goats and cattle. In many facilities in North America, South American camelids are protected against tetanus and enterotoxemia, types C and D. Killed rabies vaccine is administered in rabies endemic areas and killed *Leptospira* spp. bacterins are given if problems exist in the livestock species in the area.[4]

Routine anthelmintic administration is carried out when fecal monitoring indicates a need. Captive animals should be adequately fed, but not overfed. In the free-ranging state, South American camelids undergo an annual cyclic feast and famine associated with the wet and dry seasons. Often, zoo managers fail to account for the famine part of the cycle. Obesity may be a significant problem.

Critical to any preventive medicine program is the quarantine of newly arrived animals for at least 30 days, during which time the animals are monitored for appropriate diseases and anthelmintic treatment is administered.

REFERENCES

1. Beltran, C.D. 1991. Evaluacion Parasitaria en Vicuñas del Centro de Conservacion de Fauna Silvestre de Umayo, Puno [Parasitism evaluation in vicuña at the Wildlife Conservation Center at Umayo, Puno]. Masters thesis. Universidad Nacional Altiplano, Puno.
2. Bombilla, R.T. 1996. Estudio Preliminar de Constantes Clinicas en la Especie *Vicugna vicugna* en Cala Cala, Azangaro, Puno [Preliminary study of clinical constants in vicuñas]. Masters thesis. Universidad Nacional Altiplano, Puno.
3. Choque, J.M. 1981. Estudio Parasitologico en Vicuñas (Vicugna vicugna) del Proyecto Pampa Galeras de Ayacucho, Peru [Parasitological study in vicuñas]. Masters thesis. Universidad Nacional Altiplano, Puno.
4. Flores, J. 1997. Efecto del Stress en la Captura y Esquila sobre los Niveles de Cortisol Sanguineo y Formula Leucocitaria Relativa en Vicunas (Vicugna vicugna) [The effect of capture and shearing on blood cortisol levels and leukocytes in the of vicuña]. Masters thesis. Universidad Nacional Altiplano. Puno.
5. Fowler, M.E. 1998. Medicine and Surgery of South American Camelids, 2nd Ed. Ames, Iowa, Iowa State University Press.
6. Gallegos, R.C. 1997. Descripcion Macroscopica del Aparato Reproductor Femenino de la Vicuña (*Vicugna vicugna*), su Irrigacion e Inervacion [Gross anatomy of the female reproductive organs of the vicuña]. Masters thesis. Universidad Nacional Altiplano, Puno.
7. Hoffman, E.; and Fowler, M.E. 1995. The Alpaca Book. Pioneer, California, Clay Press.
8. Montesinos, A.E. 1981. Contribucion a la Determinacion del Exterior de la Especie *Vicugna vicugna* (vicuña) del Proyecto de Pampa Galeras, Ayacucho [Contribution to the exterior morphology of the vicuña]. Masters thesis. Universidad Nacional Altiplano, Puno.
9. Urquieta, B.; and Rojas, J.R. 1990. Studies on the reproductive physiology of the vicuña (*Vicugna vicugna*). In Livestock Reproduction in Latin America. Vienna, International Atomic Energy, pp. 407–428.
10. Urquieta, B.; Cepeda, R.; Caceres, J.E.; Raggi, L.A.; and Rojas, J.R. 1994. Seasonal variation in some reproductive parameters of male vicuña in the high Andes of northern Chile. Journal of Arid Environments 26:79–87.



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Order Artiodactyla, Family Cervidae (Deer)

BIOLOGY AND MEDICINE

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TAXONOMY AND DISTRIBUTION

Neotropical cervids have not been well studied in relation to their taxonomy, especially the genus *Mazama* in which new species have been described recently. Studies have been hampered by lack of access to the majority of the species that live in dense forests. Morphological identification is not suitable for Neotropical Cervidae, and a karyotype analysis is needed. Biological data for South American cervids are found in Table 35.1.

GENETICS

Genetic research provides useful information that may help to solve conservation challenges in endangered deer. Completed research helped correct zoological classification of animals. The identification of individuals is applicable for captive breeding by avoidance of inbreeding; construction and management of pedigrees; monitoring genetic variability; fitness within captive populations; and identification and management of heritable diseases in a breeding group.

Chromosomal morphology of the South American cervid species is found in Table 35.1.

Protein polymorphism is, in general, much more definitive within populations and among taxa than chromosomes. The proteins represent the major end product of DNA-encoded information. Because some mammalian taxa exhibit chromosome variation, to study it in composition of amino acid proteins may provide useful information for analyzing at different levels.[158]

In general, large mammals such as deer display less protein polymorphism than do invertebrates and small mammals such as rodents. A study of transferrin system was performed in pampas deer.[73] Individuals from two populations (Emas National Park and Pantanal) were analyzed. Three alleles were observed (Tf^A, Tf^B, and Tf^C)

TABLE 35.1. Cervid biological data

Scientific Name	Common Names	Distribution	Habitat	Weight (kg)	Height (cm)	Karyotype (2n)	Reference
Blastocerus dichotomus	Marsh deer, ciervode los pantanos, cervo-do-pantanal	Central region of Brazil to Bolivia, northern Argentina, Paraguay	Swamps and lowland bordered by water	Male 130, Female 100	130	66	48, 54, 134, 178
Ozotoceros bezoarticus	Pampas deer, venado de las pampas, venado de campo, veado-campeiro	Central region of Brazil, Uruguay, Bolivia, Paraguay	Open fields, steppes, but never dense forests	30	65	68	29, 31, 48, 55, 124, 133, 168
Odocoileus virginianus	White-tailed deer, cariacu, veado-galheiro	Canada, USA, Central America, northern South America to Amazon River	Varied from forests to open areas	50	67	70	20, 48
Hippocamelus antisensis	Taruca	Andes from Ecuador south to Chile and Argentina	Rocky areas from 2400 to 4500 m	—	—	—	31, 154
Hippocamelus bisulcus	Huemul	Southern Andes of Argentina and Chile, to the Straits of Magellan	Rocky open areas	40–95	80–100	70	31, 168
Pudu puda	Southern pudu	Southern Chile, Valvidian forest	Wettest subantarctic grooves up to 1700 m	7–12	30–44	70	31, 109, 154
Pudu mephistophiles	Northern pudu	Paramos in Colombia, Ecuador, and central Peru	1700 to 4000 m	3.5–6.0	—	—	31, 42
Mazama gouazoubira	Gray brocket deer, brown brocket, Guazubira, soche gris, veado-catingueiro, veado-vira	From Brazil east of the Andes and south to Uruguay	Varied from forests to open and modified areas	18–20	50	70	46, 48, 56, 57, 134
Mazama nana	Brazilian dwarf brocket, corzuela enana, veado-bororó, mão-curta	From Paraná in Brazil, south to northern Argentina and Paraguay	Mountain areas covered by dense vegetation	15	45	36	48, 49, 56, 57, 182
Mazama americana	Red brocket, corzuela roja, soche colorado, veado-mateiro	Mexico to northern Argentina	Dense forests	30	65	7 different cytotypes in Brazil (42 to 53)	46, 48, 49, 57, 134

(continued)

TABLE 35.1. Cervid biological data—*continued*

Scentific Name	Common Names	Distribution	Habitat	Weight (kg)	Height (cm)	Karyotype (2n)	Reference
Mazama rondoni	Small gray brocket, veado-roxo, foboca	Southern Amazon region	No information	15	48	66–70	10, 30, 42, 48, 49, 146, 156
Mazama rufina	Little red brocket, Ecuadorian dwarf red brocket, black-faced brocket, soche enano, cervicabra	Andes in Ecuador, Colombia, and Venezuela	Mountains up to 2000 m	8.2	45	—	59, 179
Mazama chunyi	Peruvian dwarf red brocket	Andes of northwestern Bolivia and southeastern Peru		—	—	—	89
Mazama bororo	Small red brocket, bororo-de-São-Paulo	Atlantic rain forest of Brazil	Dense forests on steep hillsides	25	50	34	46, 48, 56, 57

in codominance methods. It was not possible to distinguish significant differences in the allele frequencies.

In a study of the *Mazama* genus, several polymorphisms were found in the transferrin, albumin, and hemoglobin systems.[74] Five species and nine phenotypes were found among *Mazama* species using the transferrin system. The species *Mazama nana, Mazama americana,* and *Mazama bororo* showed the same elctrophoretic pattern. Meanwhile, the species *M. nana and M. bororo* showed one similar phenotype. In *M. americana* and *Mazama rondoni* two phenotypes were found. The phenotypes observed in *M. rondoni* had a pattern similar to those found in *Mazama gouazoubira.* The most polymorphic was *M. gouazoubira,* which showed six phenotypes.

The differences observed between *M. nana* and *M. americana* were less than those observed among these species and *M. gouazoubira.* In conclusion, for species classification the markers should display enough variation among species to be distinctive. The systems analyzed were not suitable for identification of all the *Mazama* species.[74]

When protein polymorphism was analyzed in conjunction with chromosomal data, an unexpected association was found. The polymorphism level among the species is in inverse proportion to the chromosome variation detected.[46,49] The species *M. nana* and *M. americana,* which have restricted geographic distribution, showed high levels of karyotypic variability (diploid and fundamental number), and low polymorphism levels at the transferrin system. On the contrary, *M. gouazoubira,* a *Mazama* species with a wide geographic range, showed karyotypic stability (has the ancestral number 2n = 70, being all acrocentrics) and high polymorphism levels at the transferrin system.

Recent advances in molecular biology have resulted in the availability of a wide variety of techniques that may be used to assess kinship, patterns, and levels of genetic variation and evolutionary relationships. Molecular genetic markers may be used in conservation biology.

Mitochondrial DNA is a powerful tool to use to analyze the genetic variation of wild deer populations, and it is possible to deduce genetic units for conservation.[130] It has many advantages, but one of the most important is the possibility to amplify by polymerase chain reaction (PCR) small amounts of DNA, using universal primers.[108] This provides the ability to use DNA isolated from tissues obtained from dead animals in the field and other materials, such as shed antlers, hair, and feces, in analyses.

An example of the type of information that may be obtained when analyzing the mitochondrial control region may be found in a population genetic study conducted in the endangered Neotropical pampas deer.[81] To determine levels of genetic differentiation among isolated populations, DNA sequences were examined from the mitochondrial control region of 54 individuals from six localities.[81] The pampas deer's control region is one of the most polymorphic of any mammal. The existence of high levels of variation despite present-day low numbers indicates that the decrease in population size was recent, because variability would be lost rapidly in populations that had maintained a small size for long periods of time.[11]

This remarkably high variability likely reflects large historic population sizes of millions of individuals in contrast to numbers of fewer than 80,000 today.[80] Even though the surveyed pampas deer populations were small and isolated, they were genetically differentiated.

The molecular genetic results provide a mandate for habitat restoration so that levels of genetic variation can be preserved and historic patterns of abundance may be reconstructed.

This kind of research shows the importance of extending genetic analysis to other species and populations of Neotropical deer. The advantage of working with DNA is that it is possible to obtain copies from this genome in scanty material, like ancient material or feces. DNA analysis provides a useful approach to study species from a closed habitat like *Mazama.* The information that would be obtained, in conjunction with other genetic markers and biological data, will give guidelines for management of wildlife and captive populations, to elaborate recovery plans, to avoid interbreeding differentiated genetic units and also to answer or explain basic biological statements such as phylogenetic relations.

CONSERVATION

Habitat conversion for agriculture and other human activities has changed large tracts of grasslands, forest, and wetlands, affecting many Neotropical deer species. The introduction of cattle into South America has produced trophic competition and also brought new diseases to deer, with which their immune system is not prepared to cope. The status of South American cervids is listed in Table 35.2. More details may be obtained from the references.

It is now urgent to implement conservation measures at the ex situ and the in situ levels to achieve conservation goals. At the ex situ level, all South American zoos will need to undergo important changes that only some are now adopting. The first requirement is to correctly identify all of the individuals using electronic devices, tags, and tattoos. Managers need to record clearly the source of the individuals and to follow the pedigree.

TABLE 35.2. Conservation situation of South American deer species

	Country									
Species	AR	BRA	URU	PAR	BOL	CHI	EQ	PER	CITES	1996 IUCN Red List Category
Blastocerus dichotomus	EN	Vu	EX?	EN	Vu	X	X	EX?	I	Vu
Hippocamelus antisensis	EN	X	X	X	EN	Vu	EN	EN	I	DD
Hippocamelus bisulcus	EN	X	X	X	X	EN	X	X	I	EN
Ozotoceros bezoarticus	EN	LR nt	EN	DD	Vu	X	X	X	I	LR nt
Pudu puda	LR cd	X	X	X	X	Vu	X	X	I	Vu
Pudu mephistophiles	X	X	X	X	X	X	Vu	DD	II	LR nt
Mazama gouazoubira	LR lc	LR lc	DD	DD	DD	X	WE	DD	WE	LR lc
Mazama nana	Vu	Vu	X	X	X	X	X	X	WE	LR lc
Mazama americana	LR nt	DD	X	DD	DD	X	WE	LR lc	WE	LR lc
Mazama rufina	X	X	X	X	X	X	LR cd		WE	WE
Mazama chunyi	X	X	X	X	Vu	X	X	DD	WE	DD
Mazama bricennii	X	X	X	X	X	X	X	WE	WE	WE
Mazama bororo	X	DD	X	X	X	X	X	X	WE	LR lc
Mazama rondoni	X	DD	X	X	DD	X	X	WE	WE	WE
Odocoileus virginianus	X	DD	X	X	DD	X	WE	DD	WE	WE

Source: See references 63, 75, 79, 97, 176, and 182.
EN, endangered; EX?, possibly extinct; LR cd, lower risk conservation dependent; LR lc, lower risk least concern; LR nt, lower risk near threatened; Vu, vulnerable; DD, data deficient; X, default of the species; WE, without records; AR, Argentina; BRA, Brazil; URU, Uruguay; PAR, Paraguay; BOL, Bolivia; CHI, Chile; EQ, Equador; PER, Peru.

With this information, the studbook could once again be followed and genetic management could be implemented (see following item). Managers need to avoid augmentation efforts of the captive stock by capturing animals from wild populations.

All information regarding husbandry, diseases in captivity and treatments, development, growth and reproduction, which the zookeepers have compiled from years of working with the species, must be recompiled to be used as a general manual for the species in captivity.

The value of the captive herd will increase if more research is conducted, funded, and supported. It is imperative to obtain collaboration and support from the authorities and research centers to establish long-term research programs with captive herds in order to undertake a wide variety of projects in biological research and veterinary medicine. Results would be invaluable, because most of this research can only be done in captivity.

All of these conservation measures should be undertaken in conjunction with an education program using the captive stock and the zoo facilities to implement it. It would be a unique opportunity to offer general biological information about the species, the values of the native fauna, the conservation situation, and how the public can help.

A policy for the local environment must be considered. A conservation project should link the central policy, and, if necessary, try to change or create new legal instruments. It is important also to consider biological and socioeconomic criteria in the conservation project design.

A coordinated management strategy for a protected area is urgently required. The protected areas in these countries are in different conditions of management. Some of them have instrumented special programs for in situ conservation for the flag species. Others need to prepare recovery and management plans for the deer species. Also, effective controls should be implemented to control poaching.

GENETIC MANAGEMENT IN CAPTIVITY

Deer populations are declining so it is necessary to develop adequate genetic and reproductive methodologies for captive populations to ensure survival of some species and possibly to preserve the original alleles' genetic stock from natural populations. A major problem observed when trying to keep viable genetic populations ex situ is the selective process imposed by captivity, which may favor reproduction of only a few individuals. An example is *Blastocerus dichotomus,* whose founder population started with captured individuals from the inundated area of the Porto Primavera Hydroelectric Project in Brazil. One hundred fifteen animals were captured and maintained in quarantine for about 60 days before being moved to their new environment to start a new population. During quarantine, 35% of them died, mainly because of failure to adapt to captivity. The loss changed the breeding group. There are however means to minimize the effects generated by an artificial selection based on initial population using cryopreserved genetic material.[184] That genetic "picture" must be obtained by systematic material collection from the desired population to be preserved. After several captive generations, the genetic material may be reintroduced into the populations to recover the original allelic frequency.

The initial step taken to an effective program to keep viable an ex situ population is to define what is supposed to be conserved (species, subspecies, populations).[81]

Any good program to maintain viable captive populations must be based on a knowledge of the reproductive and behavioral features of these species in the wild. Then the application of genetic identification techniques is necessary to be certain that such populations are kept viable through generations.[70,170,174] A decrease in genetic diversity may cause low reproductive performance, less resistance against infectious and parasitic diseases, and less adaptation flexibility to a given environment.[113]

The initial heterozygosis in captive populations may be maximized if formed by 20–30 founders not related to each other, since this number must be representative from their original population allelic lot.[70]

It is more convenient to subdivide great populations into small ones, managed by several institutions, thus avoiding the risk of accidents that would certainly decrease the population. The integrity of a subdivided population (metapopulation) needs the use of supervised reproduction methodologies, mentioned above, because animal transport is a risk that must be strongly avoided.

Consanguinity must be avoided in small populations because it might increase substantially the possibility of deleterious recessive allele bindings that may cause young deaths. Consanguine crossbreeding may eventually be recommended in an equal contributor founders program that preferentially breeds descendants of underrepresented founders and restricts reproduction in those of overrepresented founders to avoid loss of founder alleles. However, endogamy must generally be avoided through pedigree control reports and the exchange of individuals and/or germplasm between different institutions, when necessary.

The size of the founder population must be large enough to exclude endogamous depression to keep the species at adaptive potential. It is thought that endogamous depression may be diminished if founder populations are composed of at least 50 individuals that are active reproducers, despite the fact that the negative effects of endogamous crossbreeding may be inevitable through generations. Populations containing 500 effective reproducers seem to be enough to keep the adaptive potential despite mutations.[71]

The concern about keeping a viable captive population bearing genetic diversity and reproductive capabilities unchanged from the original population will be of no value unless an effort is made to preserve the ecosystems from which those species came. In a few decades it might be possible to have great captive populations and be impossible to find a home for them.

HABITATS AND FOOD HABITS

Habitats are listed in Table 35.1. Deer are basically browsers, consuming a wide variety of plants in their habitat, including trees, shrubs, forbs, fruits, aquatic vegetation, and, to a limited extent, grass.[152]

REPRODUCTION

Male Reproduction

The male cervid reproductive cycle in northern temperate climates is well known. They have a defined breeding season (rut) related to the photoperiod that determines the secretion of reproductive hormones. The breeding season is strongly linked to secondary sexual characteristics such as the antler cycle, testicular diameter, and development of the musculature of the neck.[8]

Reproductive cycles of South American cervids are poorly known, especially the seasonal condition that seems to be occurring in *Ozotoceros* and *Blastocerus* but is controversial in *Mazama*. It was verified in a study accomplished with *Ozotoceros bezoarticus* in the pantanal as well as in the Parque Nacional de Emas, that the animals had a normal quality semen during the summer (December to February), but semen quality was poor during the winter (July to September).[53] Weekly semen collection of *M. gouazoubira* indicated a decrease in the semen quality during spring (September to November). It can be pointed out that the antler cycle in *Mazama* does not necessarily match the patterns of other species. In captive animals the antler cycle may extend to 11 years. Instead, they may lose the antlers when they are subjected to constant stress, for instance during quarantine periods or when they are frequently manipulated for ample collections. However some individuals from *M. americana* and *M. nana* species may have annual antler cycles.[72]

SEMEN COLLECTION AND CRYOPRESERVATION Semen collection in cervids may be accomplished as in domestic mammals by using an artificial vagina, vaginal collection, and electroejaculation. Electroejaculation is the most commonly used method for free-ranging animals. Chemical immobilization using 1–2 mg/kg of xylazine and 5 mg/kg of ketamine by intramuscular or intravenous injection is usually required, but it may not be needed if there has been previous conditioning of the male. Seemingly, these drugs do not interfere in the ejaculatory process. The electroejaculator is the same as that used for domestic animals, except for the electrode, which is designed especially for cervids. It was possible to obtain ejaculates in 90% of all animals from the genus *Mazama, Ozotoceros,* and *Blastocerus,* applying 10 electric stimuli of 3 seconds each, with increasing intensity from 250 to 750 mA and pauses of 3 seconds. The semen volume produced by males of *Mazama* and *Ozotoceros* varied from 0.1 to 0.7 mL, and in *Blastocerus* it reached 1.5 mL. In spite of the small volume, the concentration of spermatozoa was high, reaching 3×10^9 spermatozoa/mL, with an average of 1×10^9 spermatozoa/mL. The need for sedation is clearly a disadvantage, limiting the frequency of collection, because those drugs may be stressful.[52] Semen was frozen using a citric acid–buffered solution (4.54% Tris, 2.6% citric acid, and 0.75% glucose) with 2.5–10% egg yolk and 6% glycerol for dilution of the semen from several species of Brazilian cervids.[52] Better results were obtained with a special diluent made of 1.2 g Tes, 1.2 g Tris, 1.6 g glucose, 1.6 g fructose, 1.5 mL surfactant substance (amino-Na-lauryl sulfate), 20 mL egg yolk, 0.04 g G penicillin, and 0.1 g streptomycin in 100 mL water. Using such diluent, spermatozoa motility loss was only 20–30% after thawing.[127]

Female Reproduction

Female cervids are seasonally polyestrous, at least for temperate climate species. It is known, however, that in a tropical climate the breeding season is not well defined, especially for *Mazama,* in which the rut occurrence is noted after parturition, and in *M. americana,* demonstrating that in these species reproduction is not seasonal.[76] In *O. bezoarticus* a certain sexual season is seen, but births are concentrated during the spring (September to November) in Brazil and Argentina.[153,51,99] This period extends to December in Uruguay.[100] It should be emphasized that all experts consulted mention that births occur all year long, indicating that some males and females may be fertile throughout that time. Captive *Blastocerus* do not have an apparent breeding season, with the interval between parturitions approximately 9 months, coinciding with the gestational period that is also from 8.5 to 9 months. The estrous cycle in *M. gouazoubira* is approximately 21 days and in *Blastocerus* about 24 days.[131]

ESTROUS SYNCHRONIZATION Vaginal devices containing progesterone are used in several species of mammals with success, including cervids. Their function is to gradually release exogenous progesterone, which keeps them in the progestational phase. When such devices are removed, the abrupt fall of the progesterone level stimulates the production of gonadotrophic hormones, thus inducing ovulation after an average of 3 days after removal. Intravaginal devices soaked with medroxyprogesterone acetate or 17-hydroxy progesterone for 10–14 days, associated with prostaglandin administration on the last day of exogenous progesterone or when applied between the days 7 and 14 of the normal cycle, induces ovulation after 3 days in *M. gouazoubira.*[53]

SUPEROVULATION In cervids, superovulation has been used successfully after vaginal progesterone implants for 10–14 days and stimulation with 700–800 IU of pregnant mares' serum gonadotrophin (PMSG) in a single dose 2 days before or at the day of the prostaglandin injection, as well as removing the progesterone device. This has resulted in up to eight ova and embryos after vaginal or cervical insemination with fresh semen in *M. gouazoubira.*[53]

ARTIFICIAL INSEMINATION Knowledge of artificial insemination techniques in cervids will facilitate genetic exchange between nurseries and zoos, as well as allow better reproductive genetic handling of individual herds. Animals at the estrous phase exhibiting normal or progesterone-induced cycles, stimulated or not with PMSG or follicle-stimulating hormone (FSH), became pregnant when fresh semen was deposited in the vagina; however, pregnancy did not occur when frozen semen was used in Brazilian cervids.[53] The intrauterine insemination technique through laparoscopy has provided better pregnancy results if frozen semen is used. The latter has become the method of choice for cervids.[9,127]

EMBRYO COLLECTION AND TRANSFER Embryo transfer in cervids is based on experimental data from many individuals. Embryo collection using a catheter positioned at the horn of the uterus or the body of the uterus using an uterine flow and reflux wash through a syringe, provides the collection of few embryos. A surgical method consisting of positioning the catheter in the horn of the uterus followed by flow through a needle positioned at the uterus-tubal junction, toward the uterine body, provides larger collections in *Cervus elaphus.*[183,53]

There is not an actual need for embryo transfer for genetic improvement. However, species threatened with extinction may be aided by this technique, especially using interspecific embryo transfer. This technique might produce a threatened animal using nonthreatened ones. For instance, several offspring from *M. bororo* (a species strongly threatened with extinction) could be generated by implanting embryos in *M. gouazoubira* (the most common South American cervid). Thus, a single female could potentially produce 10–15 fawns a year instead of one.

RESTRAINT AND HANDLING

The capture of free-ranging deer becomes a fundamental base for studies that require closer access to the animals for sample collection, radio-transmitter installation, and biometry.

Only a few reports exist describing cervid capture in South America. The most common methods used in South America are drive nets, fast-setting nets, net guns, and wrestling.

- Drive nets are positioned at predefined places, where it is thought that the animal passes after some incentive or is forced to pass through to the net. This is a commonly used technique for the capture of wild ungulates.
- Fast-setting nets may be used only in open country. This method requires nets made of black polypropylene with 20-cm (8-in.) mesh, 100 m (328 ft) in length, and 2.5 m (8 ft) high. A van-type vehicle is used to locate the animals and to transport people needed for the operation. After visual contact is made, the vehicle is driven around the animal at a great distance so that the staff is able to remain a minimum distance for setting the nets. The vehicle then reduces its speed to allow handlers to get out and begin to erect the net. The net is supported by iron rods used to reinforce concrete 4 mm (1/8 in.) in diameter and 2 m (6.5 ft) high, with a hook on one end. The net is erected with the vehicle moving, completing a semicircle. Once the net is assembled, the vehicle continues its circular route, leaving the people hidden opposite the open side of the net. At the end of such a process there is a circle formed by the net side and another circle made by people, who little by little close up to direct the animal toward the net. When the deer collides with the net it collapses, falls from the rods, and entangles the deer. As fast as possible the animal is physically restrained, and the anesthetic agent is administered intravenously.
- Net guns, fired from a helicopter, were pioneered by New Zealanders and are now a commonly used capture technique. Shooter and pilot training is fundamental for the safety of such an operation because of the high danger present when using weapons inside an aircraft. Any negligence by the shooter may cause a fatal accident.

  This technique has been used to capture *Mazama* in forested areas; however, a helicopter is not used. Instead, the shooters are positioned in trees for capturing the animals at night. The shooters are positioned in places where signs of deer have been noticed previously or places used for feeding purposes.
- Wresting (bulldogging) technique was used for the capture of *B. dichotomus* in the flooded area of the Porto Primavera Hydroelectric Project. The technique consists of directing the animal to an area with heavy vegetation density or water courses, using a helicopter. The animal becomes entangled in the vegetation or becomes impeded in the water. A person jumps from the helicopter to restrain the animal, being aided by another person that jumps shortly after. If anesthesia is needed, an anesthetist will also be part of the crew, jumping after the first two people jump.
- Dogs are used for hunting in South America, and they may also be used for capturing deer, but special conditions are necessary for that. Dogs were efficient in capturing deer during flooding processes behind hydroelectric dams. The dogs are released in isolated areas to drive the deer toward the water, where a team should be waiting with boats. The animals are easily captured from the water and are restrained and anesthetized by personnel in the boat. In situations where the route of escape is known, it is possible to work with dogs driving the deer toward a net.

CAPTURE MYOPATHY

Capture myopathy (CM) is the generic name given to a complex syndrome clinically characterized by pain, muscular rigidity, incoordination, oliguria, and depression, followed by death. The condition was described originally in herbivores from East Africa,[102] but has subsequently been described in primates, pinnipeds, marsupials, canids, and birds.[15,36,180] Diseases of domestic animals, with similar pathophysiological features include equine exertional rhabdomyolysis, swine malignant hyperthermia, and ruminant white muscle disease.[96,180]

Capture myopathy has been reported to occur in Neotropical cervids[27,34,121,167,169,180] (E.R. Matushima, personal communication, 1999).

HOUSING

Enclosures for cervids are usually constructed as square or rectangular fenced areas. Although the concept is acceptable, in the majority of situations it hinders the handling and management of deer. Deer may not recognize a fence as an obstacle, especially when frightened.[77] It is important to keep a visual barrier at the fence, such as vegetation, masonry, wood, or even canvas. Vegetation at the fences seems to be the most viable option for zoos because it is inexpensive and provides a natural appearance for the enclosure. Corners should be rounded to avoid collision when a deer runs alongside the fence.

Fences should be between 1.8–2.5 m (6–8 ft) high. The mesh of fences should be of a size that prevents accidents that may occur when cervids poke their feet and antlers into the open spaces of the fence.[77] Fence posts must always be positioned outside the fencing material to avoid trauma associated with collision.

The surface of the enclosure should be dirt with grass providing a natural appearance. Most South American cervid species do not feed on grass regularly, therefore do not use rapidly growing grass. Plant a grass species that is adapted to the area and resistant to trampling. A mixture including a legume is always desirable, but there will be a loss of the legume over a period of time because of selective preference.

Ponds for maintaining marsh deer must always be accompanied by efficient parasite control because such an environment provides optimal conditions for parasites.

Trees should be planted in the yards to provide shaded areas for the animals in the hottest period of the day. Grass thickets or other shrubbery species should be avoided because cervids will tend to hide themselves during the day, hindering the visitor's view. Logs offer naturalistic conditions and may be placed in the center of an enclosure because deer avoid the center when stressed.

Some species, especially the marsh deer, are sensitive to heat, so a pond or lake should be included in the enclosure. Ponds must be shallow (1 m [3 ft] deep), preferably made with a dirt bottom to prevent slipping. For esthetic purposes, aquatic plants may be placed inside the ponds.

The handling system for cervids is extremely important, but is often forgotten in designing and constructing enclosures. Handling systems must have individual pens to separate animals from each other. The animals should stay in the exhibit enclosure during the day but be moved into the handling complex at night for feeding and observation. The majority of Neotropical cervids are crepuscular or nocturnal, which would make it logical to reverse where the deer are kept during the day and at night, but that would impair visitor viewing.

The handling complex should be constructed of masonry stalls similar to those used for equines, with their size varying according to the species. For large species such as *B. dichotomus,* a 4 × 4 m (13 × 13 ft) stall is recommended, whereas for small species such as *Mazama* or *Pudu* stalls measuring 2 × 3 m (6.5 × 9.8 ft) are acceptable. The minimum height of the wall should be 2 m (6.5 ft), but some may even jump that if excited. The walls must be as smooth as possible to avoid abrasions and prevent climbing. Stall floors should be sand or dirt and not concrete, if at all possible, because the hooves may be damaged by excessive wear or slipping on the concrete.

Food and water containers should be situated at the stall corners and must have an external access through a small door so that it is not necessary to enter the stall to feed them. Mangers should be portable to allow removal for capturing deer. An inclined manger near the stall door allows easy removal.

The stall is the safest and easiest place to handle the animals, and that is where the animals feel calm. Recently acquired deer should stay initially in stalls, and little by little they may be released to larger enclosures.

NUTRITION IN CAPTIVITY

South American cervids are browsers, consuming highly nutritious leaves and forbs. An exception is the pampas deer that feeds on newly sprouted grass. Several kinds of food are offered to South American captive cervids including fruits, green vegetables, legume hay, and grass. Species in the genus *Mazama* are frugivorous, but fruits can not be the basis of a captive nutritional program because these species need a minimum level of fiber. Low-fiber diets may cause animals to eat the bark of trees that are inside the enclosure. However, fruits may be used in moderation by all South American species. Deer are particularly fond of bananas. The fruit nutritional value should not be considered in the composition of a diet. Deer require time to adapt to forages offered in captivity. Concentrates may also be offered to complete the dietetic needs. Plants that are accepted by deer include lucerne (alfalfa) *Medicago sativa, Neonotonia wightii, Leucaena leucocephala, Galactia striata, Boehmeria nivea, Amaranthus deflexus, Commelina benghalensis,* and *Bidens pilosa*. If leguminous plants are used for hay in the diet, the protein levels of the concentrate should be between 14 and 16%, but if grass and fruits are offered in large amounts the protein level of the concentrate should be between 18 and 20%.

SEMIOLOGY

Semiology is the recognition of clinical signs. Deer are easily frightened, which makes evaluation of clinical signs difficult. Deer do not allow auscultation, palpation, percussion, and body temperature measurement.

The use of binoculars will help visual inspection and respiratory rate evaluation.

Physical restraint of animals, including those of the *Blastocerus* genera, is possible, but animals will struggle to free themselves and parameters will be altered. The use of immobilizing agents may allow collection of some data from palpation and percussion and samples for laboratory examination. However, these drugs may mask other signs. The continuous use of the same immobilization protocol may help to standardize information and establish a pattern to be used by the veterinarian.

ANESTHESIA

Chemical immobilization and anesthesia of deer are similar to that performed in other ungulates. The drugs and dosages used are described extensively in the references. Table 35.3 lists the drugs used in deer.

SURGERY

Surgical conditions of deer are managed as in cattle or horses.

Fractures of antlers are common in cervids, but are of limited importance because antlers are shed annually. Mature antlers will not heal, but when the antler is in velvet, fractures may be splinted or bone plates used to fix the segments together. Antler amputation may be necessary to correct deviations or fractures or even to minimize the risk of an accident with other animals or humans. The technique is simple. A single cut is made 5 cm (2 in.) above the pedicle with a hand or electric saw. Avoid cutting the antler when in velvet as it may hemorrhage profusely. If necessary, a tourniquet should be placed just proximal to the cut.

Elongated hooves are a constant problem in captive deer. Trimming necessitates chemical immobilization. Trimming and management of foot infections are handled as in cattle.

TABLE 35.3. Drugs used in some South American deer

Animal	Drug	Dose (mg/kg)	Comments
Brown brocket deer	Tiletamine + Zolazepan[141]	4–15 IV	Effects last 20–90 minutes
(Mazama gouazoupira)	Ketamine +	5–10 IM	Use atropine if needed
	Xylazine[b]	05–1.5 IM	
Red brocket deer	Ketamine +	8–10 IM	Add ketamine if needed
(Mazama americana)	Xylazine[b]	1 IM	
	Xylazine[b]	2–3	Minor surgical process
Dwarf red brocket deer	Ketamine +	8–10 IM, 5 IV	Suitable for electroejaculation
(Mazama rufina)	Xylazine[b]	1 IM, 2 IV	
Pampas deer	Ketamine +	8–10 IM	Minor surgical process
(Ozotoceros bezoarticus)	Xylazine[b]	1 IM	
	Ketamine +	5 IM	Antler amputation
	Xylazine[b]	1.5 IM	
	Diazepan	12 Oral	Good results, allowing easy
	Ketamine +	5 IM	induction
	Xylazine[b]	1 IM	
	Ketamine +	8–10	Deep immobilization
	Xylazine	1 IM	
	Diazepan (after 15 min)[b]	1 IM	
Marsh deer	Ketamine +	5–8 IV	Drugs injected in free-ranging
(Blastocerus dichotomus)	Detomidine +	100 μg IV	deer after physical restraint for
	Atropine[37]	0.05 IV	translocation purposes
	Ketamine + Midazolan[a]	5 IV	Poor muscle relaxation
		1 IV	
	Ketamine +	5 IV	Good immobilization, smooth
	Midazolan +	0.5 IV	recovery, stable physiological
	Acepromazine[a]	0.05 IV	data
	Ketamine +	5–8 IV	Good immobilization, rough
	Xylazine[a]	1 IV	recovery, hypotension
	Ketamine +	7 IV	Good immobilization, smooth
	Midazolan +	0.5 IV	recovery, hypotension
	Xylazine[a]	0.5 IV	

Source: See references 4 and 47.
[a]A.L.V. Nunes and M. L. Cruz, personal communication, 1999, not published.
[b]A.L.V. Nunes, personal communication, 1999, not published.

Periodontal diseases and alveolar abscesses are frequent in old nondomestic ruminants.[125] The problems are similar to those of cattle, sheep, goats, and other ungulates.

Castration may also be necessary in aggressive animals or if it is desired to sterilize a male. Some secondary sexual features such as color and antler regrowth may also be lost.[183]

ECTOPARASITES

A list of ectoparasites found in South American deer is found in Table 35.4.The diagnosis and management of ectoparasitism is the same as in domestic ruminants. The prevalence of babesiosis is reported in Table 35.5.

TABLE 35.4. Ectoparasites of South American deer

Host	Ectoparasite	Species	Reference
Blastocerus dichotomus	Fleas	*Ctenocephalides felis*	40, 171
	Flies	*Dermatobia hominis*	171
		Cochliomyia sp.	171
	Louse flies	*Lipoptena* sp.	160
	Ticks	*Amblyomma tigrinum*	32, 159, 171
		Amblyomma cajennense	32
		Boophilus microplus	32, 159, 171
		Anocentor nitens	171
Mazama sp.	Flies	*Dermatobia hominis*	160
	Louse flies	*Lipoptena* sp.	160
	Ticks	*Amblyomma cajennense*	32, 159
		Amblyomma maculatum	3
		Amblyomma mantiquirense	159
		Amblyomma ovale	4
		Amblyomma parvum	32, 86, 159
		Anocentor nitens	159
		Boophilus microplus	32, 159
		Haemaphysalis juxtakochi	87
		Haemaphysalis kohlsi	14,159
		Ixodes luciae	159
		Ixodes aragaoi	159
Ozotoceros bezoarticus	Flies	*Dermatobia hominis*	160
	Louse flies	*Lipoptena* sp.	160
	Ticks	*Amblyomma mantiquirense*	159
		Boophilus microplus	32, 116, 159
		Haemaphysalis kohlsi	159
Odocoileus virginianus	Ticks	*Amblyomma cajennense*	159
		Boophilus microplus	159

TABLE 35.5. Number and percentage of the species of studied deer, seropositives or seronegatives for *Babesia bovis* and *Babesia bigemina*, through indirect immunofluoresence assay, in the different Brazilian geographical areas

	Babesia bovis		*Babesia bigemina*	
Species	Positive (%)	Negative (%)	Positive (%)	Negative (%)
A (n = 38)	2 (5.26)	36 (94.74)	18 (47.37)	20 (52.63)
B (n = 69)	4 (95.80)	65 (94.20)	20 (28.99)	49 (71.01)
C (n = 27)	8 (29.63)	19 (70.37)	23 (85.19)	4 (14.81)
D (n = 5)	2 (40.00)	3 (60.00)	2 (40.00)	3 (60.00)
E (n = 3)	0 (0.00)	3 (100.00)	2 (66.67)	1 (33.33)
F (n = 3)	0 (0.00)	3 (100.00)	0 (0.00)	3 (100.00)
G (n = 41)	5 (12.20)	36 (87.80)	9 (21.95)	32 (78.05)
Total (%) (n = 186)	21 (11.29)	165 (88.71)	74 (39.78)	112 (60.22)

Note: n = number of animals; A, *M. nana;* B, *M. gouazoubira;* C, *M. americana;* D, *M. rondoni;* E, *M. bororo;* F, hibrid; G, *Ozotocerus bezoarticus.*

ENDOPARASITES

The endoparasites of deer vary according to the habitat and geographical location of the animals. The species of parasites are similar to those of domestic ruminants, and parasitism is managed as for domestic ruminants. Table 35.6 lists parasites recorded in South America. Therapy for nematodes is also similar to that used in domestic ruminants, including fenbendazole (5–15 mg/kg); oxfendazole (5–9 mg/kg) administered in two doses with a 48-hour interval between doses; rafoxanide (12.5 mg/kg) during 2 consecutive days; and ivermectin at 0.4 mg/kg, which must be administered subcutaneously.

Niclosamide at 90 mg/kg is recommended for flukes in sheep, and may be effective in deer. Rafoxanide 15–22.5 mg/kg and clorsulon 7 mg/kg are also used to treat flukes.

Hemoparasites

Hemoparasite infections caused by protozoa and rickettsial organisms are widely distributed in all continents and are responsible for great economic losses in ruminants found in the tropical and subtropical areas.

TABLE 35.6. Etiology of the helminthoses of South American deer

		Host		
Superfamilies and Species	**Habitat**	***Mazama***	***Ozotoceros***	***Blastocerus***
Trichostrongyloidea				
Haemonchus contortus	Abomasum	+	+	+
Haemonchus similis	Abomasum	+	+	+
Trichostrongylus axei	Abomasum	+	+	+
Trichostrongylus colubriformis	Small intestine	+	+	–
Trichostrongylus sp	Abomasum	–	–	+
Cooperia punctata	Small intestine	+	+	+
Cooperia pectinata	Small intestine	+	–	–
Spiculopteragia asymmetrica	Abomasum	–	–	+
Spiculopteragia trinitatis	Abomasum	–	–	+
Ancylostomatoidea				
Monodontus aguiari	Small intestine	+	+	–
Monodontus semicircularis	Small intestine	+	–	–
Bunostomum phlebotomum	Small intestine	–	–	–
Strongyloidea				
Eucyathostomum longesubulatum	Large intestine	+	–	–
Oesophagostomum dentatum	Large intestine	+	–	–
Spiruroidea				
Physocephalus sexalatus	Abomasum	–	+	–
Physocephalus lassancei	Abomasum	+	–	–
Texicospirura turki	Abomasum	–	+	–
Pygarginema verrucosa	Abomasum	+	–	+
Trichuroidea				
Capillaria bovis	Small intestine	+	–	–
Trichuris globulosa	Large intestine	–	–	+
Filarioidea				
Artionema bidentata	Abdominal cavity	–	+	–
Paramphistomoidea				
Paramphistomum liorchis	Rumen	+	+	+
Paramphistomum cervi	Rumen	+	+	–
Balanorchis anastrophus	Rumen	–	–	+
Zygocotyle lunatum	Large intestine	–	–	+
Taenioidea				
Moniezia benedeni	Small intestine	+	–	–
Moniezia expansa	Small intestine	+	–	–
Thysanosoma actinioides	Small intestine	+	–	–
Fascioloidea				
Fasciola hepatica	Liver	–	+	–

Boophilus microplus is a seriously harmful ectoparasite because it is a major vector for hemoparasites. Hemoparasites are a great problem in domestic ruminants in South America, and these animals are in close contact with wild animals, particularly deer.

Although babesiosis, caused by *Babesia bigemina* or *Babesia bovis* is an important disease of cattle in South America, the disease is not important in deer.

Bovine anaplasmosis has been responsible for decreased meat and milk production, abortion, weight loss, and death in the acute phase of the infection in South America; the main vector is the tick *Boophilus microplus,* although it may also be transmitted by other Ixodidae insects, hematophage insects, and by the transplacental route.[1,2,186] Little information is known about the prevalence of this rickettsia in wild South American deer. However, it is known that the coastal black-tailed deer *Odocoileus hemionus,* from California in North America, is infected naturally with *Anaplasma marginale* and is a carrier of the infection.[140]

Four *M. gouazoubira* were captured in the Pantanal region of Brazil and were transported to a farm located in an endemic area for anaplasmosis in the São Paulo State. Later, they developed characteristic clinical signs of anaplasmosis within 18 to 45 days after their transfer. It was verified that those deer were separated from cattle only by a single fence. The cattle harbored numerous *B. microplus* ticks containing rickettsia. Two deer died and the other two recovered after treatment with imidocarb and tetracycline.

The following species of trypanosomes are found in South America: *Trypanosoma vivax, Trypanosoma equiperdum, Trypanosoma theileri,* and *Trypanosoma evansi. T. evansi* has been reported in *Mazama sortorii* and *Odocoileus chirinquensos* in Panama.[115] Recently *T. evansi* was found in dogs and equines and *T. vivax* in ruminants in the Brazilian pantanal, but there are no reports regarding their presence in cervids in the same area.[161, 162, 163, 164] Hereafter we believe that other protozoan and rickettsial organisms will be diagnosed in Brazilian cervids.

INFECTIOUS DISEASES

Infectious diseases of deer that are ubiquitous and common in all ungulates include abscesses caused by numerous species of bacteria including *Yersinia pseudotuberculosis, Corynebacterium pyogenes, Escherichia coli, Pasteurella multocida, Streptococcus* spp., *Staphylococcus* spp., *Fusobacterium necrophorum,* and *Pseudomonas* spp. Other infectious diseases include clostridial diseases, leptospirosis, pneumonias, blue tongue and epizootic hemorrhagic disease (EHD).

Blue tongue (BT) and epizootic hemorrhagic disease of deer (EHD) are infectious, noncontagious, insect-borne viral diseases of domestic and wild ruminants.[62, 91] Such diseases, caused by oribiviruses, family Reoviridae, are characterized by congestion, edema, and hemorrhage of oral and nasal mucous membranes and laminitis.[179]

The epidemiology of BT and EHD depends on interactions of host, vector, climate, environment, and virus.[82] In South America only a few studies have been carried out to elucidate the epidemiology of these diseases.

Serological surveys of BT antibodies in llamas (*Lama glama*) in Argentina reported only negative animals, and in Chile and Ecuador the first positive animals were detected in 1985.[151,172,114] In the same year, the presence of BT and EHD virus serotypes was reported in serum samples from Colombian cattle.[94] In addition, antibodies to BT virus were found in ruminants in Surinam and Guiana.[88] However, it should be noted that these studies were done with domestic animals only.

In Brazil, the prevalence and potential vectors of BT and EHD are also unclear. Although fatal clinical cases and pathological findings suggestive of BT or EHD are frequently observed, only a few serological data have been reported in domestic and wild ruminant populations. The first serological investigation was done by agar gel immunodiffusion (AGID) in samples from deer and revealed only 13% seropositivity.[6] Recently, it was shown by a competitive enzyme-linked immunosorbent assay (C-ELISA) that 23 (9%) of captive brocket deer (genus *Mazama*) and 88 (74%) of wild marsh deer (*B. dichotomus*) were seropositive to BT and EHD.[142,143] It is necessary to conduct further investigations about these diseases in South America. These studies should include not only serological surveys but viral isolation, nucleic acid detection, serotyping, and vector characterization to establish solid epidemiological data and to improve the control of these diseases. Although the epidemiology is unclear, BT and EHD have been considered two of the most important infectious diseases of wild ruminants in the last 10 years. The epidemiological significance of BT and EHD in South America must be greater among domestic animals than in deer. Additionally, it may be assumed that both viruses are actively distributed all over South America.

During the period from 1994 to 1999, 25% of the necropsies of Cervidae performed at the Department of Pathology from the Veterinary Faculty of Jaboticabal (FCAV-UNESP) State São Paulo/Brazil showed typical lesions caused by an orbivirus, responsible for the hemorrhagic diseases like EHD and BT (Table 35.7). Because of the great similarity between the macroscopic lesions caused by this virus, necropsy results would be

TABLE 35.7. Results of necropsies in cervids during the period from 1994 to 1999

Cause Mortis	*N*	%
Suggestive lesions for EHD/BT*	16	25.00
Urinary bladder rupture, urethral compression	3	4.69
Respiratory insufficiency/pneumonia	21	32.81
Trauma (running over by car, shot)	2	3.13
Renal insufficiency	1	1.56
Sepsis/toxemic shock	8	12.50
Cardiac insufficiency	4	6.25
Ectoparasitism (fleas)	2	3.13
Endoparasitism (cestodas)	1	1.56
Diarrhea nonspecific	3	4.69
Peritonitis	1	1.56
Hepatic and renal congestion	1	1.56
Fracture	1	1.56
Total	64	100

*Epizootic hemorrhagic disease/blue tongue (see specific section in this chapter). Necropsies performed in the Department of Pathology of FCAV/UNESP of Jaboticabal, Brazil. The following species were appraised: *Mazama gouazoubira* (46), *M. americana* (2), *M. bororo* (3), *M. rondoni* (1), *Blastocerus dichotomus* (11), and *Ozotoceros bezoarticus* (1).

designed to be only suggestive for orbiviruses. The ages of the 16 affected animals varied between 1 and 10 years, and the disease occurred in both males (7) and females (9). Most of the necropsied deer were from the installations of the University of State of São Paulo. The lesions suggestive of EHD/BT were found in several deer species: *M. americana* (2), *M. nana* (1), *M. gouazoubira* (12), and *M. bororo* (1).

The classical antemortem clinical signs observed were facial edema (observed in 6 cases); submandibular edema (4 cases); lesions in the mouth, tongue swelling, petechiae on the gingivae, lip, and tongue (6 cases); intense salivation (1 case); and hemorrhagic feces (2 cases). The time between the first signs and death was 2–3 days. Other clinical signs were apathy, anorexia, prostration. These data agree with literature.[69] At necropsy, hydropericardium, hydrothorax, hydroperitoneum, and congested lymph nodes were observed as well as petechial and echymotic hemorrhages on the heart (endocardium, papillary muscles where the chordae tendineae insert, pericardium, bases of the large vessels at the atrium, apices of the heart, and thickening valves). Also affected were the lungs; bronchial mucous membrane and trachea; liver; spleen; kidneys; serum and mucus from the intestine; mucous surface of the rumen, reticulum, omasum, and abomasum; diaphragm; subcutaneous of the abdominal wall. The internal aspect of the legs; urinary bladder; and hard palate were also affected. Other alterations were hepatomegaly and splenomegaly with congestion. The intestinal contents were hemorrhagic. In one case, a lot of small granules were observed in the blood vessels suggestive of disseminated intravascular coagulation.

Lesions compatible with orbiviruses were described for a *B. dichotomus* in Brazil. This animal was serologically negative for orbiviruses, but may have died of an acute infection before an immune response occurred.[128]

There is no specific treatment for these diseases, but symptomatic therapy may be attempted to maintain the animal, until it can combat the virus through its own defense. Fluid therapy is crucial since the deer will refuse water and food. Long-acting antibiotics (benzathine penicillin, tetracycline) are recommended to avoid secondary infections. In that period the animal should receive a light feeding, especially with fruits, and be kept in a quiet, dark place. Even with these procedures, depending on the serotype involved, the prognosis is unfavorable.[7]

FOOT-AND-MOUTH DISEASE

Foot-and-mouth disease (FMD; aftosa), family *Picornaviridae,* genus *aphthovirus,* is one of the most economically important diseases of domestic livestock. All cloven-hoofed animals, including deer, are susceptible to the disease. The disease is characterized by rapid spread and considerable morbidity, approaching 100% in nonimmune populations. Infection with this virus may cause a severe, although rarely fatal, vesicular and acute febrile disease. Foot-and-mouth disease (FMD) has occurred in most countries of South America, often causing extensive epidemics in domestic cattle, sheep, and swine. Antibodies against FMD were found in free-ranging marsh deer, *B. dichotomus* (Araujo Jr. and Duarte, unpublished data, 1999) indicating previous infection and susceptibility to FMD. Data on the duration of the carrier state in deer is still unknown, but cattle continue to excrete virus for up 2.5 years after infection, sheep and goats for up to 9 months, and African buffalo for at least 5 years. Strain variation also affects the capacity of the virus to establish persistent infections. The preservation of wildlife is of increasing importance in many countries in South America, but because of the hazards of possible transmission of disease, mainly foot-and-mouth disease, from wild to domestic species, the interests of the conservationist may conflict with those of the livestock owner. To help attain the goal of control of FMD procedures must be established to prevent the transmission of FMD virus from wild to susceptible stock. When any susceptible species are captured, the detection of FMD virus–persistent carriers among animals is of paramount importance. To do this, FMD virus–persistent antibody should be promptly investigated by liquid-phase blocking sandwich ELISA since FMD virus establishes a persistent infection in animals in the presence of high levels of antibodies.[5,16] With negative results, the animal may be transported to areas FMD free.

In countries where prevention is accomplished by vaccination, these animals must be vaccinated using commercial vaccine. The pattern of response to vaccination is similar to that of cattle but generally of a lower order. However, in positive cases, quarantine is recommended, and when possible, oropharyngeal fluid should be collected, using the "probang" cup, for detection of persistent virus infection by isolation in cell culture (baby hamster kidney cell line BHK-21 and swine kidney cell line IBRS-2) and further identification by PCR.[170]

REFERENCES

1. Alonso, M.; Arrelano-Sota, C.; Ceresser, V.H.; Cordoves, C.O.; Guclielmone, A.A.; Kessler, R.; Mangold, A.J.; Nari, A.; Patarroyo, J.H.; Solari, M.A.; Veja, C.A.; Vizcaíno, O.; and Camus, E. 1992. Epidemiology of bovine anaplasmosis and babesiosis in Latin America and the Caribbean. Revue Scientifique et Technique de Office International des Épizooties 11(3):713–733.
2. Andrade, G.M. 1998. Estudos sobre a prevalência e infecção natural por *Anaplasma marginale* em bovinos da raça holandesa na região de Londrina, PR. Masters thesis, State University of Londrina.
3. Aragão, H.B. 1936. Ixodidas brasileiros e de alguns países limítrofes [Ixodida from Brazil and some neighboring countries]. Memórias do Instituto Oswaldo Cruz 31(4):759–844.
4. Aragão, H.B.; and Fonseca, F. 1961. Notas de ixodologia, VIII. Lista e chave para os representantes da fauna ixodológica brasileira [Notes on Ixodida, VIII. List and key for Brazilian ixodofauna]. Memorias do Instituto Oswaldo Cruz 59(2):115–130.
5. Araujo, J.P., Jr.; Montassier, H.J.; and Pinto, A.A. 1996. Liquid-phase blocking sandwich enzyme-linked immunosorbent assay for detection of antibodies against foot-and-mouth disease virus in water buffalo sera. American Journal of Veterinary Research 57:840–843,.
6. Arita, G.M.M. 1996. Bluetongue: Diagnostic in LARA/campinas. In Anais Congresso Panamericano de Ciencias Veterinárias. Campo Grande, p. 15.
7. Arita, G.M.M.; Morato, R.G.M.; and Duarte, J.M.B. 1997. Língua azul e/ou doença epizootica hemorrágica. In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 1–21.
8. Asher, G.W.; and Fisher, M.W. 1991. Reproductive physiology of farmed red deer (*Cervus elaphus*) and fallow deer (*Dama dama*). In L.A. Renecker and R.J. Hudson, eds., Wildlife Production: Conservation and Sustainable Development. Fairbanks, Alaska, University of Alaska Press, pp. 474–484.
9. Asher, G.W.; Kraemer, D.C.; Magyar, S.J.; Brunner, M.; Moerbe, R.; and Giaquinto, M. 1990. Intrauterine insemination of farmed Fallow deer (*Dama dama)* with frozen-thawed semen via laparoscopy. Theriogenology 34(3):567–577.
10. Avila-Pires, F.D. 1959. As formas Sul-Americanas do veado-virá. Anais da Academia Brasileira de Ciências 31(4):547–556.
11. Ballou, J. 1994. Small population overview. In R.C Lacy, K.A. Hughes, and T.J. Kreeger, eds., Vortex Users Manual: A Stochastic Simulation of the Extinction Process. Apple Valley Minnesota, IUCN SSC/CBSG, pp. 2–11.
12. Barbour, A.G. 1998. Fall and rise of Lyme disease and other Ixodes tick-borne infections in North America and Europe. British Medical Bulletin 54(3):647–58.
13. Barker, I.K.; Van Dreumel, A.A.; and Palmer, N. 1993. The alimentary system. In K.V.F. Jubb, P.C. Kennedy, and N. Palmer, eds., Pathology of Domestic Animals, Vol. 2, 4th Ed. San Diego, Academic Press, pp. 173–76
14. Barros, D.M.; and Baggio, D. 1992. Ectoparasites Ixodida, Leach 1817, on wild mammals in the state of Paraná, Brazil. Memorias do Instituto Oswaldo Cruz 87(2):291–296.
15. Bartsch, R.C.; McConnell, E.E.; Imes, G.D.; and Schimidt, J.M. 1977. A review of exertional rhabdomyolysis in wild and domestic animals and man. Veterinary Pathology 14:314–324
16. Baxt, B.; and Mason, P.W. 1995. Foot-and-mouth disease virus undergoes restricted replication in macrophage cell cultures following Fc receptor-mediated adsorption. Virology 207:503–509.
17. Beccaceci, M.D. 1994. A census of marsh deer in Ibera Natural Reserve, its Argentinean stronghold. Oryx 28:131–134
18. Beccaceci, M.D. 1996. Dieta del ciervo de los pantanos (*Blastocerus dichotomus*), en la Reserva Iberá, Corrientes, Argentina. Mastozoologia Neotropical 3(2):193–198
19. Beccaceci, M.D.; and Merino, M.L. 1994. Dieta del ciervo de los pantanos en la Reserva Iberá, Corrientes, Argentina. In L. Pinder and U. Seal, eds., Population and Habitat Viability Assessment for Cervo-do-pantanal (*Blastocerus dichotomus*). Apple Valley, Minnesota, IUCN/SSC Conservation Breeding Specialist Group, pp. 129–136.
20. Benirschke, K.; Brownhill, L.; Low, R.; and Hoefnagel, D. 1963. The chromosomes of the white-tailed deer, *Odocoileus virginiana borealis,* Miller. Mammalian Chromosomes Newsletter 10:82–84.
21. Beringer, J.; Hansen, L.P.; Wiling, W.; Fischer, J.; and Sheriff, S.L. 1996. Factors affecting capture myopathy in white-tailed deer. Journal of Wildlife Management 60:373–380
22. Birman, D. 1990. Doença de Lyme [Lyme disease]. Manchete Julho:58–61.
23. Blumer, E. 1991. A review of the use of selected neuroleptic drugs in the management of nondomestic hoofstock. In Proceedings of the American Association of Zoo Veterinarians. Calgary, pp. 333–339

24. Bodmer, R. 1989. Frugivory in Amazon Ungulates. Doctoral thesis, University of Cambridge.
25. Boever, W.J. 1986. Noninfectious diseases. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 1–127.
26. Branan, W.; Werkhoven, M.; and Marchinton, R. 1985. Food habits of brocket and white-tailed deer in Suriname. Journal of Wildlife Management 49:972–976.
27. Breazile, J.E. 1988. The physiology of stress and its relationship to mechanism of disease and therapeutics. Veterinary Clinics of North America Small Animal Practice 4:441.
28. Busch, M. 1996. A technique for endotracheal intubation of nondomestic bovids and cervids. Journal of Zoo and Wild Animal Medicine 27(3):378–381.
29. Cabrera, A. 1943. Sobre la sistemática del venado y su variación individual y geográfica. Revista del Museo de La Plata, Zoologia (nueva serie), Tomo III, 18:5–41.
30. Cabrera, A. 1960. Catalogo de los mamiferos de America del Sur. Revista Museo Argentino Bernardino Rivadavia 4:309–732.
31. Cabrera, A.; and Yepes, J. 1961. Los Mamíferos Sudamericanos. Buenos Aires, Ediar.
32. Campos Pereira, M.; Szabó, M.P.J.; Bechara, G.H.; Matushima, E.R.; Duarte, J.M.B.; Fielden, L.; Keirans, J.E.; and Rechav, Y. 1999. Ticks associated with wild animals in the Pantanal region of Brazil. Journal of Medical Entomology (submitted).
33. Carrigan, M.J.; Dawkins, H.J.S.; Cockram, F.A.; and Hansen, A T. 1991. *Pasteurella multocida s*epticaemia in fallow deer (*Dama dama*). Australian Veterinary Journal 68(6):201–203.
34. Catão-Dias, J.L.C. 1997. Miopatia de captura. In J.M.B. Duarte, ed., Biologia, Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotocerus* e *Mazama*. Jaboticabal, FUNEP, pp. 172–179.
35. Chakarboty, A.; Chauhury, B.; and Sarma, D.K. 1995. Pneumonia as the cause of death in deer. Indian Journal of Veterinary Pathology 19(1):30–34.
36. Chalmers, G.A.; and Barrett, M.W. 1982. Capture myopathy. In J.L. Hoof and J.W. Davis, eds., Noninfectious Diseases of Wildlife. Ames, Iowa, Iowa State University Press, pp. 84–94.
37. Charity, S.E.; Buschinelli, M.C.P.; Gasparini, R.L.; Tomás, W.M.; Nunes, A.L.V.; Oliveira, P.; Santiago, M.E.B.; and Cury, M. 1991. The use of ketamine/detomidine association in the chemical immobilization of free living marsh deer *Blastoceros dichotomus*. In Proceedings of Congresso Mundial de Veterinaria. Rio de Janeiro, pp. 30.
38. Clarck, C.H.; Kiesel, G.K.; and Goby, C.H. 1962. Measurements of blood loss caused by *Haemonchus contortus* infection in sheep. American Journal Veterinary Research 23(96):977–980.
39. Clark, R.K.; and Jessup, D.A. 1992. Mechanical or physical capture of animals. In R.K. Clark and D.A. Jessup, eds., Wildlife Restraint Series. Fort Collins, International Wildlife Veterinary Services, pp. 13–33.
40. Costa Lima, A.; and Hathaway, C.R. 1946. Pulgas: Bibliografia, catálogo e animais por elas sugados [Fleas: Bibliography, catalogue and parasitized animals]. Monografias do Instituto Oswaldo Cruz 4:1–522.
41. Cotran, R.S.; Kumar, V.; and Robbins, S.L. 1994. Pathologic Basis of Disease, 5th Ed. Philadelphia, W.B. Saunders, p. 1400.
42. Czernay, S. 1987. Spießhirshe und Pudus. Die Gattungen Mazama und Pudu. A. Ziemsen Verlag, Wittenberg Lutherstadt, pp. 84.
43. Dakkak, A. 1984. Physiopathologie digestive des trichostrongylidoses ovines. Note 2: Physiopathologie digestive de l'haemonchose ovine. Revue Medicine Veterinaire 135(7):459–467
44. Dingwall, J.S.; Duncan, D.B.; and Horney, F.D. 1971. Compression plating in large animal orthopedics. Journal of American Veterinary Medical Association 158:1651–1657.
45. Dresser, B.L.; Pope, C.E.; Kramer, L.; Kuehn, G.; Dahlhausen, R.D.; Maruska, E.J.; Reece, B.; and Thomas, W.D. 1986. Birth of Bongo antelope (*Tragelaphus euryceros*) to Eland antelope (*Tragelaphus oryx*) and cryopreservation of Bongo embryos. Theriogenology 23:190.
46. Duarte, J.M.B. 1992. Aspectos Taxonômicos e Citogenéticos de Algumas Espécies de Cervídeos Brasileiros. Masters thesis, Faculdade e Ciencias Agrárias e Veterinárias, Jaboticabal.
47. Duarte, J.M.B. 1993. A Ação da pipotiazina sobre o ponto de fuga do veado catingueiro *Mazama gouazoubira*. Arquivos da Sociedade de Zoológicos do Brasil no. 14:93–94.
48. Duarte, J.M.B. 1996. Guia de Identificação de Cervídeos Brasileiros. Jaboticabal, FUNEP.
49. Duarte, J.M.B. 1998. Análise Citogenética e Taxonômica do Gênero *Mazama* (Cervidae; Artiodactyla) no Brasil. Doctoral thesis, Instituto de Biociencias, Botucatu.
50. Duarte, J.M.B.; and Arita, G.M.M. 1993. Aspectos patológicos e epizootiologicos da doença hemorrágica causada por orbivirus em *Mazama gouazoubira*. In Anais 17° Congresso Brasileiro e 1° Encontro Internacional da Sociedade de Zoológicos do Brasil. Goiânia, SZB, p. 93.
51. Duarte, J.M.B.; Arantes, I.G.; Garcia, J.M.; and Nascimento, A.A. 1993. Captura e avaliação de uma população de *Ozotoceros bezoarticus* leucogaster no Brasil. In Population and Viability Assessment for the Pampas Deer. SSC/UICN, Montevideo.
52. Duarte, J.M.B.; and Garcia, J.M. 1989. Colheita e criopreservação do sêmen de Veado Catingueiro (*Mazama gouazoubira*). Ciencia Veterinária, Jaboticabal 3(1):8–10.
53. Duarte, J.M.B.; and Garcia, J.M. 1995. Reprodução assistida em Cervidae brasileiros. Revista Brasileira de Reprodução Animal 19(1(2):111–121.
54. Duarte, J.M.B.; and Giannoni, M.L. 1995. Cytogenetic analysis of the marsh deer (*Blastocerus dichotomus*). Revista Brasileira Genética 18(2):245–248.

55. Duarte, J.M.B.; and Giannoni, M.L. 1995. Cytogenetic analysis of the pampas deer, *Ozotoceros bezoarticus* (Mammalia, Cervidae). Revista Brasileira Genética 18(3):485–488.
56. Duarte, J.M.B.; and Jorge, W. 1996. Chromosomal polimorphism in several populations of deer (genus *Mazama*) from Brazil. Archivos de Zootecnia 45(170(171):281–287.
57. Duarte, J.M.B.; and Merino, M.L. 1997. Taxonomia e evolução. In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 1–21.
58. Edebes, H.; and Burroughs, R. 1989. Long acting neuroleptics in wildlife. Paper presented at the Tranquilizer Symposium, National Zoological Gardens, Pretoria.
59. Eisemberg, J.F. 1987. The evolutionary history of the cervidae with special reference to the South American radiation. In C.M. Wemmer, ed., Biology and Management of the Cervidae. Washington, D.C., Smithsonian Institution Press, pp. 60–64.
60. Eisemberg, J.F. 1989. Mammals of the Neotropics. Vol. 1. Chicago, University of Chicago Press.
61. Eldridge, W.D.; MacNamara, M.; and Pacheco, N. 1987. Activity patterns and habitat utilization of pudu (*Pudu puda*) in south-central Chile. In C.M. Wennner, ed., Biology and Management of the Cervidae. Washington, D.C., Smithsonian Institution Press, pp. 352–370.
62. Erasmus, B.J. 1975. Bluetongue in sheep and goats. Australian Veterinary Journal 51:165–170.
63. Ergueta, P.S.; and De Morales, C. 1996. Libro Rojo de los Vertebrados de Bolivia. La Paz, Centro de Datos para la Conservación, p. 345.
64. Faine, S. 1994. Leptospira and Leptospirosis. Boca Raton, Florida, CRC Press, p. 353.
65. Foreyt, W.; and Trainer, D.O. 1970. Experimental haemonchosis in white-tailed deer. Journal of Wildlife Diseases 6:35–42.
66. Forrester, D.J.; Taylor, W.J.; and Nair, P.C. 1974. Strongyloidiasis in captive white-tailed deer. Journal of Wildlife Diseases 10:11–17.
67. Fournier, J.S.; Gordon, J.C.; and Dorn, R. 1986. Comparison of antibodies to *Leptospira* in white-tailed deer (*Odocoileus virginianus*) and cattle in Ohio. Journal of Wildlife Diseases 22(3):335–339.
68. Fowler, M.E. Ed. 1986. Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders.
69. Frank, J.F.; and Willis, N.G. 1975. Bluetongue-like disease of deer. Australian Veterinary Journal 51:174–177.
70. Frankham, R. 1995. Conservation genetics. Annual Review of Genetics 29:305–327.
71. Franklin, I.R. 1980. Evolutionary changes in small populations. In M.E. Soulé and B.A. Wilcox, eds., Conservation Biology: An Evolutionary Ecological Perspective. Sunderland, Massachusetts, Sinauer, 135–149.
72. García, J.E.; and Duarte, J.M.B. 1994. Variação anual do espermiograma do veado catingueiro, sob condições de cativeiro. In Anais 18° Congresso da Sociedade de Zoológicos do Brasil. Rio de Janeiro, SZB, p. 41.
73. García, J.E.; and Rodríguez Ramos, P.R. 1996. Análise do polimorfismo genético-bioquímico das transferrinas em *Ozotoceros bezoarticus*. In J.M.B. Duarte, ed., Relatorio Final de Pesquisa, Projeto Veado Campeiro *Ozotoceros bezoarticus*. Jaboticabal, FUNEP, pp. 85–95.
74. García, J.E. 1997. Polimorfismo genetico-bioquimico das tranferrinas, albuminas sericas e hemoglobinas em cervideos do genero *Mazama*. Masters thesis, Instituto de Biociencias, Universidade Estadual Paulista, p. 61.
75. García Fernandez, J.J.; Ojeda, R.A ; Fraga, R.M.; Diaz, G.B.; and Baigun, R.B. 1997. Libro Rojo: Mamíferos y Aves Amenazados de la Argentina. Buenos Aires, Fucema, Sarem and Aop, p. 221.
76. Gardner, A.L. 1971. Postpartum estrus in a Red Brocket Deer, *Mazama americana*, from Peru. Journal of Mammalogy 52(3):623–624.
77. Gasparini, R.L.; Nunes, A.L.V.; and Duarte, J.M.B. 1997. Manejo em cativeiro. In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 125–140.
78. Geller, S.A. 1974. Extreme exertional rhabdomyolysis: A histopathologic study of 31 cases. Human Pathology 4:241–250.
79. Glade, A. 1993. Red List of Chilean Terrestrial Vertebrates. Santiago, CONAF, p. 69.
80. González, S.; Merino, M.; Gimenez-Dixon, M.; Ellis, S.; and Seal, U.S. 1994. Population Habitat Viability Assessment for the Pampas Deer (*Ozotoceros bezoarticus*). Workshop Report. Apple Valley, Minnesota, CBSG/IUCN, p. 171.
81. González, S.; Maldonado, J.E.; Leonard, J.A.; Vilà, C.; Barbanti Duarte, J.M.; Merino, M.; Brum-Zorrilla, N.; and Wayne, R.K. 1998. Conservation genetics of the endangered pampas deer (*Ozotoceros bezoarticus*). Molecular Ecology 7:47–56.
82. Gorman, B.M. 1990. The bluetongue viruses. Current Topics in Microbiology and Immunology 162:1–19.
83. Goyal, S.M.; Mech, L.M.; and Nelson, M.E. 1992. Prevalence of antibody titers to *Leptospira* spp. in Minnesota white-tailed deer. Journal of Wildlife Diseases 28(3):445–448.
84. Griffin, J.F.T. 1987. Acute bacterial infections in farmed deer. Irish Veterinary Journal 41(9):328–331.
85. Groves, C.P.; and Grubb, P. 1987. Relationships of living deer. In C.M. Wemmer, ed., Biology and Management of the Cervidae. Washington, D.C., Smithsonian Institution Press, pp. 21–59.
86. Guglielmone, A.A.; Mangold, A.J.; and Keirans, J.E. 1990. Redescription of the male and female of *Amblyomma parvum* Aragao, 1908, and description of the nymph and larva, and description of all stages of *Amblyomma pseudoparvum* sp. (Acari: Ixodida: Ixodidae). Acarologia 31(2):143–159.
87. Guglielmone, A.A.; Mangold, A.J.; and Aufranc, C.R. 1992. *Haemaphysalis juxtakochi, Ixodes pararicinus* (Ixodidae) and *Otobius megnini* (Argasidae) in relation to the phytogeography of Argentina. Annales de Parasitologie Humaine et Comparee 67(3):91–93.

88. Gumm, I.D.; Taylor, W.P.; Roach, C.J.; Alexander, F.C.; Greiner, E.C.; and Gibbs, E.P. 1984. A serologic study of ruminant livestock in some countries of the Caribbean and South America for type specific antibody to bluetongue and epizootic haemorrhagic disease of deer. Veterinary Records 114:635–638.
89. Hershkovitz, P. 1959. A new species of South American Brocket, Genus *Mazama* (Cevidae). Proceedings of the Biological Society of Washington 72:45–54.
90. Hickox, J.P. 1970. Treatment of fractures with hirschorn compression plates. Journal of American Veterinary Medical Association 156:187–196.
91. Hoff, G.L.; and Trainer, D.O. 1978. Bluetongue and epizootic hemorrhagic disease viruses: Their relationship to wildlife species. Advances in Veterinary Science and Comparative Medicine 22:111–132.
92. Hofmann, R.R.; and Stewart, D.R. 1972 Grazer or browser? A classification based on the stomach structure and feeding habits of East African ruminants. Mammalia 36:226–240.
93. Hofmann, R.R. 1985. Digestive physiology of the deer: Their morphophysiological specialisation and adaptation. In A. Dobson and P. Fenessy, eds., Biology of Deer Production. Royal Society of New Zealand Bulletin 22:393–407.
94. Homan, E.J.; Taylor, W.P.; de Ruiz, H.L.; and Yuill, T.M. 1985. Bluetongue virus and epizootic haemorrhagic disease of deer virus serotypes in northern Colombian cattle. Journal of Hygiene 95:165–172.
95. Horack, I.G. 1971. Paramphistomiasis of domestic ruminants. Advanced Parasitology 9:33–72
96. Hulland, T.G. 1993. Muscle and tendon. In K.V.F. Jubb, P.C. Kennedy, and N. Palmer, eds., Pathology of Domestic Animals, Vol. 1. San Diego, California, Academic Press, pp. 183–264.
97. IUCN 1996. Red List of Threatened Animals. Gland, Switzerland, IUCN, p. 366.
98. Jackson, J.; and Giulietti, J. 1988. The food habits of pampas deer *Ozotoceros bezoarticus celer* in relation to its conservation in a relict natural grassland in Argentina. Biological Conservation 45:1–10.
99. Jackson, J.E.; and Langguth, A. 1987. Ecology and status of the pampas deer in the Argentinean pampas and Uruguay. In C.M. Wemmer, ed., Biology and Management of the Cervidae. Washington, D.C., Smithsonian Institution Press, pp. 402–409.
100. Jackson, J.; Landa, P.; and Langguth, A. 1980. Pampas deer in Uruguay. Oryx 15:267–272.
101. Jalanka, H.H.; Teravainen, E.; and Kivalo, M. 1992. Propofol—A potentially useful intravenous anesthetic agent in nondomestic ruminants and camelids. In Proceedings of the American Association of Zoo Veterinarians, Oakland, pp. 264–270.
102. Jarrett, W.F.H; and Murray, M. 1967. Muscular dystrophy in antelope and gazelle in Kenya. Veterinary Record 80: 483.
103. Jeandel, M.F.M.R. 1969. Contribution à l'Etude des Infections Parasitaires dés Cérvides. Doctoral thesis, Ecole Nationale Veterinaire D'Alfort, p. 114.
104. Jensen, H.E.; Jorgensen, J.B.; and Schonheyder, H. 1989. Pulmonary mycosis in farmed deer: Allergic zygomycosis and invasive aspergillosis. Journal of Medical and Veterinary Mycology 27(5):329–334.
105. Jones, D.M. 1983. The capture and handling of deer. In A.J.B. Rudge, ed., The Capture and Handling of Deer. Nature Conservancy Council Handbook. London, pp. 1–115.
106. Jorge, W.; and Benirschke, K. 1977. Centromeric heterochromatin and G-banding of the red brocket deer, *Mazama americana temama* (Cervoidea, Artiodactyla) with a probable non-robertsonian translocation. Cytologia 42:711–721.
107. Jubb, K.V.F.; Kennedy, P.C.; and Palmer, N. 1993. Pathology of Domestic Animals, 4th Ed. San Diego, Academic Press, pp. 655–659.
108. Kocher, T.D.; Thomas, W.K.; Edwards, A.; Pääbo, S.V.; Villablanca, F.X.; and Wilson, A.C. 1989. Dynamics of mitochondrial DNA evolution in animals: Amplification and sequencing with conserved primers. Proceedings of the national Academy of Science USA 86:6196–6200.
109. Koulischer, L.; Tyskens, J.; and Mortelmans, J. 1972. Mammalian Cytogenetics, VII. *Elaphurus davidianus, Cervus nippon* and *Pudu pudu*. Acta Zoologica et Pathologica Antverpiensia 56:25–30.
110. Krogh, H.V.; and Jensen, A.M. 1988. Diagnostic examinations of autopsy material submitted from farmed deer in Denmark: Management and health of farmed deer. In Current Topics in Veterinary Medicine 48. Dordrecht, Kluwer Academic Publishers, pp. 71–78.
111. Kuttler, K.L. 1988. World-wide Impact of Babesiosis. In M. Ristic, ed., Babesiosis of Domestic Animals and Man. Boca Raton, Florida, CRC Press, pp. 2–22.
112. Kutzer, E. 1988. Ectoparasite control with ivermectin (Ivomec) on cloven-hoofed animals (red deer, roe deer, wild boar). Mitteilungen der Deutschen Gesellschaft für Allgemeine und Angewandte Entomologie 6–1(3):217–222.
113. Lacy, R.C. 1997. Importance of genetic variation to the variability of mammalian populations. Journal of Mammalogy 78(2):320–335.
114. Lopez, W.A.; Nicoletti, P.; and Gibbs, E.P. 1985. Antibody to bluetongue virus in cattle in Ecuador. Tropical Animal Health Production 17:82.
115. Losos, G.J. 1980. Diseases caused by *Trypanosoma evansi:* A review. Veterinary Research Communications 4:165–181.
116. Machado, R.Z.; Ferreira, F.A.; Machado, C.R.; Rocha, U.F.; and Toledo, C.Z.P. 1985. Ecology of ticks, XIII. *Boophilus microplus* (Canestrini, 1887) in natural infestation of deer (*Ozotocerus bezoarticus bezoarticus,* Linnaeus, 1766) and capybara (*Hydrochoerus hydrochoeris hydrochoeris,* Linnaeus, 1762) in the states of Sao Paulo and Mato Grosso do Sul. Ars Veterinaria 1(1):47–50.
117. Machado, R.Z.; and Müller, E. 1996. Freqüência de anticorpos contra *Babesia* em veado campeiro (*Ozotocerus bezoarticus*) no Pantanal e no Parque das Emas. Relatório Final de Pesquisa Projeto Veado Campeiro (*Ozotocerus bezoarticus*). Jaboticabal, UNESP, pp. 124–(132.

118. Malviya, H.C.; Patanaik, B.; Tiwari, H.C.; and Sharma, B.K. 1979. Measurement of the blood loss caused by *Haemonchus contortus* infection in sheep. Indian Veterinary Journal 56:709–710
119. Martin, C.J.; and Ross, J.C. 1934. A minimal computation of the amount of blood removed daily by *Haemonchus contortus*. Journal of Helminthology 12(3)137–142.
120. Mathias, L. A.; Girio, R.J.S.; and Duarte, J.M.B. 1999. Serosurvey for antibodies against *Brucella abortus* and *Leptospira interrogans* in pampas deer from Brazil. Journal of Wildlife Diseases 35(1):112–114.
121. Menezes, S.R. 1998. Relato de caso de miopatia de captura em *Mazama gouazoubira* tratado por homeopatia. In Proceedings of the 2nd National Congress of the Brazilian Association of Zoo and Wild Animals. Foz do Iguaçu, ABRAVAS, p. 5.
122. Merino, M.L. 1993. Dieta estival del huemul (*Hipocamelus bisulcus,* Molina 1782) en el Parque Nacional "Los Glaciares", Santa Cruz, Argentina. In J.A. Monjeau, ed., Resúmenes VIII Jornadas Argentinas de Mastozoología, Bariloche, pp. 46.
123. Merino, M.L. 1993. Dieta del venado de las pampas (*Ozotoceros bezoarticus* Linneus, 1758) en la Reserva de Vida Silvestre "Campos del Tuyú", Bahía de Samborombon, Provincia de Buenos Aires, Argentina. In J.A. Monjeau, ed., Resúmenes VIII Jornadas Argentinas de Mastozoología.
124. Merino, M.L.; Gonzalez, S.; Leeuwenberg, F.; Rodrigues, F.H.G.; Pinder, L.; and Tomas, W.M. 1997. Veado-campeiro (*Ozotoceros bezoarticus*). In J.M.B. Duarte, ed., Biologia e conservação de cervídeos Sul-Americanos: *Blastocerus, Ozotoceros e Mazama.* Jaboticabal, FUNEP, pp. 41–58.
125. Miller, F.L.; Cowley, A.J.; Choquette, P.L.E.; and Broughton, E. 1975. Radiographic Examination of mandibular lesions in barrenground caribou. Journal of Wildlife Diseases 11:465–470.
126. Mitrofano, P.M.; and Lunitsyn, V.G. 1985. Pathology or pasteurellosis in deer kept for pantocrine production (*Cervus nippon* and *C. elaphus*). Veterinariya [Moscow] 11:43–45.
127. Monfort, S.L.; Asher, G.W.; Wildt, D.E.; Wood, T.C.; Schiewe, M.C.; Williamson, L.R.; Bush, M.; and Rall, W.F. 1993. Successful intrauterine insemination of Eld's deer (*Cervus eldi thamin*) with frozen-thawed spermatozoa. Journal of Reproduction and Fertility 99:459–465.
128. Morato, R.G.; Dias, J.L.C.; and Arita, G.M.M. 1993. Ocorrência de doença hemorrágica em cervo do pantanal (*Blastocerus dichotomus*). In Anais 17° Congresso Brasileiro e 1° Encontro Internacional da Sociedade de Zoológicos do Brasil. Goiânia, SZB,p. 92.
129. Moreno, D.I. 1993. Ciervos autoctonos de la República Argentina. Boletin Técnico da Fundacion Vida Silvestre Argentina 17:1–40.
130. Moritz, C. 1995. Uses of molecular phylogenies for conservation. Philosophical Transactions of Royal Society of London B 349:113–118.
131. Müller, E.; and Duarte, J.M.B. 1992. Utilização da citologia vaginal esfoliativa para monitoração do ciclo estral em veado catingueiro. In Anais 16° Congresso da Sociedade de Zoológicos do Brasil. Americana, SZB, pp. 1–2.
132. Müller, E.; Machado, R.Z.; and Duarte, J.M.B. 1999. Prevalência de *Babesia bovis* e *Babesia bigemina* em cervídeos brasileiros. Veterinary Parasitology (in review).
133. Neitzel, H. 1979. Chromosome evolution in der Familie de Hirsche (Cervidae). Bongo 3:27–38.
134. Neitzel, H. 1987. Chromosome evolution of Cervidae: Karyotypic and molecular aspects. In G. Obe and A. Basler, eds., Cytogenetics: Basic and Applied Aspects. Berlin, Springer Verlag, pp. 90–112.
135. New, J.C.; Wathen, W.G.; and Dlutkowski, S. 1993. Prevalence of *Leptospira* antibodies in white-tailed deer, Cades Cove, Great Smoky Mountains National Park, Tennessee, USA. Journal of Wildlife Diseases 29(4):561–567.
136. Nielsen, L. 1999. Chemical Immobilization of Wild and Exotic Animals. Ames, Iowa, Iowa State University Press.
137. Nunes, A.L.V. 1991. Uso de antagonista alfa 2 adrenergico (cloridrato de ioimbina) em animais silvestres. Arquivos da Sociedade de Zoologicos do Brasil no. 12.
138. Nunes, A.L.V.; Buschinelli, M.C.P.; Charity, S.E.; and Tomás, W.M. 1989. Testes com drogas anestesicas em alguns cervídeos neo-tropicais. In Anexo VII EIA RIMA CESP. Ilha Solteira, pp. 47.
139. Nunes, A.L.V.; Gasparini, R.L.; Duarte, J.M.B.; Pinder, L.; and Buschinelli, M.C. 1997. Captura, contenção e manuseio. In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos*: Blastocerus, Ozotoceros* e *Mazama.* Jaboticabal, FUNEP, pp. 141–170.
140. Ozebold, J.W.; Douglas, J.R.; and Christensen, J.F. 1962. Transmission of anaplasmosis to cattle by ticks obtained from deer. American Journal of Veterinary Research 23:21–23.
141. Pachaly, J.R. 1992. Utilização da Associação de Tiletamina e Zolazepan na Contenção de Mazama gouazoubira e Mazama rufina. In 22° Congresso Brasileiro de Medicina Veterinaria. Curitiba, pp. 47.
142. Pandolfi, J.R.C.; Tamanini, M.L.F.; Anderson, J.; Thevassagayam, J.; Pinto, A.A.; and Montassier, H.J. 1998. Estudo prospectivo da infecção dos vírus da língua azul e da doença hemorrágica epizoótica dos cervídeos em ruminantes criados na Fazenda Experimental da UNESP(Campus de Jaboticabal. Virus Reviews and Research 3(suppl.):55.
143. Pandolfi, J.R.C.; Tamanini, M.L.F.; Arújo, J.P., Jr.: Duarte, J.M.B.; Anderson, J.; Thevassagayam, J.; Pinto, A.A.; and Montassier, H.J. 1998. Presença da infecção pelos vírus da lingua azul e da doença hemorrágica epizoótica dos cervídeos em uma população de vida livre de cervos-do-pantanal (*Blastocerus dichotomus*). Virus Reviews and Research 3(suppl.):55.
144. Parás Garcia, A.; Suárez, G.F.; and Peña, M.A. 1992. Serology of *Leptospira* and *Brucella* in white-tailed deer (*Odocoileus virginianus*) at Chapultepec Zoo, Mexico City. Veterinária Mexico 23:349–352.

145. Piermattei, D.L. 1969. Construction of the Thomas Splint. Veterinary Medicine Small Animal Clinicians 64:41–46.
146. Pinder, L.; and Leeuwenberg, F. 1997. Veado-Catingueiro (*Mazama gouazoubira*). In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 59–68.
147. Prestwood, A.K.; and Kellogg, F.E. 1971. Naturally occurring haemonchosis in a white-tailed deer. Journal of Wildlife Diseases 7:133–134.
148. Prestwood, A.K.; Haynes, F.A.; Eve, J.H.; and Smith, J.F. 1973. Abomasal helminths of white-tailed deer in southeastern United States, Texas and the Virgin Islands. Journal of American Veterinary Medical Association 164(6):556–561.
149. Prestwood, A.K.; Smith, J.F.; and Mahan, W.E. 1970. Geographic distribution of *Gongylonema verrucosum* and *Paramphistomum liorchis* in white-tailed deer of the south-eastern United States. Journal of Parasitology 56(1):123–127.
150. Pursglove, S.R.; Prestwood, A.K.; Nettles, V.F.; and Hayes, F.A. 1976. Intestinal nematodes of white-tailed deer in south-eastern United States. Journal of American Veterinary Medical Association 169(9):896–900.
151. Puntel, M. 1997. Seroprevalence of viral infections in llamas (*Lama glama*) in the Republic of Argentina. Revista Argentina Microbiologia 29:38–46.
152. Putman, R. 1988. The Natural History of Deer. London, Christopher Helm.
153. Redford, K.H. 1987. The pampas deer (*Ozotoceros bezoarticus*) in central Brazil. In C.M. Wemmer, ed., Biology and Management of the Cervidae. Washington, D.C., Smithsonian Institution Press, pp. 410–414.
154. Redford, K; and Eisemberg, J. 1992. Mammals of the Neotropics: The Southern Cone, Vol. 2. Chile, Argentina, Uruguay, Paraguay. Chicago, The University of Chicago Press, p. 406.
155. Rhyan, J.C.; Aune, K.; Ewalt, D.R.; Marquardt, J.; Mertins, J.W.; Payeur, J.B.; Saari, D.A; Schladweiler, P.; Sheehan, E.J.; and Worley, D. 1997. Survey of free-ranging elk from Wyoming and Montana for selected pathogens. Journal of Wildlife Diseases 33(2):290–298.
156. Ribeiro, A.M. 1919. Os veados do Brasil segundo as colecçães Rondon e de varios museus nacionaes e estrangeiros. Revista do Museu Paulista 11:209–308.
157. Rodrigues, F.H.G. 1996. Historia Natural e Biologia Comportamental do Veado Campeiro (*Ozotoceros bezoarticus*) em Cerrado do Brasil Central. Masters thesis, Universidade Estadual de Campinas.
158. Selander, R.K. 1976. Genetic variation in natural populations. In F.J. Ayala, ed., Molecular Evolution. Sunderland, Massachusetts, Sinauer, pp. 21–45.
159. Serra-Freire, N.M.; Amorim, M.; Gazêta, G.S.; Guerim, L.; and Desidério, M.H.G. 1996. Deer ixodofauna in Brazil. Revista Brasileira de Ciência Veterinária 3(2):51–54.
160. Serra-Freire, N.M. 1997. Ectoparasitos. In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 181–194.
161. Silva, R.A.M.S.; Arosemena, N.A.E.; Herrera, H.M.; Sahib, C.A.; and Ferreira, M.S.J. 1995. Outbreak of trypanosomosis due to *Trypanosoma evansi* in horses of Pantanal Mato-Grossense, Brazil. Veterinary Parasitology 60:167–171.
162. Silva, R.A.M.S.; Herrera, H.M.; Domingos, L.B.S.; Ximeses, F.A.; and Dávila, A.M.R. 1995. Pathogenesis of *Trypanosoma evansi* infection in dogs and horses: Hematological and clinical aspects. Ciência Rural 25:233–238.
163. Silva, R.A.M.S.; Barros, A.T.M.; and Herrera, H.M. 1995. Trypanosomosis outbreak due to *Trypanosoma evansi* in the Pantanal, Brazil: A preliminary approach on risk factors. Revue D'élevage et de Médecine Véterinaire des Pays Tropicus 4:315–319.
164. Silva, R.A.M.S.; Da Silva, J.A.; Schneider, R.C.; De Freitas, J.; Mesquita, D.; Mesquita, T.; Ramirez, L.; Dávila, A.M.R.; and Pereira, M.E.B. 1996. Outbreak of Trypanosomiasis due to *Trypanosoma vivax* (Ziemann, 1905) in bovines to the Pantanal, Brazil. Memorias do Instituto Oswaldo Cruz 5:561–562.
165. Smith-Flueck, J.; and W. Flueck. 1997. Relevamiento de una poblacion de huemul en la provincia de Río Negro, Argentina. Mastozoologia Neotropical 4(1):25–35.
166. Smits, J.E.G.; and Brown, R.D. 1992. Elk disease survey in western Canada and northwestern United States: The biology of deer. In Proceedings of the International Symposium on the Biology of Deer. New York, Springer Verlag, pp. 101–106.
167. Sonoda, M.C.; Gasparini, R.L.; and Catão-Dias, J.L. 1996. Miopatia de captura em cervos do pantanal (*Blastocerus dichotomus*) [Captive myopathy in marsh deer *Blastocerus dichotomus*]. Proceedings of the 15th Panamerican Congress of Veterinary Sciences. Campo Grande, Sociedade Brasileira de Medicina Veterinaria, p. 81.
168. Spotorno, A.E.; Brum ,N.; and Tomaso, M.D. 1987. Comparative cytogenetics of South American Deer. Fieldiana 39:473–483.
169. Spraker, T.R. 1993. Stress and capture myopathy in Artiodactylid. In M.E. Fowler, ed., Zoo and Wildlife Animal Medicine, Vol. 3. Current Therapy. Philadelphia, W.B. Saunders, pp. 481–488.
170. Sutmoller, P.; and Gaggero, G.E. 1965. Foot-and-mouth disease carriers. Veterinary Record 77:968–969.
171. Szabó, M.P.J.; Matushima, E.R.; Campos Pereira, M.; Werther, K.; and Duarte, J.M.B. 2000. Flea infestation outbreak at Porto Primavera's hydroelectric power station marsh deer (*Blastocerus dichotomus*) quarantine. Veterinary Parasitology. (Submitted).
172. Tamayo, R.; Schoebitz, R.; Alonso, O.; and Wenzel, J. 1985. First report of bluetongue antibody in Chile. Progress Clinics Biological Research 178:555–558.
173. Teer, J. 1994. El venado cola blanca: Historia natural y principios de manejo. In C. Vaughan and M. Rodriguez, eds., Ecologia y Manejo del Venado Cola Blanca en Mexico y Costa Rica. Serie Conservacion Biologica y Desarrollo Sostenible No. 2. Heredia, pp. 17–46.

174. Templeton, A.R.; and Read, B. 1984. Factors eliminating inbreeding depression in a captive herd of Speke's gazelle (*Gazella spekei*). Zoo Biology 3:177–199.
175. Tillmann, S.M.; and Meinecke, B. 1984. Experimental chimeras—Removal of reproductive barrier between sheep and goat. Nature 307:637–638.
176. Tirira, D. 1999. Mamiferos del Ecuador. Publicación Especial No. 2 Museo de Zoología. Quito, Centro de Biodiversidad y Ambiente PUCE, p. 392.
177. Tomas, W. 1986. Observaçães Preliminares sobre a Biologia do Cervo-do-pantanal *Blastocerus dichotomus* Illiger 1811 (Mammalia: Cervidae) no Pantanal de Pocone. Monografia Universidade Federal de Mato Grosso. Cuiabá, Instituto de Biociências.
178. Tomas, W.M.; Beccaceci, M.D.; and Pinder, L. 1997. Cervo-do-pantanal (*Blastocerus dichotomus*). In J.M.B. Duarte, ed., Biologia e Conservação de Cervídeos Sul-Americanos: *Blastocerus, Ozotoceros* e *Mazama*. Jaboticabal, FUNEP, pp. 23–40.
179. Uren, M.F. 1986. Clinical and pathological responses of sheep and cattle to experimental infection with five different viruses of the epizootic haemorrhagic disease of deer serogroup. Australian Veterinary Journal 63(6):199–201.
180. Wallace, R.S.; Bush, M.; and Montali, R.J. 1987. Deaths from exertional myopathy at the National Zoological Park from 1975 to 1985. Journal of Wildlife Diseases 23:454–462.
181. Wallach, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals. Philadelphia, W.B. Saunders,
182. Wemmer, C. 1998. Status survey and conservation action plan: Deer. Oxford, IUCN/SSC Deer Specialist Group, Information Press, p. 106.
183. Wenkoff, M.S.; and Bringans, M.J. 1991. Embryo transfer in cervids. In L.A. Renecker and R.J. Hudson, eds., Wildlife Production: Conservation and Sustainable Development. Fairbanks, Alaska, University of Alaska Press, pp. 461–463.
184. Wildt, D.E. 1989. Reproductive research in conservation biology: Priorities and avenues for support. Journal of Zoo and Wildlife Medicine 20(4):391–395.
185. Yoshinari, N.H.; Steere, A.C.; and Cossermelli, W. 1989. Revisão da borreliose de Lyme [Review of Lyme borreliosis]. Revista da Associação Médica Brasileira 35:34–38.
186. Zaugg, J.L. 1985. Bovine anaplasmosis: Transplacental transmission as it relates to stage of gestation. American Journal of Veterinary Research 46(3):570–572.

Special Topics

36
Nutrition and Nutritional Problems in Wild Animals

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INTRODUCTION

The challenges facing the wild animal nutritionist are quite different than those for domestic animal nutritionists. There are nearly 50,000 species of vertebrate animals in the world. Zoos may exhibit approximately 3000 of those species, which represents only 6% of the total.[31] The challenge this reality presents is immense because of the enormous variety of animals, dietary habits, and nutritional, dietary, and behavioral requirements that the nutritionist should know. The study of wild animal nutrition started in North America in the 1870s with the investigation of dietary habits of wild animals in relation to human welfare. Dietary habits have been the direction of the first studies and still are the main issue addressed in the reports on wild animal nutrition.

NUTRITIONAL WISDOM AND CAPTIVITY FEEDING

In natural ecosystems, animals have several strategies: feeding on or under the ground, in the water, in the air or on trees. Mineral substances and animal and vegetable tissue are part of the diet that can reach up to 80 items, as in the case of eastern rosellas (*Platycercus eximius*)[6] or be restricted to 2 items, as in the case of hyacinthine macaw (*Anodorhynchus Hyacinthinus*).[14] Survival of a given species in the wild is evidence of natural nutritional wisdom. This wisdom, however, is a result of many factors that are absent in captivity. Seasonal and geographical distribution of feeds follows plant phenology, flower and fruit parasitism by insects and larvae that are also ingested, the energy consumption in foraging and reproduction, and many other particular characteristics of each species.[32]

An example of the care that must be taken when data on natural feeding is extrapolated to captivity feeding can be seen in the hyacinthine macaw. In the wild, this bird feeds only on the endosperm of two nuts (*Acrocomia totai* and *Sheelea phalerata*). These nuts present 10.37% of crude protein and 63.67% of fat in dry matter.[7] When flying, birds use 11 to 20 times more energy than at rest.[12] If a hyacinthine macaw flies 2.5 hours a day and rests for the next 21.5 hours, during 10.42% of the day it spends at least 11 times more energy than when at rest. Therefore, energy consumption should be divided into a period of 21.5 hours (89.58% of the time) when only maintenance energy is consumed and a period of 2.5 hours when the consumption may be 11 times higher. The results of these calculations lead to energy and feed consumption that is 2.04 times higher for the wild bird than for the captive one. If feed naturally consumed presents a low protein content (10.42%), when the bird consumes two times more in the wild, it also consumes two times more protein; it can be concluded that the simple repetition of natural feeding patterns for captive animals is not always correct.

In captivity, animals are offered fruit, vegetables, fish, seeds, meat, and gramineous plants of commercial importance to humans. They are also fed commercial rations for birds, dogs, cats, horses, and other animals. There is little variation throughout the year or according to animal species; the quantity that is offered generally exceeds the ingestion rate and animals consume only what is more palatable. One cannot expect nutritional wisdom from an animal that is out of its natural ecosystem and faces unknown feed. This assertion is confirmed by frequent reports of nutritional disorders in animals in captivity. Therefore, the wild animal nutritionist must make these choices, offering feeds in such a way that a balanced diet is achieved and checking intake to be sure that animals are well fed.

Nutrition and Feeding

There is a great difference between nourishing and feeding an animal. Feeding can be defined as an empirical selection of a combination of foodstuffs, without the knowledge of their chemical composition and without a definite objective for the diet and the final nutritional content. Nourishing is a well-established nutritional concept that depends on information about the animal, its environment, and the feed to be offered. It is a fact that animals do not need feed (such as fruits and seeds), they need nutrients (such as amino acids, vitamins, and minerals). These nutrients may be obtained from this or that feed with more or less difficulty, depending on the animal dietary habits and digestive system. Generic classification based on natural dietary habits (carnivorous, grainivorous, herbivorous, insectivorous, etc.) does not define wild animal nutritional requirements, either. It only indicates the main kind of feed that is consumed preferentially in the wild, and probably the kind of feed that provides the best digestibility and palatability in captivity. Unfortunately, using only this classification in the past has led to many mistakes in the management of wild animals and produced several disorders of nutritional origin in these animals.[32]

Four aspects should be considered in order to integrate all these factors into a feeding plan: (a) dietary habits in the wild; (b) oral cavity and digestive system morphology; (c) nutritional requirements of the nearest related domestic species; and (d) enclosures and environment in which the animals are kept in captivity.[34]

NUTRIENTS AND EVALUATION OF FEEDS

The nutrients that are found in the great variety of feeds provided for wild animals are the same as for domestic animals. The reader is referred to texts on animal nutrition.

Carbohydrates

Carbohydrates are the main components of plant tissue and represent more than 70% of the chemical composition in forages and up to 85% in some grains.[26] Their functions in animal diet are to provide energy and fermentative substrate for the fore-stomachs and intestines and to produce volatile fatty acids, which are important for the maintenance of the intestinal mucous membrane, even in carnivores.

Bacterial hydrolysis of polysaccharides in the rumen produces volatile fatty acids (VFA: acetic, propionic, and butyric acids), which may account for up to 70% of the energy used by ruminants. For carnivores and granivores, which are not able to ferment a large quantity of structural carbohydrates, their nutritional value is limited. Animals that perform pre- or postgastric fermentation processes need an adequate allowance of structural carbohydrates in the diet, otherwise they may present serious digestive problems such as ruminal lactic acidosis, diarrhea, and colic.

South American primates *Allouata* sp. are vegetarians and ingest a great quantity of greens; their cecum is well developed and an important site of postgastric fermentation. Their quantitative and qualitative dietary fiber requirements are very specific. Milk presents different lactose concentrations, according to the species. The use of milk replacers or the sudden introduction of lactose in the diet of weaned animals may induce diarrhea. Some orphaned marsupials were intolerant to galactose and developed cataracts when fed cows' milk.[34]

In monogastric animals, carbohydrates are broken down by digestive enzymes to form glucose and simple sugars, which are absorbed directly and transported by portal blood. In ruminants, most of the carbohydrates undergo fermentation in the rumen and result in VFA production. VFAs are used as energy sources, stored as fat (acetic and butyric acids) or transformed into glucose (propionic acid) by gluconeogenesis. Animals presenting postgastric fermentation also produce a great quantity of VFAs from carbohydrates that escaped from enzymatic digestion in the small intestine.

Minerals

It is believed that at least 22 inorganic elements are essential for animals. They are important for structural functions (calcium and phosphorus), for acid-base balance (sodium, chlorine, and potassium) and in several enzymatic systems (zinc, copper, iron, magnesium, etc.). Disorders related to the lack or excess of these elements have been reported by many researchers, both for free and captive animals.

There are two groups of minerals: macro- and microelements. Macroelement (calcium, phosphorus, potassium, chlorine, sodium, sulphur, and magnesium) requirements are expressed as percentages and are present in the organism in higher quantities than microelements (iron, copper, iodine, magnesium, zinc, cobalt, selenium, molybdenum, chromium, fluorine, silicon, nickel, vanadium, cadmium, lead), which are expressed as parts per million (ppm) and are required in only small quantities.

Mineral requirements are not static; they are influenced by physiological status, age (requirements decrease after growth is complete), bioavailability of the nutrient in the mineral source and feed, interaction with other nutrients (e.g., production of soaps of difficult absorption in diets with a high fat content), interaction with other minerals (e.g., the classic calcium/phosphorus ratio), competition for transport proteins that carry minerals from the lumen to the intestinal cell cytoplasm, etc.

Biochemical and nutritional knowledge on minerals and their role in the organism increased sharply, mainly during this century. For deeper and more detailed information on minerals, reader should refer to Pond et al.,[26] MacDowell,[17] Robbins,[27] and Underwood.[35]

CALCIUM AND PHOSPHORUS These are the minerals that are present in the animal organism in the highest quantity. As much as 98% calcium and 80% of body phosphorus are in the bones and teeth and are responsible for their rigidity. Calcium is also important in blood clotting, muscle and nerve excitability, acid-base balance, egg shell formation, enzyme activation, and muscle contraction. Phosphorus is related to almost all aspects of animal metabolism, such as energy metabolism (it is part of ATP), muscle contraction, nervous system functions, carbohydrate, fat and amino acid metabolism, metabolite transport, and nucleic acid structure.

From a historical standpoint, the most common mineral deficiency in wild animals in captivity is related to calcium.[27] Grains, fruits, meat, and insects present low levels of calcium, and supplementation is always required. Possible sources are: leguminous forages, greens, limestone, dicalcium phosphate, bone meal, meat and bone meal, milk and milk by-products. Both calcium and phosphorus absolute and relative concentrations are important. Recommended ratios are 2:1–1:1. Calcium deficiency may occur when feeding a diet with low calcium levels or a diet with normal calcium and high phosphorus levels or a diet with normal calcium level but with low levels of vitamin D. Calcium deficiency causes nutritional secondary hyperparathyroidism, which leads to skeletal deformity in long bones (Figures 36.1 and 36.2), teeth loss and facial bone deformities, pathological fractures, and lameness. Excess calcium leads to decreases in the absorption of phosphorus, magnesium, and zinc (it may also cause zinc deficiency symptoms, when dietary levels of zinc are marginal) and to bone development disorders, such osteochondrosis, enostosis, and wobbler syndrome.

MICROELEMENTS Also called trace elements, microelements act mainly in the activation of enzymatic systems or as structural components of organic compounds. There are hundred of enzymes that depend on these elements in order to function.

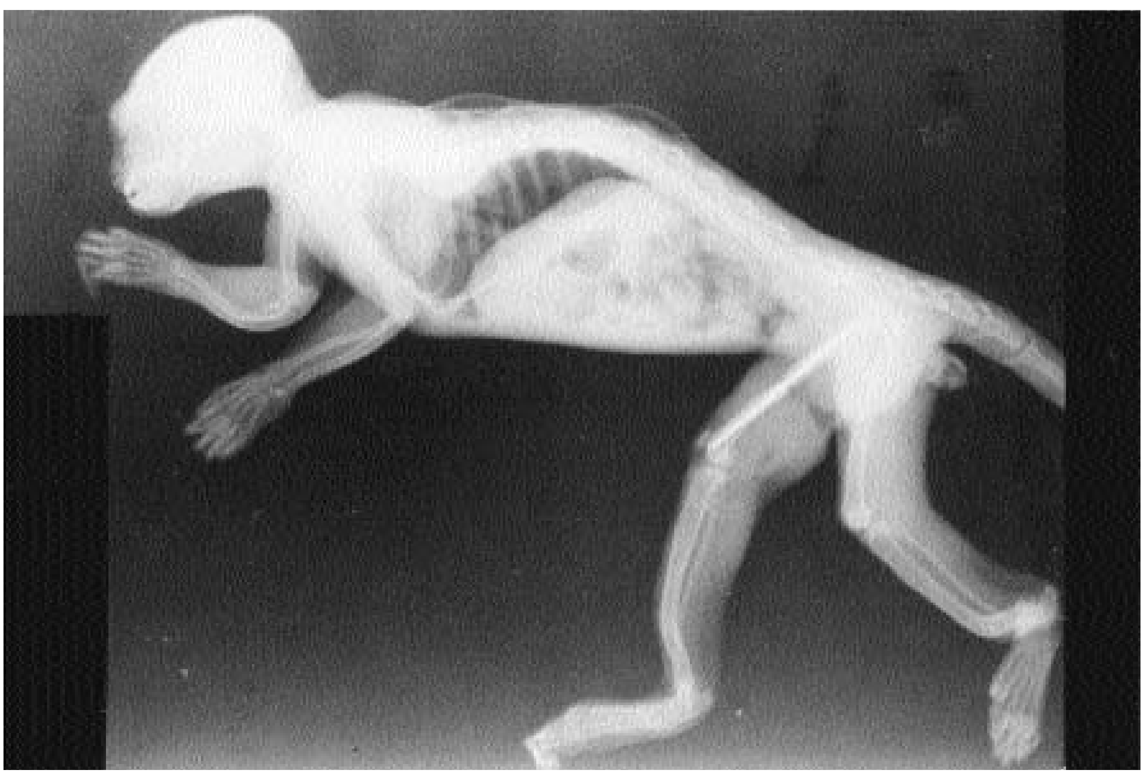

FIGURE 36.1. Common marmoset (*Callitrix jachus*) presenting nutritional secondary hyperparathyroidism. Animal was fed on fruit and meat, and calcium supplementation was not administered. Angular deformation and bowing of radium, ulna, and tibia, and a fracture in femur should be noted. (Courtesy of Karin Werther.)

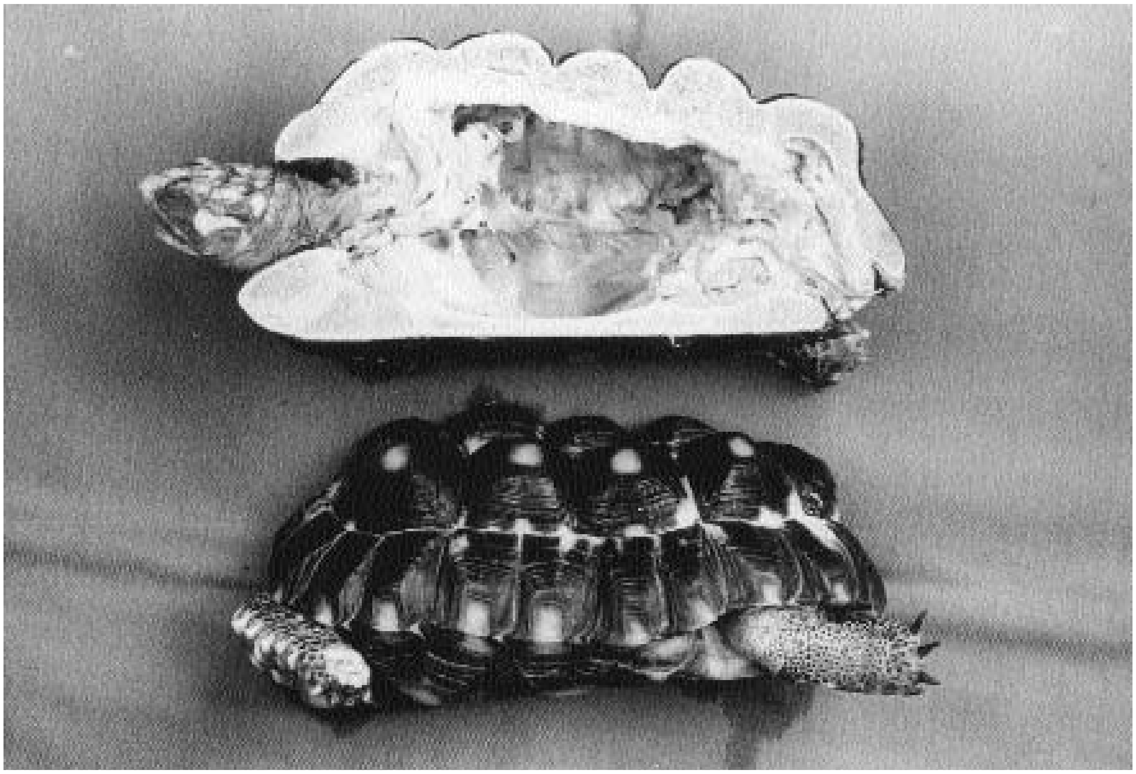

FIGURE 33.2. Red-foot tortoise (*Geochelonia carbonaria*) fed on fruit and not supplemented with calcium, presenting nutritional secondary hyperparathyroidism. Osteofibrosis, deformation, and thickening of the carapace, affecting normal position of the vertebra, should be noted. (Courtesy of Karin Werther.)

Vitamins

Vitamins A and D are generally expressed in international units per kilogram (IU/kg). Vitamin E may be either expressed in international units or in milligrams per kilogram (1 IU = 1 mg vitamin E). The other vitamins are expressed in milligrams per kilogram and are required in very small quantities, except for choline.

There are many nutritional interactions among vitamins, among vitamins and minerals, and among vitamins and the other components of the diet (carbohydrates, lipids, and proteins). A better understanding of these interactions will bring enormous progress to animal nutrition. For further information, readers may refer to Pond et al.,[26] MacDowell,[17] and Robbins.[27]

VITAMINS A AND E Vitamin A and E deficiencies are reported commonly in some species of wild animals. Vitamin A has many functions: It takes part in the production of rhodopsin (visual purple) and is responsible for normal night vision; is responsible for the structural maintenance of epithelial cells and tissues that cover the body and the cavities in the respiratory, urogenital, and digestive systems; takes part in the osteoblastic activity and is essential for bone growth and reshaping; possesses antioxidative and antimutagenic properties; and is necessary for protein synthesis. Vitamin A also induces several activities: mitosis, cell differentiation, and protein, RNA, and esteroid synthesis.

Vitamin A deficiency causes interference in rhodopsin production, which causes difficulty in adaptation to low luminosity (night blindness); squamous metaplasia, affecting mainly the skin, intestinal and conjunctiva mucous membranes, respiratory epithelium, and salivary glands; xerophtalmia (drying of the conjunctiva, corneal neovascularization, opacity, and infection); suppression in local defense mechanisms (squamous metaplasia and keratinization); abortion; embryonic malformation; testicular degeneration; increase in osteoblastic activity and change in the chondroitin sulfate and mucopolysaccharide metabolism, which interferes with bone growth and reshaping and causes partial occlusion of the spinal cord; increase in cranial pressure, and optical nerve compression, leading to ataxia, unsteady gait, convulsions, and blindness in young animals.

Many of these signs are reported in wild animals.[2] Birds, such as psittacines, fed grains and nuts, generally present vitamin A deficiency due to the limited quantity of carotenoids in these foodstuffs.[33] Wild carnivores fed only muscle supplemented with calcium also present this deficiency, because muscle supplies less than 40%

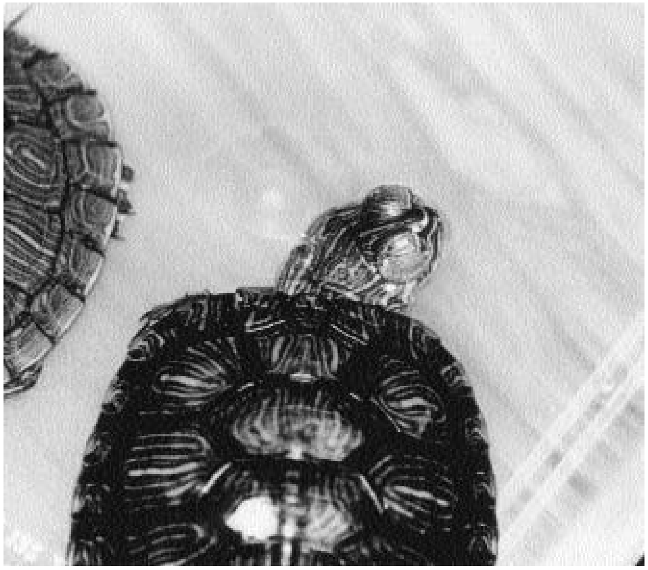

FIGURE 36.3. Red-eared tortoise (*Trachemys dorbignii*) fed only meat, presenting hypovitaminosis A. Edema of the eye adnexal structures should be noted. (Courtesy of Karin Werther.)

of the minimum requirements for domestic cats. Aquatic turtle offspring fed on meat and dehydrated shrimp quickly show signs of hypovitaminosis A: edema of the adnexal structures of the eye, anorexia, and emaciation (Figure 36.3).

VITAMIN E Vitamin E deficiency leads to reproductive disorders, such as embryonic degeneration, ovarian failure, and irreversible inhibition of spermatogenesis. Cell permeability disorders affect the brain, kidneys, liver, muscle, and blood capillaries leading to liver necrosis, hemolysis, kidney degeneration, steatitis, encephalomalacia, and exudative diathesis in birds. Nutritional muscular dystrophy is one important disease caused by vitamin E deficiency: White bands are seen in skeletal and cardiac muscles, due to cell and tissue degeneration. Clinical signs include ataxia, prostration, hemoglobinuria after exertion, high levels of serum creatine phosphokinase, etc. Myocardium degeneration may cause sudden death. Except for hemolysis and encephalomalacia, all other signs may also be prevented with selenium supplementation. Levels of polyunsaturated fatty acids are directly related to vitamin E: the higher their content in the diet, the bigger the vitamin E requirements.

Dierenfeld[10] reviews vitamin E deficiency in zoo reptiles, birds, and ungulates. Many authors reported steatitis, fat necrosis, and muscular degeneration in reptiles (crocodilians, snakes, and lizards); the problems are mainly related to diets with high levels of polyunsaturated fatty acids, fish, or feeds that underwent oxidative rancidity. In birds, disorder reports deal primarily with piscivorous species and raptors. Related to ungulates, deficiencies have been reported in elephants, nyalas, deer, etc.

VITAMIN D The biological activity of these sterols is interwoven with the metabolism of calcium and phosphorus. Ergosterol, a compound of vegetable origin, under the effect of ultraviolet (UV) radiation (sunlight) becomes ergocalciferol (or vitamin D_2). In animals, cholesterol present in the skin is converted to 7-dehydrocholesterol and when irradiated by UV light becomes cholecalciferol (or vitamin D_3). Because dogs do not possess ability to sufficiently synthesize vitamin D from the skin, it is advisable to supplement the diet of carnivores; New World primates, birds, reptiles, and amphibians use vitamin D_3 much more efficiently than D_2. After skin synthesis or absorption from feed, vitamin D is carried to the liver where it is hydroxylated to produce 25-hydroxyvitamin D. For the vitamin to be active, further hydroxylation is needed. The biologically active molecule 1,25-dihydroxyvitamin D is formed in the kidneys, under the stimulus of parathyroid hormone.

The action of vitamin D is related to body retention and increase in plasma levels of calcium and phosphorus; it stimulates an increase of both renal reabsorption and intestinal absorption of these minerals. In the intestines, vitamin D stimulates transcription and synthesis of calcium-carrier protein, which is necessary for the absorption of this mineral. In the bones, it has a "permissive effect" for parathyroid hormone and bone mineralization; it also stimulates the production of organic bone matrix. Vitamin D deficiency leads to abnormal bone development and calcification, which produces rickets in growing animals and osteoporosis in adults. Some lizard species, such as the iguana (*Iguana iguana*) may require not only adequate dietary vitamin D_3, but exposure to UV light as well.[2]

NUTRITIONAL REQUIREMENTS

To keep animals healthy, nutritionists must supply them with adequate quantities of more than 45 nutrients. Feeding is also responsible for the integrity of the digestive tract, cleaning of the teeth, intestine functioning, and behavioral improvement.

Balance is a fundamental concept when dealing with nutrition. Every nutrient must be supplied in adequate quantities and according to the energy level of the diet. It may be said, figuratively, that health status is determined by the nutrient in the lowest proportional concentration: that is, the first limiting nutrient of the diet.

The estimated nutrient requirements for most species of domestic and laboratory animals have been published by the National Research Council of the United States National Academy of Sciences. These estimates represent the knowledge produced, reviewed, and updated periodically all over the world about nutrition of these species. In other countries, other institutions also collect and publish information related to the subject. These data are important references for the establishment of animals' nutritional programs.

The correct use of these databases by the wild animal nutritionist is connected to the understanding of how data on nutritional requirements are produced, the various interactions among feeds, nutrients and their processing, and the differences related to the objectives established for farm animals (i.e., precocity, performance, and productivity) and for wild animals (i.e., health, longevity, welfare, and adequate reproduction).

Experiments on nutritional requirements are normally performed using "purified diets," which present a small number of industrially purified ingredients (starch, cellulose, isolated soy proteins, casein), or chemically defined diets, made up of pure nutrients such as individual amino acids, minerals, vitamins, saccharose, dextrose, etc. Animals are kept under tightly controlled environment and management conditions (sometimes controlled temperature, humidity, and luminosity) and animals are totally free of pathogens. Biological responses of importance in animal production are measured, such as growth rate, carcass composition, blood and tissue metabolites, egg production rate, etc.

In normal conditions, animals are submitted to stress factors, such as heat, rain, ecto- and endoparasites, competition with other members of the group, exercise, etc., which lead to nutritional requirements that, in some occasions, may be higher than the minimum established in laboratory conditions. The last few decades have witnessed a marked development of immunology information making clear the concepts that minimum requirements must be reviewed, because minimal allowance for adequate growth or good production rates is not necessarily the minimum required to optimize immunological response.[8]

In day-to-day diet formulations, feeds that are chosen present, together with essential nutrients, detrimental substances that are called antinutritional factors. Some examples are: tannins, alkaloids, trypsin inhibitors, oxalic acid (which binds strongly to calcium) and phytic acid (which binds to calcium, phosphorus, magnesium, iron, and zinc, and decreases greatly the availability of these minerals for monogastric animals). The interaction among nutrients also affects their utilization, such as molybdenum and copper; calcium and zinc; calcium, phosphorus, and vitamin D; polyunsaturated fatty acids and vitamin E; choline, biotin, and manganese in bird perosis; sodium, potassium, and chlorine in acid-base balance; etc. All these factors determine a great difference between crude nutrients and bioavailable ones, depending on the composition and ingredients of the diet. Concentration of specific nutrients must be carefully analyzed, even if the minimum quantities are respected, otherwise nutritional disorders may occur. Some tables for nutritional programs, like the ones of the Association of American Feed Control Officials for dogs and cats, take these factors into account and provide safety margins for nutritional programs.

There is a great difference in the feeding of farm animals and wild or domestic animals. Profit maximization is based on nutritional schemes that produce maximum growth or productivity rates with minimum feeding costs. Sometimes, minimal allowance for maximum performance is excess for wild animals, which are not genetically selected and from which all that is expected is health and longevity. Metabolic disorders, such as ascites and hepatic degeneration, bone development problems, such as bowing of the legs and tendon deviation, sudden death syndrome, and other complications show that achieving the maximum genetic potential of an animal is not directly related to making it healthy. For example, the ration for laying hens that produce 300 eggs a year requires a very high calcium level (3.5–3.75%), because each eggshell is composed of nearly 6 g of the mineral. This ration is not appropriate for any other bird or animal because wild birds seldom lay more than 20 or 40 eggs a year, even if oviposition occurs twice during the same year. This excess of calcium will bring several problems to the animals, as pointed out earlier. Studies on protein requirements for the growth of *Cairina moschata* verified that maximum growth rates are achieved with 18% crude protein in the ration, which is less than the National Research Council[23] recommendation for ducks (*Anas platyrhynchos*), 22%.[7] Despite the fact that both species present similar weights when animals are adults, at 21 days of age ducks weight 1330 g, and *Cairina moschata* weight 288 g, on average, thus representing a slower growth pattern for the latter. Finally, it must be emphasized that recommended requirements are related to the efficiency in the utilization of foodstuffs and absorption of nutrients. Knowledge of digestibility of feeds by different wild species is lacking.

Logic is the key word for determining the quantity of each nutrient that will be offered at different development stages; knowledge of nutrition and physiology and a focus on what is the objective when feeding one specific animal are the most important requirements in the formulation of diets.

Tables 36.1, 36.2, and 36.3 present requirements for growth of several species of domestic and laboratory

TABLE 36.1. Nutrient requirements for growth of some domestic and laboratory animals

Nutrient	Units	Rats[d]	Dog[e]	Cat[f]	Nonhuman Primates[i]	Swine[j]
Protein	%	16.50	22	30	16.30	19.80
Arginine	%	0.47	0.62	1.25		0.44
Histidine	%	0.31	0.22	0.31		0.28
Isoleucine	%	0.68	0.45	0.52		0.58
Leucine	%	1.18	0.72	1.25		0.77
Lysine	%	1.01	0.77	1.20		1.05
Meth + Cyst[a]	%	1.08	0.53	1.10		0.53
Phen + Tyr[b]	%	1.12	0.89	0.88		0.85
Treonine	%	0.68	0.58	0.73		0.62
Tryptophan	%	0.22	0.20	0.25		0.15
Valine	%	0.81	0.48	0.62		0.62
Taurine	%	NR	NR	0.1–0.2[g]		NR
Fat	%	5.5	8	9		ND
Linoleic acid	%	0.66	1	0.50		0.11
Arachidonic acid	%	NR	NR	0.02		NR
Minerals						
Calcium	%	0.55	1	1	0.54	0.77
Phosphorus	%	0.33	0.80	0.80	0.43	0.66
Potassium	%	0.40	0.60	0.60		0.29
Sodium	%	0.06	0.30	0.20		0.11
Chloride	%	0.06	0.45	0.30		0.09
Magnesium	%	0.06	0.04	0.08	0.16	0.05
Iron	ppm	38.50	80	80.0	196	88
Copper	ppm	5.50	7.30	5.0–15.0[h]		5.50
Manganese	ppm	11	5	7.5		3.30
Zinc	ppm	13.20	120	75	11	88
Iodine	ppm	0.17	1.50	0.35	2.20	0.15
Selenium	ppm	0.17	0.11	0.10		0.28
Vitamins						
Vitamin A	IU/kg	2,530	5,000	9,000	10,900	1,925
Vitamin D	IU/kg	1,100	500	750	2,170	220
Vitamin E	IU/kg	29.70	50	30	54	12.10
Vitamin K	mg/kg	1.10	ND	0.10		0.55
Thiamin	mg/kg	4.40	1	5		1.10
Riboflavin	mg/kg	3.30	2.20	4	5.40	3.30
Pantoth. Acid[c]	mg/kg	11	10	5		9.90
Niacin	mg/kg	16.50	11.40	60	54	13.75
Pyridoxine	mg/kg	6.60	1	4	2.70	1.65
Folic acid	mg/kg	1.10	0.18	0.80	0.22	0.33
Biotin	mg/kg	0.22	ND	0.07		0.06
Vitamin B_{12}	µg/kg	55	22	20		16.50
Choline	mg/kg	825	1,200	2,400		440
Ascorbic acid	mg/kg	NR	NR	NR	109	NR

[a] Methionine plus cystine.
[b] Phenylalanine plus tyrosine.
[c] Pantothenic acid.
[d] Nutrient requirements for growth, diets containing 3.8–4.1 kcal ME/g, margin of safety is not included.[24]
[e] Nutrient requirements for growth, diets containing 3.5 kcal/g.[3]
[f] Nutrient requirements for growth, diets containing 4.0 kcal/g.[3]
[g] Extruded or canned, respectively.
[h] Canned or extruded, respectively.
[i] Nutrient requirements for all stages, diets containing 4 kcal EM/g.[19]
[j] Nutrient requirements for growth (10–12 kg live weight), diets containing 3.25 kcal EM/g. Dietary level of calcium higher than 1% may be toxic in the presence of limited zinc.[21]
Requirements on dry matter basis; NR, not required; ND, not determined.

TABLE 36.2. Nutrient requirements for the growth of some ruminant, domestic, and laboratory animals

Nutrient	Units	Rabbits[f]	Guinea Pig[g]	Browsers[h]	Grazers[j]	Horse[k]
Protein	%	17.60	19.80	18.70	16.5	14.50
Arginine	%	0.66	1.32			
Histidine	%	0.33	0.40			
Isoleucine	%	0.66	0.66			
Leucine	%	1.21	1.19			
Lysine	%	0.72	0.92	0.88	0.77	0.61
Meth + Cyst [a]	%	0.66	0.70			
Phen + Tyr [b]	%	1.21	1.19			
Treonine	%	0.66	0.66			
Tryptophan	%	0.22	0.20			
Valine	%	0.77	0.92			
Fiber						
Crude fiber	%	11	15			
ADF [c]	%			14.3–18.7 [i]	23.1–28.6 [i]	
Fat	%	2.20	ND	3.30	3.30	
Linoleic acid	%	ND	0.44	1.10	1.10	
Minerals [d]	%			8.80	11.75	
Calcium	%	0.44	0.88	0.72–1.10 [i]	0.95–1.32 [i]	0.56
Phosphorus	%	0.24	0.44	0.72	0.83	0.31
Potassium	%	0.66	0.55	1.32	1.65	0.30
Sodium	%	0.22	0.06	0.28	0.55	0.10
Chloride	%	0.33	0.06			
Magnesium	%	0.04	0.11	0.22	0.28	0.08
Sulfur	%			0.22	0.22	0.15
Iron	ppm	ND	55	165	220	40
Copper	ppm	3.30	6.60	22	22	10
Manganese	ppm	9.35	44	99	99	40
Zinc	ppm	ND	22	132	132	40
Iodine	ppm	0.22	0.17	0.88	0.88	0.1–0.6
cobalt	ppm			0.33	0.33	0.10
Selenium	ppm	ND	0.17	0.44	0.44	0.10
Vitamins						
Vitamin A	IU/kg	638	21,960	5,500	5,500	2,000
Vitamin D	IU/kg	ND	1,100	1,320	1,320	800
Vitamin E	IU/kg	44	44	330	300	80
Vitamin K	mg/kg	ND	5.50			
Thiamin	mg/kg	ND	2.20	5.50	5.50	3
Riboflavin	mg/kg	ND	3.30	9.90	9.90	2
Pantoth. Acid [e]	mg/kg	ND	22	33	33	
Niacin	mg/kg	198	11	55	55	
Pyridoxine	mg/kg	42.9	2.75			
Folic acid	mg/kg	ND	4.95			
Biotin	mg/kg	ND	0.22	0.28	0.28	
Vitamin B_{12}	μg/kg	ND	—	22	22	
Choline	mg/kg	1,320	1,980	1,650	1,650	

[a] Methionine plus cystine.
[b] Phenylalanine plus tyrosine.
[c] Acid detergent fiber.
[d] Maximum ash.
[e] Pantothenic acid.
[f] Nutrient requirements for growth, diets containing 2.75 kcal DE/g, margin of safety is not included.[18]
[g] Nutrient requirements for growth, diets containing 2.8–3.5 kcal ME/g, margin of safety is not included.[24]
[h] Nutrient specifications for a complete pellet designed for herbivorous concentrate selectors.[31]
[i] Minimum and maximum, respectively.
[j] Nutrient specifications for a complete pellet designed for bulk or roughage eaters.[31]
[k] Nutrient requirements for growth (weaning, 6 months, moderate growth), margin of safety is not included. Minimum of 30% hay in the diet.[22]
Requirements on dry matter basis; ND, not determined.

TABLE 36.3. Nutrient requirements for growth of some domestic birds

Nutrient	Units	Chickens[e]	Turkeys[g]	Ducks[h]	Ring-necked Pheasants[i]	Japanese Quail[j]
Protein	%	19.80	30.80	24.20	30.80	26.40
Arginine	%	1.10	1.76	1.21	ND	1.38
Glycine + Serine		0.77	1.10	ND	1.98	1.27
Histidine	%	0.29	0.64	ND	ND	0.40
Isoleucine	%	0.66	1.21	0.69	ND	1.08
Leucine	%	1.21	2.09	1.39	ND	1.86
Lysine	%	0.94	1.76	0.99	1.65	1.43
Methionine		0.33	0.61	0.44	0.55	0.55
Meth + Cyst [a]	%	0.68	1.16	0.77	1.10	0.83
Phenilalanine		0.59	1.10	ND	ND	1.06
Phen + Tyr [b]	%	1.10	1.98	ND	ND	1.98
Treonine	%	0.75	1.10	ND	ND	1.12
Tryptophan	%	0.19	0.29	0.25	ND	0.24
Valine	%	0.68	1.32	0.86	ND	1.05
Fat						
Linoleic acid	%	1.10	1.10	ND	1.10	1.10
Minerals						
Calcium	%	0.99 [f]	1.32	0.72	1.10	0.88
Nonph Phosph [c]	%	0.44	0.66	0.44	0.61	0.33
Potassium	%	0.28	0.77	ND	ND	0.44
Sodium	%	0.17	0.19	0.17	0.17	0.17
Chloride	%	0.17	0.17	0.13	0.12	0.15
Magnesium	%	0.07	0.06	0.06	ND	0.03
Manganese	ppm	66	66	55	77	66
Zinc	ppm	44	77	66	66	27.50
Iron	ppm	88	88	ND	ND	132
Copper	ppm	5.50	8.80	ND	ND	5.50
Iodine	ppm	0.39	0.44	ND	ND	0.33
Selenium	ppm	0.17	0.22	0.22	ND	0.22
Vitamins						
Vitamin A	IU/kg	1,650	5,500	2,750	ND	1,815
Vitamin D_3	IU/kg	220	1,210	440	ND	825
Vitamin E	IU/kg	11	13.20	11	ND	13.20
Vitamin K	mg/kg	0.55	1.93	ND	ND	1.10
Vitamin B_{12}	μg/kg	9.90	3.30	ND	ND	3.30
Biotin	mg/kg	0.17	0.28	ND	ND	0.33
Choline	mg/kg	1,430	1,760	ND	1,573	2,200
Folacin	mg/kg	0.61	1.10	ND	ND	1.10
Niacin	mg/kg	29.7	66	55	77	44
Pantoth. Acid [d]	mg/kg	11	11	12.10	11	11
Pyridoxine	mg/kg	3.30	4.95	2.75	ND	3.30
Riboflavin	mg/kg	3.96	4.40	4.40	3.74	4.40
Thiamin	mg/kg	1.10	2.20	ND	ND	2.20

[a] Methionine plus cystine.
[b] Phenylalanine plus tyrosine.
[c] Nonphytate phosphorus.
[d] Pantothenic acid.
[e] Nutrient requirements for growth of leghorn chickens (0–6 weeks), diets containing 3.13 kcal MEn/g, margin of safety not included.[23]
[f] Dietary level higher than1.2% of calcium are toxic.
[g] Nutrient requirements for growth (0–4 weeks), diets containing 3.08 kcal MEn/g, margin of safety not included.[23]
[h] Nutrient requirements for growth (0–2 weeks), diets containing 3.19 kcal MEn/g, margin of safety not included.[23]
[i] Nutrient requirements for starting and growth, diets containing 3.19 kcal MEn/g, margin of safety not included.[23]
Requirements on dry matter basis; ND, not determined.

animals. Lactation is the stage at which requirements are highest; it is followed by growth, pregnancy, old age, and, finally, maintenance. For example, 5- or 10-kg swine offspring require 20% crude protein (CP) in the diet, whereas animals of 50 to 110 kg require only 13%. Chicks of 0 to 2 weeks of age require 23% CP, whereas cockerels require 12%. Because the majority of zoo animals are out-of-reproduction adults, nutritional level of their diets is expected to be lower. However, this kind of information is not available because the category "adult animals out of reproduction" is not part of the productive system.

ENERGY REQUIREMENTS

Evaluation and balance of diets can be performed only when energy requirements of the animal and metabolizable energy of feeds are known. As a rule, satiety is controlled by energy requirements, and nutrient intake is ruled by the bioavailable energy/nutrient ratio, which is inversely proportional to metabolizable energy in the feed[30] (that is, the more energy the diet supplies, the less the intake will be). Therefore, the other nutrients should be supplied in proportionally greater quantity.

Energy requirements of an animal are directly related to body surface[5]—which is difficult to measure—and not to body weight. Mathematical models were developed to establish a correlation between body surface and weight, which is much easier to measure. This correlation is called metabolic weight (MW). The most used of the constants defined in the mathematical models is $MW = (LW)^{0.75}$, where LW is live weight in kilograms.

Maintenance metabolizable energy (MME) is the daily requirement for a moderately active animal in a thermoneutral environment. It is calculated multiplying MW by K, as follows: $MME = K(LW)^{0.75}$.

Each group of animals presents a specific value for K. Desert rodents and carnivores, for example, have lower values, whereas sea mammals, mustelides, and lagomorphs have higher values. Individual variation is important, and research trials show that variation coefficients reach up to 25%. Therefore, results found using the formula are only starting points; animal response in weight gain or loss should be carefully evaluated. Table 36.4 presents MME formulas for some groups of animals.

The requirements above are related to the maintenance of adult animals. Sick animals or those in other physiological status require more energy, and some adjustment factors should be used. Table 36.5 presents some suggested factors.

BASIC PRINCIPLES FOR DIET FORMULATION

Ration formulation is the route for nutritionists to achieve their objectives. Formulation is use of foodstuffs in definite quantities in such a way that balance and proportion of essential nutrients are respected and animals' requirements are totally met. These are some of the basic principles for diet formulation: no isolated

TABLE 36.4. Formulas for maintenance metabolizable energy (MME) of some groups of animals

Animals	MME (kcal/dia)	Animals	MME (kcal/dia)
Passerine birds[a]	$200–250(LW)^{0.75}$	Reptiles (all)[b]	$32\ (LW)^{0.77}$
Non-passerine birds	$130–160(LW)^{0.75}$	Lizards	$28\ (LW)^{0.83}$
Placental mammals	$140\ (LW)^{0.75}$	Lizards	$48\ (LW)^{0.82}$
Marsupial mammals	$100\ (LW)^{0.75}$	Snakes	$32\ (LW)^{0.76}$
		Turtles	$32\ (LW)^{0.86}$

[a] The classification in passarine and non-passarine birds does not seem to demonstrate adequately the energy requirements of those birds. For example, the K value for the budgerigar (*Melopsittacus undulatus*) is 200.[13]

[b] Reptiles and amphibians are poikiloterms. They increase or decrease basal metabolism according to the environmental temperature. Therefore, their energy requirements are variable.

TABLE 36.5. MME adjustment factors, according to physiological and health status

Status	MME (kcal/dia)	Status	MME (kcal/dia)
Inactive animal	MME × (0.7–0.9)	Septicemia	MME × (1.2–2.0)
Postsurgical period	MME × 1.2	Pregnancy	MME × (1.2–1.3)
Severe trauma	MME × (1.2–2.0)	Lactation	MME × (2.0–4.0)
Burning	MME × (1.2–2.0)	Growth	MME × (1.5–3.0)

feed supplies all essential nutrients for every life stage of the animals; when animals are out of their natural environment, they are not able to balance their own diets; animals present dietary preferences, and, when in a group, they compete for feed. Some strategies must be developed in order to overcome these difficulties, otherwise feed will not be ingested in the same proportion that it was offered.

Simple calculation for ration formulation involves only basic mathematical knowledge; although anyone can calculate the combination and balance of ingredients, the result will not always be a balanced and complete diet.[28] There are at least six phases in diet formulation:

1. Adequate knowledge of feed to be used, that is, chemical composition, palatability, digestibility, antinutritional factors, preservation, and storage techniques;
2. Determination of actual intake. How much is offered and how much is not ingested should be quantified. Although it is difficult to determine the exact intake, mainly when animals are kept in groups inside the enclosures, this information is essential for diet formulation. To know the real importance of every feed that comprises the diet intake must be always expressed on a dry matter basis;
3. Knowledge of the nutritional composition of the diet actually ingested. Using data on intake and chemical composition of feeds in ration formulation software programs or computer workbooks, it is possible to determine the exact nutritional composition of the diet;
4. Determination of nutritional patterns for the animal species and physiological status. This may be achieved using data on nutritional requirements of domestic animals, as was pointed out earlier. Thus, tapir nutritional requirements may be compared with those of horses, those of peccaries to those of swine. Brazilian deer, which include red brocket deer (*Mazama americana*), grey brocket deer (*Mazama gouazoubira*), Brazilian dwarf (*Mazama nana*), small red brocket deer (*Mazama bororo*), pampas deer (*Ozotoceros bezoaticus*), and marsh deer (*Blastocerus dichotomus*) are all grazers;[11] nutrient requirements of poultry may be applied to a wide variety of birds. Nutritional requirements of psittacines will be dealt with in specific chapters in this book.
5. Review of the information gathered and, if necessary, reformulation of the formula;
6. Determination of the mechanisms that ensure correct and proportional intake of what is offered.

Diet Formulation

There are many different diet formulation software programs available nowadays, and although they are specific for domestic animals, they may be used to formulate diets for wild animals.

The following example is related to a diet formulated in a software program used for domestic animals. All calculations are based on dry matter.

Example:
Animal: Capuchin monkey (***Cebus apela***)
Life stage: growth
Nutritional requirements: nonhuman primates
Final report on diet composition:

Feed	Quantity (%)	Cost per Unit	Total Cost	Minimum Quantity (%)	Maximum Quantity (%)
Dog ration (27% CP)	47	0	0	30	47
Banana	22	0	0	15	25
Papaya	16	0	0	10	16
Tomato	10	0	0	5	10
Cabbage	5	0	0	2	5
Total	100	0	0		

Meeting of nutritional requirements:

Nutrient	Unit	Meeting of Nutritional Requirements	Minimum Quantity	Maximum Quantity
Calcium	x%	1.06	1.0	1.20
Ash	x%	8.51	—	—
Metabolizable energy	kcal/kg	3632	3000	9999.9
Crude fiber	%	5.49	0	9999.9
Total phosphorus	%	0.61	0.60	9999.9
Ether extract	%	5.45	0	9.0
Crude protein	%	19.06	18.0	9999.9

After diet is defined, it must be estimated how much of it will be offered. To do that, metabolizable energy (ME) of feed (3632 kcal ME/kg, in the example) and MME must be considered. The monkey weighs 2 kg live weight and is in the growth stage:

$$\text{MME} = [140\,(2.0)^{0.75}\,] \times 2 = [140\,(1.68)] \times 2 = 471 \text{ kcal ME/day.}$$

To determine the quantity of feed that should be offered, MME is divided by feed ME.

$$\text{Feed quantity per day} = 471 \div 3632 = 0.1295 \text{ kg or } 129.5 \text{ g per day.}$$

The next step is to transform the percentage of each foodstuff in the 129.5 g to be offered. The quantity then defined is transformed from dry matter to original matter (OM) using a rule of proportion, and diet formulation is complete.

The last step is to check the consumption and to make sure that intake occurs in the quantity that was determined for the animal.

REFERENCES

1. Ackerman, L. 1995. Reviewing the biochemical properties of fatty acids. Veterinary Medicine 90(2):1138–1148
2. Allen, M.E.; and Montali, R.J. 1995. Nutrition and disease in zoo animals. Verh. Ber. Erkrankungen Der Zootiere 37:215–232.
3. Association of American Feed Control Officials. 1995. Official Methods of Analysis, 16th Ed. Arlington, AOAC International.
4. Borges, F.M.O.; and Nunes, I.J. 1998. Nutrição e manejo alimentar de cães na saúde e na doença [Nutrition and alimentary handling of dogs in the health and in the disease]. Cadernos Técnicos da Escola de Veterinária da Universidade Federal de Minas Gerais no. 23:103.
5. Campbell, K.L. 1995. Fatty acid supplements in dermatology. In Proceedings of the 13th Annual Veterinary Medical Forum, American College of Veterinary Internal Medicine.
6. Cannon, C.E. 1980. The diet of Eastern and Pale-headed Rosellas. Emu 81(2):101–110.
7. Carciofi, A.C.; and Prada, F. 1999. Unpublished data.
8. Chandra, R.K.; and Kumari, D. 1994. Nutrition and immunity: An overview. Journal of Nutrition 124(85): 1433S–1435S.
9. Coimbra-Filho, A.F.; and Rocha, N.C. 1973. Aspectos do processo nutricional de animais selvagens em cativeiro [Nutritional feature of wild animals in captivity]. Brasil Florestal 4(14):19–35.
10. Dierenfeld, E.S. 1989. Vitamin E deficiency in zoo reptiles, birds, and ungulates. Journal of Zoo and Wildlife Medicine 20(1):3–11.
11. Duarte, J.M.D. 1999. Personal commununication,
12. Earle, K.E. 1993. Calculations of energy requirements of dogs, cats and small psittacine birds. Journal of Small Animal Practice 34(4):163–173.
13. Earle, K.E.; and Clarke, N.R. 1991. The nutrition of the Budgerigar (*Melopsittacus undulatus*). Journal of Nutrition 121(11S):186S–192S.
14. Guedes, N.M.R. 1993. Biologia reproductiva da Arara-azul (*Anodorhynchus hyacinthinus*) no Pantanal, MS, Brasil [Reproductive biology of Hyacinthe macaw (*Anodorhynchus hyacinthinus*) in Pantanal, MS, Brazil]. Masters thesis, Escola Superior de Agricultura “Luiz de Queiroz”, Universidade de São Paulo.
15. Maynard, L.A.; Loosli, J.K.; Hintz, H.F.; and Warner, R.G. 1979. Animal Nutrition, 7th Ed. New York, McGraw-Hill, p. 602.
16. McDowell, L.R. 1989. Vitamins in animal nutrition. San Diego, California, Academic Press, p. 486.
17. McDowell, L.R. 1992 Minerals in animal and human nutrition. San Diego, California, Academic Press, p. 524.
18. National Research Council. 1977. Nutrient Requirements of Rabbits, 2nd Ed. Washington, D.C., National Academy of Science.
19. National Research Council. 1978. Nutrient Requirements of Nonhuman Primates. Washington, D.C., National Academy of Science.
20. National Research Council. 1985. Nutrient Requirements of Dogs. Washington, D.C., National Academy of Science.
21. National Research Council. 1988. Nutrient Requirements of Swine, 9th Ed. Washington, D.C., National Academy of Science.
22. National Research Council. 1989. Nutrient Requirements of Horses, 5th Ed. Washington, D.C., National Academy Press.
23. National Research Council. 1994. Nutrient Requirements of Poultry, 9th Ed. Washington, D.C., National Academy Press.
24. National Research Council. 1995. Nutrient Requirements of Laboratory Animals, 4th Ed. Washington, D.C., National Academy Press.
25. Nunes, I.J. 1995. Nutrição Animal Básica [Basic animal nutrition]. Minas Gerais, Veterinary School Press, Federal University of Minas Gerais, p. 334.
26. Pond, W.G.; Church, D.C.; and Pond, K.R. 1995. Basic Animal Nutrition and Feeding, 4th Ed. New York, John Wiley & Sons, p. 615.
27. Robbins, C.T. 1993. Wildlife Feeding and Nutrition, 2nd Ed. New York, Academic Press, p. 352.
28. Saad, C.E.P. 1998. Formulação de dietas e nutrição de animais silvestres em cativeiro [Formulation of diets and nutrition of wild animals in captivity]. In Congresso da Sociedade de Zoológicos do Brasil. Salvador, Socidade de Zológicos do Brasil, p. 182.
29. Saad, C.E.P. 1992. Degradabilidade In Situ dos Farelos de Soja e de Algodão e das Farinhas de Carne e Osso e de Peixe sob Duas Relações Concentrado: Vovumoso em Bovinos [In situ degradability of soybean meal, cottonseed meal, meat and bone meal and fish meal under two different concentrate: Roughage relationship in bovines]. Masters thesis, Escola de Veterinária, Universidade Federal de Minas Gerais.
30. Sibbald, I.R. 1982. Measurement of bioavailable energy in poultry feedingstuffs: A review. Canadian Journal of Animal Science 62(4):983–1048.
31. Ullrey, D.E. 1995. Wild animals. In W.G. Pond, D.C. Church, and K.R. Pond, eds., Basic Animal Nutrition and Feeding, 4th Ed. New York, John Wiley & Sons, pp. 565–574.
32. Ullrey, D.E. 1989. Nutritional wisdom. Journal of Zoo and Wildlife Medicine 20(1):1–2.
33. Ullrey, D.E.; Allen, M.E.; and Baer, D.J. 1991. Formulated diets versus seed mixtures for psittacines. Journal of Nutrition 121(11S):S193–S205.
34. Ullrey, D.E.; and Allen, M.E. 1986. Principles of zoo mammal nutrition. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 516–532.
35. Underwood, E.J. 1981. The Mineral Nutrition of Livestock. Slough, U.K., Commonwealth Agricultural Bureaux.
36. Van Soest, P.J. 1982. Nutritional Ecology of the Ruminant, 2nd Ed. Ithaca, New York, Cornell University Press.

37
Ophthalmology

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INTRODUCTION

Ophthalmology has become an important facet of managing the health of wild animals, just as it is for domestic animals. The same sophisticated examination techniques and treatment protocols are used.

It is important to learn about the special features of ocular anatomy, physiology, and vision of every group of wild animals. The transition to the study of wild animal ophthalmology is not complicated for the veterinary ophthalmologist, because there are more similarities than differences among animal eyes. Additionally, the causes of ocular lesions in wild animals are often identical to those responsible for inducing ophthalmic disease in domestic animals. Therefore, the clinical diagnosis and medical or surgical management of eye and/or periorbital diseases in wild animals change little from those employed in the dog or cat. The only difference sometimes is choosing an ideal protocol, taking into consideration the form in which the drug is supplied, the route of administration, and the frequency of administration. Since wild animals should not be stressed by handling several times a day in order to receive medication, ophthalmic ointments are preferred because drug availability is prolonged despite the fact that eye drops are usually better absorbed by the globe.

A general understanding of the anatomy of the wild animal eye, mainly how the avian and reptilian eye differs from the mammal eye, is vital when differentiating between the normal and abnormal features in wild animal ophthalmology.[5,49]

Currently, some ophthalmic research is being conducted in South American native animals by several veterinary ophthalmologists who formerly worked exclusively with domestic animals. Consequently, data is being generated and clinical and surgical case communications are being presented in scientific meetings and published in periodicals. An important step would be to establish normal ranges for ocular tests routinely used in domestic animals. This type of activity should be encouraged because it is fundamental for the progress of wild animal ophthalmology. The following text is based on the work of pioneers such as Dr. Christopher J. Murphy, Dr. Michael G. Davidson, Dr. Thomas J. Kern, and Dr. David Williams, adding the author's experience with wild animal ophthalmology.

RESTRAINT FOR WILD ANIMAL OPHTHALMIC EXAMINATION

Methods of restraint for wild animal ophthalmic examination include manual, mechanical, chemical, and combinations of these methods. The best method varies with the species involved, individual temperament, and effect desired. In mammals, a number of methods may be used based on the size and behavior of each species. Generally, a combination of methods is best. For instance, the use of snares and muzzles with manual restraint has been shown to be an effective restraint technique for performing proper eye examinations in canids such as the maned wolf (*Chrysocyon brachyurus*). On the other hand, wild felids such as the cougar (*Puma concolor*) or ocelot (*Leopardus pardalis*) should always be examined under chemical restraint. It is not the size that matters, but the risk of injury for the ophthalmologist or patient during the examination that counts; for instance pacas (*Agouti paca*) are relatively small mammals but proper ophthalmic examination is virtually impossible without chemical restraint. Generally, ophthalmic examinations in birds may be performed with physical restraint only, but there are exceptions. Raptors tolerate well physical restraint but wild psittacines, such as macaws, and wild galliformes, such as currassows, may become stressed during the examination and lose a lot of feathers. Vocalization in macaws may be a problem for the ophthalmologist, mainly when trying to hear sound-producing instruments such as the Tono-Pen for intraocular pressure evaluation. Generally, reptiles tolerate ophthalmic examination well solely with physical restraint.

The type of restraint will influence directly the quality of the ocular examination. Sometimes the use of a particular method will alter some parameters checked by the ophthalmologist. For instance, the use of snares or any kind of restraint on the neck, even with hands, will increase the intraocular pressure (IOP) by impairing venous blood flow. Likewise, the use of some anesthetic drugs also may alter several parameters. Ketamine may cause an increase in IOP.[1] In spite of economic factors, staff availability, and stress to the patient, the ocular examination on large or very aggressive wild animals should be performed, ideally, with the patient under chemical restraint. Some birds, small mammals, and reptiles may be examined with proper physical restraint. When a manatee is out of the water, normally it keeps the eyes closed, making it difficult for an ophthalmic evaluation, but gentle digital stimulation of the animal's lips can encourage the eye exposition for examination.[38]

Darkness is required for a proper ophthalmic examination. Fortunately, darkness is one of the best manual restraint techniques for ratites such as the rhea (*Rhea americana*). Fractious birds consistently assume a quieter demeanor in the darkness and ophthalmic examination is greatly facilitated.[15]

WILD ANIMAL OPHTHALMIC EXAMINATION

The techniques of evaluating the wild animal eye are similar to those used regularly in domestic animals; this is especially true with mammals. However, some differences, mainly when examining reptiles and birds, will be emphasized here.

Each ophthalmologist has a particular pattern for ophthalmic examination. The important principle for an effective evaluation is to develop a logical, consistent use of the pattern of examination for each eye and for each patient, remembering that even when the lesion is obvious, all "apparently normal" portions of the eye should be examined very carefully. The algorithm in Figure 37.1 presents a suggestion for an effective ophthalmic examination that may be used in a wild or domestic animal (Figure 37.1).

It is worth mentioning that sometimes it is impossible to perform some parts of the examination because of special characteristics of each animal such as size of the eye globe, presence of striated iridal musculature, presence of spectacles, and the method of physical restraint chosen for each patient.

Basically, the instruments for evaluating the wild animal eye are the same as those used regularly in domestic animals.[9] Examination of the anterior segment of the eye may be performed with a bright penlight, a binocular loupe, an operating microscope, a direct ophthalmoscope set on +20 diopters or, ideally, a slit lamp (Figure 37.2).

Several special procedures and diagnostic methodologies must be associated with the ocular examination of wild animals, such as the use of biomicroscopes, vital dyes, tonometers, the Schirmer tear test, and ophthalmoscopes (Figure 37.3).

Domestic and practically all wild animal corneas are similar histologically, thus the clinical use of sodium fluorescein and rose bengal dyes for detection of corneal and conjunctival epithelial defects, erosions, and/or dead cells is also important in wild animal ophthalmology. Rose bengal stains dead cells and cells free of albumin and tear film. Thus, rose bengal can stain living corneal epithelial cells in animals suffering from keratoconjunctivitis sicca.[10] Fluorescein stains intercellular spaces. With corneal ulcers, abrasions, or lacerations, the dye enters the water-soluble corneal stroma and stains the intercellular spaces. The peak of fluorescence is best viewed when using a cobalt blue filter in the light source. The dye may also be used to check the patency of the nasolacrimal apparatus. Fluorescein and rose bengal are routinely used in wild animal patients in zoos by veterinary ophthalmologists in South America.

Tonometry

The Schiotz tonometer cannot be used in most small mammals, birds, and reptiles because of its large footplate. However, the portable Tono-Pen· applanation tonometer is ideal for use in most domestic and wild animal species, ideally those with corneal diameters over 9 mm (Figure 37.4). Sometimes it is possible to obtain reliable readings even in corneal diameters as small as 5 mm.[49] A tonometric evaluation of 275 birds (39 species) showed intraocular pressures in normal eyes between 9.2 and 16.3 mm Hg. Among 14 species of psittacine birds, IOP values were found to be 14.4 ± 4.2 mm Hg.[49] Another tonometric evaluation of normal eyes in raptors suggested mean IOPs ranging from 10.8 to 21.5 mm Hg in different North American raptors.[45] In birds and reptiles, the presence of a very active nictitant membrane may make tonometry a bit more difficult to perform. In snakes, the presence of the spectacle renders tonometry impossible. Table 37.1 lists normal IOPs of several different South American wild animal

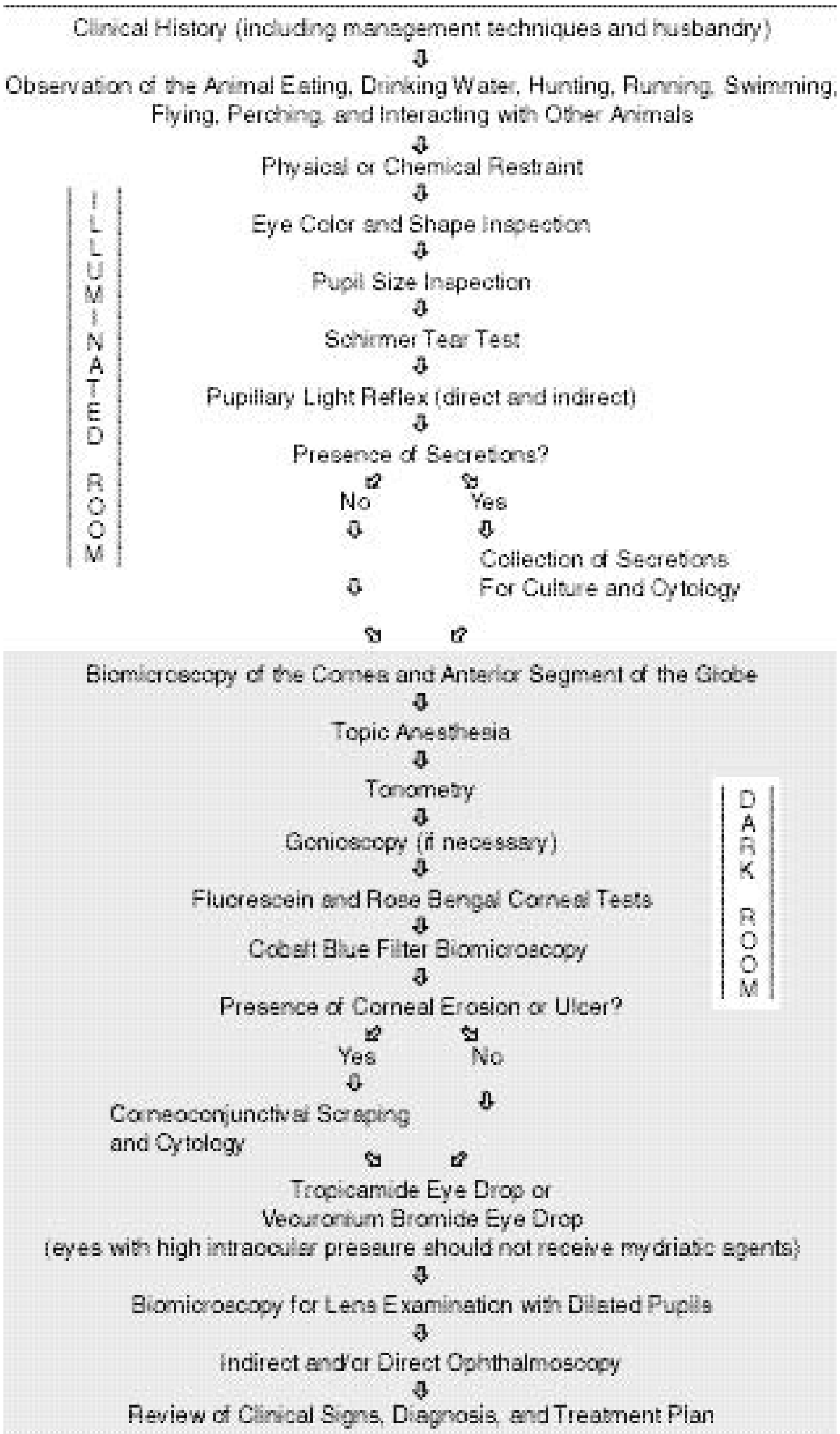

FIGURE 37.1. Algorithm for the wild animal ophthalmic examination.

FIGURE 37.2. Slit-lamp biomicroscopy is the ideal method of examining the anterior segment of the wild animal eye. The picture shows a black-hawk eagle (*Spizaetus tyrannus*) being examined.

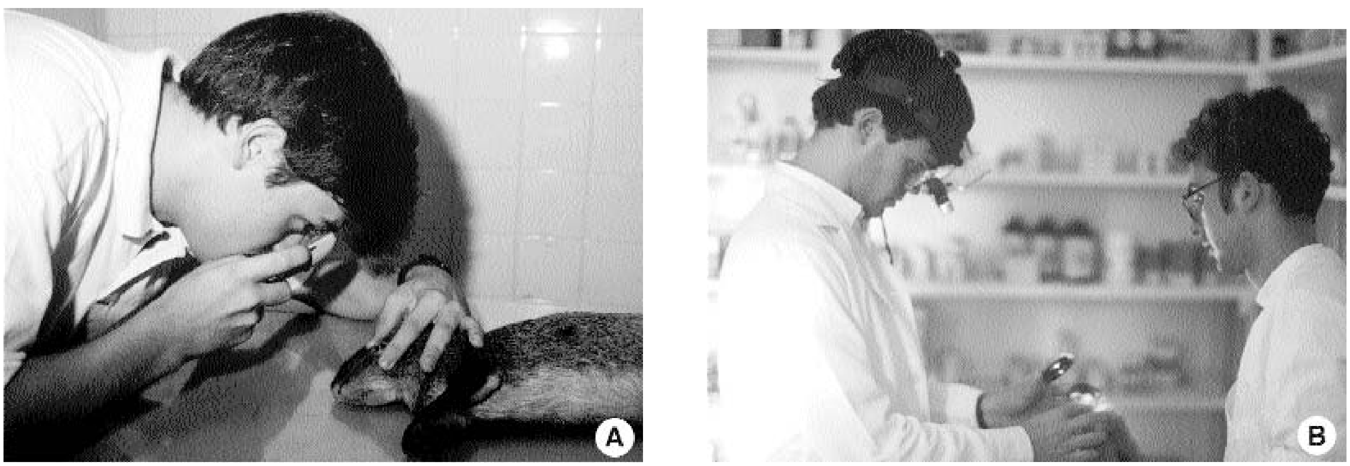

FIGURE 37.3. **A.** Monocular direct ophthalmoscopy for fundus evaluations being performed in an anesthetized adult agouti. When a slit-lamp biomicroscope is not available, the ophthalmoscope can be used to evaluate the anterior segment of the eye with some limitations. **B.** Binocular indirect ophthalmoscopy using a 28-diopter lens in a physically restrained mandarin. The indirect ophthalmoscopy is the best method of viewing the fundus in wild animals.

species. Further study is still needed for the establishment of normal values for the numerous species of animals found in South America.

Schirmer Tear Test

Measurement of lacrimal production is possible in wild mammals, reptiles, and birds using the Schirmer tear test (STT), just as in domestic mammals (Figure 37.5). In snakes, the presence of the spectacle renders STT impossible. Not for economical, but for practical reasons, the traditional 6-mm-wide Schirmer test filter paper strip must be trimmed to 3 mm (Figure 37.5). The trimming procedure has many advantages: It makes STT possible in small birds with small conjunctival sacs, it may be done in both eyes with one regular strip, and it promotes a higher reading of wetted strip length improving the margin of error of the test. In the conventional 6-mm-wide strip the Schirmer results are rarely over 3 mm in clinically normal patients. One report on trimmed STT values suggests 8 ± 1.5 mm for larger psittaciformes and 4.5 ± 1 mm for smaller species such as lories and conures.[49] Normal STT mean values for some South American wild animals are

FIGURE 37.4. Applanation tonometry is the ideal way of measuring intraocular pressure in wild animals. This picture shows a black-hawk eagle (*Spizaetus tyrannus*) being examined with a Tono-Pen.

TABLE 37.1. Mean intraocular normal pressures of selected South American wild animals

Common Name	Scientific Name	Number of Individuals	IOP (mm Hg)*	Type of Restraint
Mammals				
Agouti	*Dasyprocta azarae*	7	16.43 ± 4.89	Chemical [29]
Chinchilla	*Chinchila laniger*	4	20 ± 7.80	Physical
Alpaca	*Lama pacos*	2	17.25 ± 1.92	Physical
Brocket deer	*Mazama gouazoubira*	2	18 ± 2.00	Physical
Maned wolf	*Chrysocyon brachyurus*	6	18.6 ± 4.2	Physical
Ocelot	*Leopardus pardalis*	5	17.90 ± 1,97	Chemical [28]
Birds				
Bare-throated bellbird	*Procnias nudicollis*	4	09.66 ± 2.08	Physical
Bearded bellbird	*Procnias averano*	2	12.34 ± 0.58	Physical
Blue-fronted parrot	*Amazona aestiva*	5	15.33 ± 2.21	Physical
Blue and yellow macaw	*Ara ararauna*	4	15.40 ± 1.94	Physical
Coscoroba swan	*Coscoroba coscoroba*	2	16.25 ± 2.36	Physical
Razor-billed curassow	*Mitu tuberosa*	2	16.50 ± 3.54	Physical
Toco toucan	*Ramphastos toco*	2	17.50 ± 2.12	Physical
Chestnut-eared aracari	*Pteroglossus castanotis*	2	18.25 ± 2.20	Physical
Black-hawk eagle	*Spizaetus tyrannus*	3	14.50 ± 1.97	Physical
White-tailed hawk	*Buteo albicaudatus*	2	12 ± 1.40	Physical
Savanna hawk	*Buteogallus meridionalis*	2	17.75 ± 1.40	Physical
Mantled hawk	*Leucopternis polionota*	1	18 ± 0.00	Physical
Harpy eagle	*Harpia harpyja*	2	15.50 ± 3.04	Physical
Burrowing owl	*Athene cunicularia*	4	16.20 ± 0,84	Physical
Striped owl	*Asio clamator*	6	13.66 ± 2.17	Physical
Reptiles				
Green iguana	*Iguana iguana*	2	06.20 ± 1.92	Physical
Tegu	*Tupinambis merianae*	4	09.25 ± 1.19	Physical

*The IOP measurements were made using an applanation tonometer (Tono-Pen XL, Mentor, Norwell, Massachusetts, USA). Only those values with ≤ 5% variance were accepted.

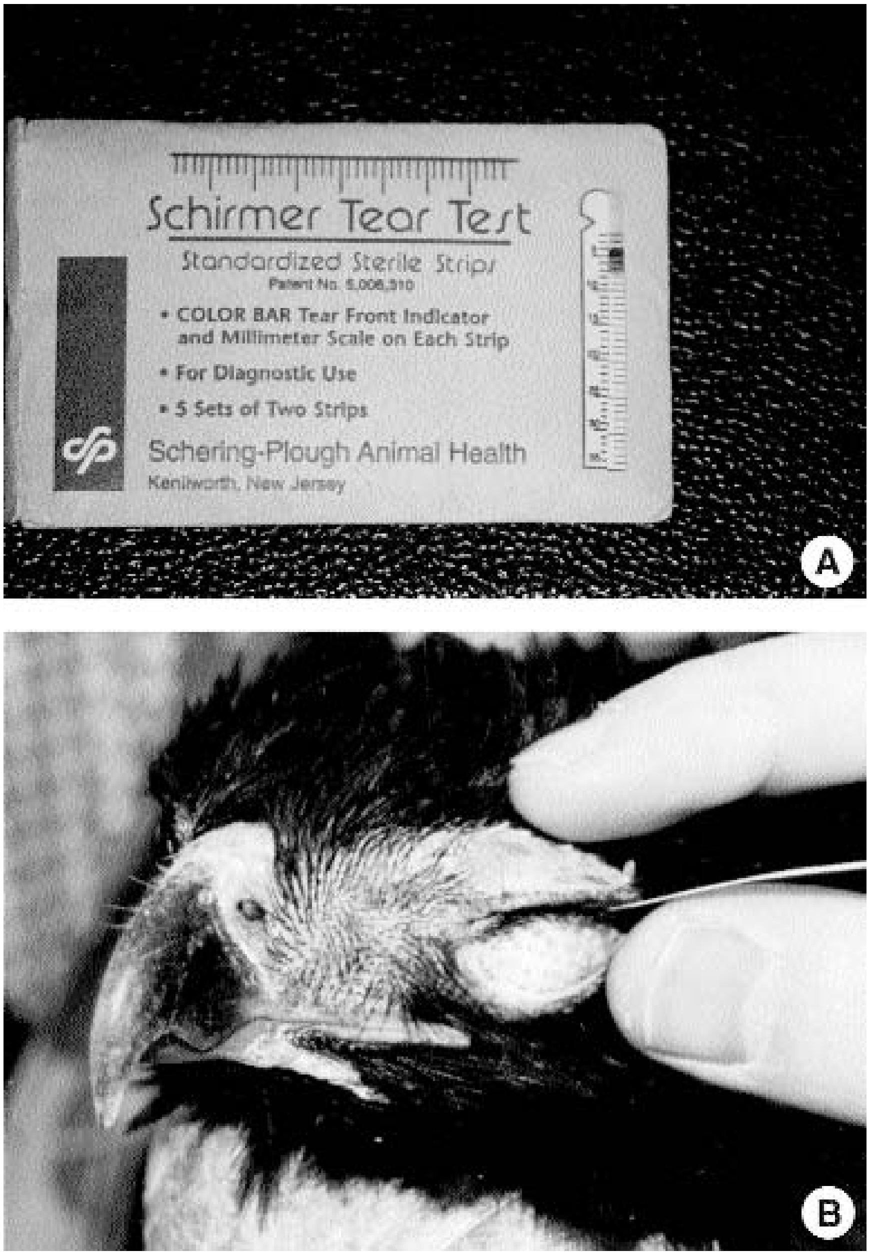

FIGURE 37.5. A. Schirmer tear test filter paper trimmed to 3 mm. This was the standard width used to determine the values in Table 37.2.
B. Schirmer tear tests can be used in several species of wild animals. This picture shows a black-hawk eagle being examined.

available on Table 37.2. Apparently, reptiles have lower STT values.

Induction of Mydriasis

The presence of striated iridal musculature, rather than smooth, in birds and reptiles necessitates modified procedures to see the posterior segment of the eye. This renders mydriasis with parasympatholytic or sympathomimetic drugs impossible. In birds and reptiles, with the exception of snakes, topical or intracameral administration of nondepolarizing muscle relaxants such as atracurium besylate, *d*-tubocurarine, and vecuronium bromide produces mydriasis. The topical use of vecuronium bromide solution (4 mg/mL) without surface-acting penetrating agents such as saponin or benzalkonium chloride, seems to be the most safe and effective way to partially paralyze the iris musculature of such species and induce mydriasis.[17,22,39,45] In snakes, the presence of spectacles renders intracameral injection of curare a necessity. Because species-related differences in absorption of the drug across the cornea occur, the same procedure may be used in reptiles and avian patients when the topical use of the drug failed to produce mydriasis.[30,49,50] However, some systemic side effects may be seen with this procedure. Another use of intracameral injection of curare would be to use it prior to cataract surgeries, when mydriasis induced by topical

TABLE 37.2. Mean Schirmer tear test (mm/min) normal values for selected South American animals

Common Name	Scientific Name	Number of Individuals	Schirmer Values (mm/min)*
Blue-fronted parrot	*Amazona aestiva*	5	6.40 ± 1.51
Burrowing owl	*Athene cunicularia*	4	7.66 ± 3.78
Black-hawk eagle	*Spizaetus tyrannus*	3	7.20 ± 1.70
Green iguana	*Iguana iguana*	2	2.5 ± 0.70

*All tests were made with standardized sterile strips (Schering-Plough Animal Health, Kenilworth, New Jersey, USA).

agents does not last long enough.[30] Sometimes, in birds and reptiles, the physical restraint itself can induce mydriasis compatible for fundoscopy. Additionally, variable mydriasis may be achieved fairly consistently under general anesthesia.[17,50] Actually, the use of general anesthesia to induce mydriasis for routine fundus examination in birds and reptiles is a reliable and safer method for achieving mydriasis rather than the use of curariform drugs.[33,50] In most of the mammals mydriasis can be easily and effectively induced by the instillation of 1% tropicamide or 1% atropine. However, the pupils of pigmented rodents such as agoutis occasionally are resistant to dilation because the melanin granules in the iris bind the mydriatic agent and thus inhibit its action. In such cases, 1% atropine solution mixed with equal parts of 10% phenylephrine solution and applied three to four times during a 10- to 15-minute period is an effective method for inducing mydriasis.[8] In small-sized eyes, such as in most companion birds, reptiles, and small mammals, the fundus may be very difficult to see, even with good pharmacologically induced mydriasis, because of the small size of the globe. Sometimes the illumination source must be turned down to the minimal intensity to allow fundus visualization.

SURGERY

Zoos in South America are usually not well equipped with material necessary for ophthalmic surgeries. Sometimes logistic limitations make transportation to a surgical room, usually available at university centers, impossible. Usually, on these occasions, the ophthalmologist has to carry everything with him or her. A customized resistant case, internally covered with foam with tight compartments for the biomicroscope, ophthalmoscope, tonometer, head loupe, goniolens, stains,

FIGURE 37.6. A portable binocular head loupe with a focusing spotlight is an economical, practical alternative when performing selected ocular surgeries at zoo facilities.

mydriatic agents, saline solution, gauze, nasolacrimal cannulae and surgical instruments is desirable. There are numerous ophthalmic surgical instruments, but a good basic set should include: one instrument case with silicon "fingers" suitable for autoclaving; adjustable Castroviejo eye speculum in three different sizes (small, medium, and large); straight blunt strabismus and/or tenotomy scissors; Castroviejo's corneal scissors (left and right handed), plus iris, straight Mayo's, curved Mayo's and curved Metzembaum's scissors; dull iris hook; Adson's tissue forceps (toothed); Von Graefe's fixation forceps; Colibri's forceps; medium rat-tooth forceps; Castroviejo's curved needle holder; Derf's needle holder; lens loupe; two-way irrigation/aspiration cannula; silicone irrigation bulb; scalpel blade handle; Graefe strabismus hook (medium). Finally, suitable light and magnification must be available for the surgeon. Ideally, a portable microscope should be carried to the zoo, but at least a focusing spotlight attached to a binocular head loupe (with interchangeable lenses with different magnification powers) (Figure 37.6) can be used for simple intra- and extraocular procedures in several species.

BIRDS

Ophthalmic Anatomy and Physiology

The avian orbit is large and open, with displacement of the brain caudally. The bones that separate the orbit from the cranial vault are thin and pneumatized. The orbits are separated by a thin bony or fibrous interorbital septum.[33] An important feature of the comparative anatomy of the avian orbit is the close proximity of the infraorbital sinus to the infraorbital diverticulum.[9] Sinusitis may easily lead to periorbital or orbital compression and signs of periorbital swelling, conjunctivitis, and sometimes intraocular disease (Figure 37.7).

In most birds the globe is large relative to the size of the body, flattened anterio-posteriorly, with a hemispherical posterior segment. Actually, the avian eye is both relatively and absolutely large. It is flat or discoid in passeriforms and in the majority of avian species, tubular in all owls and some eagles, and finally globose in falconiforms, crows, and insectivorous wing feeders.[17,33,43] Generally, the posterior segment is larger than the anterior segment. The tubular-shaped eyes have the greatest visual acuity. The four recti and two oblique extraocular muscles are not well developed. The avian eye is relatively immobile and only capable of limited movement, between 2 and 5°, in all planes. There are also two other muscles, the pyramidalis and quadratus, originating from the posterior aspect of the globe responsible for moving the nictitant membrane across the eye.

The eyelids are thin and susceptible to trauma. The lower eyelid is more mobile then the upper and generally contains a fibroelastic tarsal plate. The lid margin is generally pigmented and has delicate filoplumes associated with it. The tarsal (meibomian) glands are absent, but other gland structures are present. These include Harder's gland (present in all species) and the orbital lacrimal gland (variably present), located in the ventrotemporal part of the orbit and covered by conjunctiva. It is small in aquatic birds, large in others, and absent in owls.[43] Some marine birds have a nasal salt gland, an ovoid and yellowish glandular structure located dorsomedially to the eyeball. Its duct penetrates the frontal bone, entering the nasal cavity. The main function of the salt gland is to excrete salt solutions, presumably through a stimulus from the osmolality of the plasma.[43] The nictitating membrane is much more active than the eyelids; it is thin, nearly transparent, and capable of cleaning debris from the cornea. It distributes tears across the surface during blinking and menace response. It has a pigmented margin and a marginal plait, which forms a trough for capture and distribution of tears.[33,49] The nictitating membrane has an unusual and interesting muscular arrangement in the avian eye. The nictitating membrane is voluntarily drawn from the nasal canthus across the eye by the pyramidal muscle that originates from the posterior pole of the sclera and loops over the optic nerve through a sling formed by the insertion of the quadratus muscle (bursalis muscle). The quadratus muscle amplifies the action of the pyramidalis muscle.[17,33,49] Two lacrimal puncta are present and drain lacrimal secretions into a nasolacrimal duct.[17]

The avian eye follows the basic structure of the mammalian, consisting of three concentric tunics, aqueous humor, lens, and vitreous body. The cornea, unlike mammals, has a microscopically evident Bowman's layer. Anteriorly, a variable number of scleral ossicles (10–18, the majority have 15) unite to form a complete bony ring within the sclera. The ossicles determine the shape of the globe and provide firm sites of origin for the striated muscles.[33] The structure of the globe is also maintained posteriorly by a scleral cartilage cup. In some species, a separate ossicle is related to the exit of the optic nerve.[33]

The iris and ciliary body contains striated dilator and constrictor muscles. However, smooth muscle has also

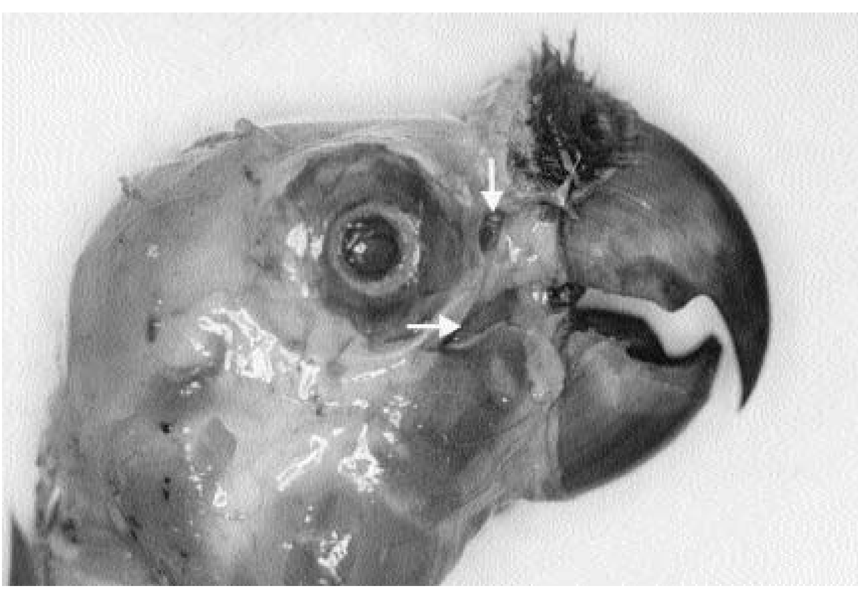

FIGURE 37.7. Note the presence of the infraorbital diverticulum of the infraorbital sinus, over and below the zygomatic arch (arrows) in a blue-fronted Amazon parrot (*Amazona aestiva*) cadaver.

been identified in the iris tissue of the great horned owl.[35] Iris colors vary with age, species, and even gender of the bird in many South American raptors.[49] Constriction and dilation of the pupil are subject to influence from retinal stimulation as well as voluntary control. There is no consensual pupillary light reflex because of the complete separation of the optic nerves, which means 100% decussation at the optic chiasm.[33,49] There is an extensive trabecular elastic fiber network of pectinate ligaments, better developed than in mammals. This network bridges the angle between the cornea, sclera, and ciliary body. The pecten is a pigmented, pleated, highly vascular structure of variable size unique to birds.[17, 43]

Primary glaucoma seems to be rare in birds. This can be explained by the very wide apertures and the superficial position of the scleral venous sinus.[20]

The intimate connection between the ciliary body processes and the lens capsule explains why lens luxation is rarely observed in birds.[20] It usually extends into the vitreous body from and over the optic nerve head, making it impossible to see the optic nerve when performing ophthalmoscopy in birds. The pecten may be visualized on ultrasonographic examination (Figure 37.8). The pecten probably has a primary nutritive function to the internal aspect of the avascular retina, but it has been credited with more than 30 possible functions.[17,43] Three main morphological types of pecten are recognized in birds: conical, vaned, and pleated. The conical type is recognized only in kiwis. The vaned form is seen in ostriches, rheas, and tinamous. Finally, the pleated type of pecten is seen in the rest of the avian species.[43]

The lens is softer than in mammals with an equatorial annular pad formed by modified lens fibers, which serves to regulate lens metabolism and accommodation. The pads are smaller in birds with small accommodative ranges (nocturnal species) and largest in diurnal birds and fast-flying species.[17] Optical accommodation in birds involves changes in corneal curvature as well as anterior movement and deformation of the lens.[17] Scleral ossicles reinforce the ciliary body and along with sclera permit a more powerful accommodative function compared with other animals. Lens power in birds is also increased by contraction of the striated meridional ciliary muscles. The ciliary muscles are Brucke's muscle posteriorly and Crampton's muscle anteriorly. Crampton's muscle inserts on the cornea and moves the ciliary body axially, thus compressing the lens by exerting pressure on the annular pad. Contraction of Crampton's muscle flattens the peripheral cornea.[17, 43]

The retina is avascular and atapetal. Considerable variation in photoreceptor type and density exist. Most domestic species are afoveate, others are monofoveate, and some are even bifoveate (hummingbirds, some raptors, and passerines). Both rods and cones are present, including variable amounts of double cones with oil droplets. The retinal pigmented epithelium contains pigment granules and is able to elongate their cell bodies between the rods in response to light stimulation.[17]

Curiously, the ostrich has the largest eye of land vertebrates.[43] In South America, the rhea probably is one of the land vertebrates with largest eyes.

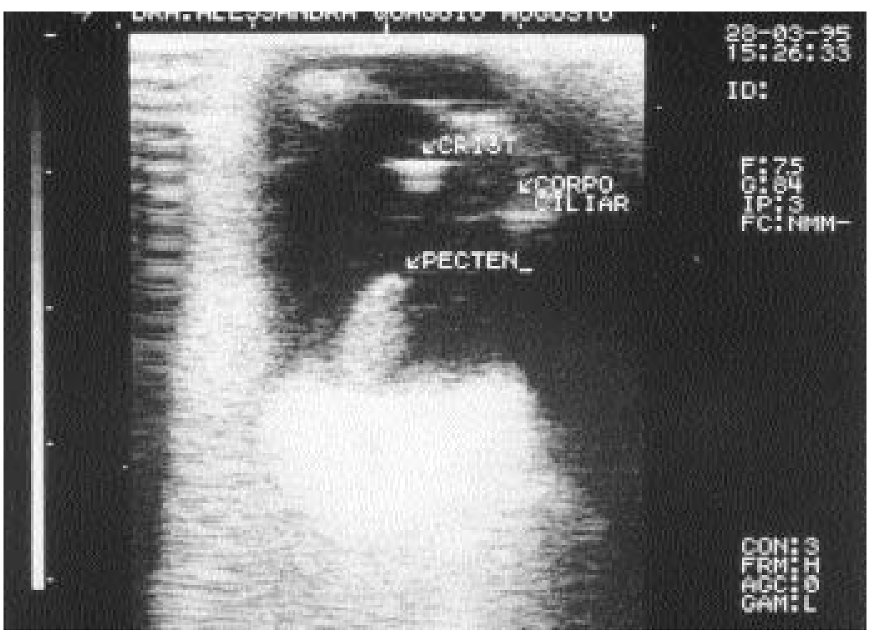

FIGURE 37.8. Longitudinal sonogram of a harpy eagle (*Harpia harpyja*) eye globe. Note the presence of the pecten protruding from the ocular fundus. (Courtesy of Dr. Alessandra Q. Augusto.)

Common Eye Diseases

INFECTIOUS DISEASES

Periorbital Disease. The interconnection of the nasal cavity, infraorbital sinuses, and the porous calvaria creates a situation in which inflammatory reactions in the sinus or nasal passages caused by bacteria, fungus, or virus may involve most of the anatomic structures of the head.[47] The large representation of the orbits in birds, as well as the close proximity of the infraorbital sinus, makes the eye a special target for inflammatory extension of respiratory disease. The periorbital disease secondary to upper respiratory infection, particularly chronic rhinitis and sinusitis, is one of the most common ophthalmic disease presentations if not the most common disorder affecting caged birds, particularly in a flock situation. The typical clinical presentation is depression, periocular swelling (Figure 37.9), dyspnea, rhinorrhea, purulent nasal discharge, open-mouthed breathing, sneezing, coughing, and occasionally voice change.

Not all clinical signs must be present for suspecting the disease. The best diagnostic method in these cases is performing needle aspiration of the infraorbital diverticulum of the infraorbital sinus, under the zygomatic arch (Figure 37.7). The needle is passed through the skin at the commissure of the mouth and directed toward a point midway between the eye and external nares. This procedure provides diagnostic material for culture and cytologic examination. The material should be examined after staining with acid fast, Giemsa, and Gram dyes. Several organisms may cause sinusitis, therefore orbital diseases may also have numerous causes and treatments according to cytology and culture. Fungal agents, gram-positive and gram-negative bacteria, *Mycoplasma* spp., as well as the intracellular bacteria *Chlamydia psittaci,* are infectious agents commonly diagnosed in these cases. Most of the cases seen by the author were diagnosed in wild caged birds. Chlamydiosis is enzootic in South America and does not seem to represent a threat to free-living wild birds under natural conditions.[7] The stress of captivity seems to predispose wild birds to innumerous ophthalmic and nonophthalmic infectious diseases. When chlamydiosis develops in caged birds there is a great risk of infection to healthy birds and humans as well.

Periocular disease also is common in patients affected with avian pox, a viral disease of many species of wild and domestic birds. Noninfectious causes of sinusitis are also possible. Hypovitaminosis A, toxins, smoke, and papilloma are the most common causes. After recognizing the etiology of upper respiratory and periorbital disease, the systemic treatment is formulated with specific antibiotics or antifungal agents chosen according to culture and sensitivity results. Sometimes, rational empiric antibiotic therapy is used, and in these cases doxycycline (25 mg/kg orally every 24 hours) or chloramphenicol (50 mg/kg intramuscularly every 12 hours) are good choices.

Diet correction is important for a satisfactory outcome. The systemic treatment alone is often unsatisfactory. Valuable procedures that should be added to the systemic therapy include: nebulization therapy with antibiotics, sinus flushing, and, occasionally, surgical debridement of the sinus. The antibiotics routinely used

FIGURE 37.9. Infraorbital sinusitis in a vinaceous parrot (*Amazona vinacea*). Note the extensive periocular swelling. Culture has revealed pure isolate of *Streptococcus* spp.

in nebulization therapy include chloramphenicol (200 mg/15 mL of saline), doxycycline (water-soluble tablets 100 mg/15 mL saline), cefotaxime (100 mg/15 mL of saline) or piperacillin (100 mg/15 mL of saline). The topical ophthalmic drug also is best chosen when culture and sensitivity are available. For rational antibiotic topical therapy for the eye, chloramphenicol or tetracycline eye drops and ointments applied three to four times a day are prescribed routinely.

Avian Pox. There are two recognized forms of avian pox, the wet (or diphtheritic) and the dry (or cutaneous), which can exist singularly or in combination. Ocular lesions in the diphtheritic form include caseous plaques on the eyelids, swelling of the eyelids, symblepharon, and severe exudative conjunctivitis. In the cutaneous form, proliferative skin lesions develop around the eyes, the beak, the nares, and the feet. Crusts form on the eyelids around 2 weeks after infection and often become secondarily infected with bacteria and fungi.[8] The canary and the blue-fronted Amazon parrot are highly susceptible to the effects of the disease.[8,21]

Avian pox is routinely diagnosed based on clinical signs. However, histopathological demonstration of characteristic intracytoplasmatic inclusion bodies is possible, if eye tissue samples, mainly from conjunctival epithelium, were taken.[8]

Treatment protocols include topical ophthalmic antibiotic (ciprofloxacin 0.3% four to six times a day), vitamin A (30,000 IU/kg once every 10 days intramuscularly) and systemic enrofloxacin (10 mg/kg every 12 hours orally or intramuscularly). Cutaneous pox lesions are treated with 0.05% povidone iodine every 12 hours.

Parasitic Diseases. Knemidokoptic mange usually involves the beak and the cere of birds, however, periorbital involvement is also common.[8] *Oxyspirura mansoni* is a small nematode that has been reported to infect the conjunctiva, conjunctival sacs, nictitating membrane, and lacrimal ducts of several species of birds, causing conjunctivitis.[36] Treatment includes flushing the conjunctiva with saline solution, mechanical removal of the organisms, plus administration of ivermectin at 0.3 mg/kg orally, diluted in water.

HYPOVITAMINOSIS A Hypovitaminosis A is frequently diagnosed in psittacines fed all-seed diets. Usually, it is seen as a complicating eye disease factor rather than a common ocular disease entity. Hypovitaminosis A should be suspected when periorbital and/or conjunctival swelling, blepharitis, keratoconjunctivitis sicca, and ocular discharge are observed for unexplained reasons, together with palatine and choanal abscesses, particularly in Amazon parrots. It has also been seen in a burrowing owl fed solely with meat. Response to oral vitamin A therapy is also diagnostic. The treatment protocol is vitamin A at 50,000 IU/kg intramuscularly weekly, including diet supplementation with leafy green or yellow vegetables.

CATARACTS The opacification of the lens, lens capsule, or both are quite common in birds and are a relatively common cause of blindness in older caged or free-living animals.[8,30,33,42] Several old canaries have been seen with cataracts as a cause of blindness. Hereditary characteristics also have been shown in the development of these opacities in canaries.[44] In addition, the phenomenon of lens resorption was exhibited in birds, although subsequent improvement in vision was not noted as it sometimes is in dogs.[44] In passeriforms, because of the small size of the globe, lens extraction usually is not attempted. However, in birds of prey, extracapsular[30,31] and phacoemulsification[18,34] lens extraction techniques are possible, and all have been successfully performed (Figure 37.10). Apparently, the avian uveal tissue does not respond as dramatically to the trauma of cataract surgeries as most small animals do, although phacoanaphylactic endophthalmitis has been already recognized in an owl.[2]

TRAUMA Traumatic lesions of the eye globe are quite common in wild free-living birds. Corneal ulcers, hyphema, traumatic uveitis, scleral ossicles fractures, synechia, and cataractous changes, as well as lesions in the posterior segment, are frequent clinical findings in those cases[6] (Figure 37.11). Frequently, there are associated injuries in the body such as skin lacerations and bone fractures.

In birds of prey, trauma is undoubtedly the most common underlying cause of ocular lesions. Because of the large size of the globe and relative lack of orbital protection, any form of cranial trauma frequently involves the eye and its accessory structures.[33] Traumatic uveitis and hyphema should be treated with topical and systemic corticosteroids. Birds with traumatic uveitis respond very well to corticosteroids. Superficial corneal wounds are treated with frequent application of topical antibiotics. Ideally, the selection of the drug may be made after culture and sensitivity testing of the agent. Routinely, most superficial ulcers in birds heal well with empiric antibiotic therapy. Ciprofloxacin and tobramycin are good choices. Tarsorrhaphy, nictitans flaps, or corneal grafting are required for deeper corneal ulcers and descemetoceles, respectively. Due to skeletal muscle supply to the nictitans, regular third eyelid (nictitan) flaps are difficult to maintain. In the author's experience the best method is fixing the sutures in the cartilaginous margin plate of the nictitans and in the

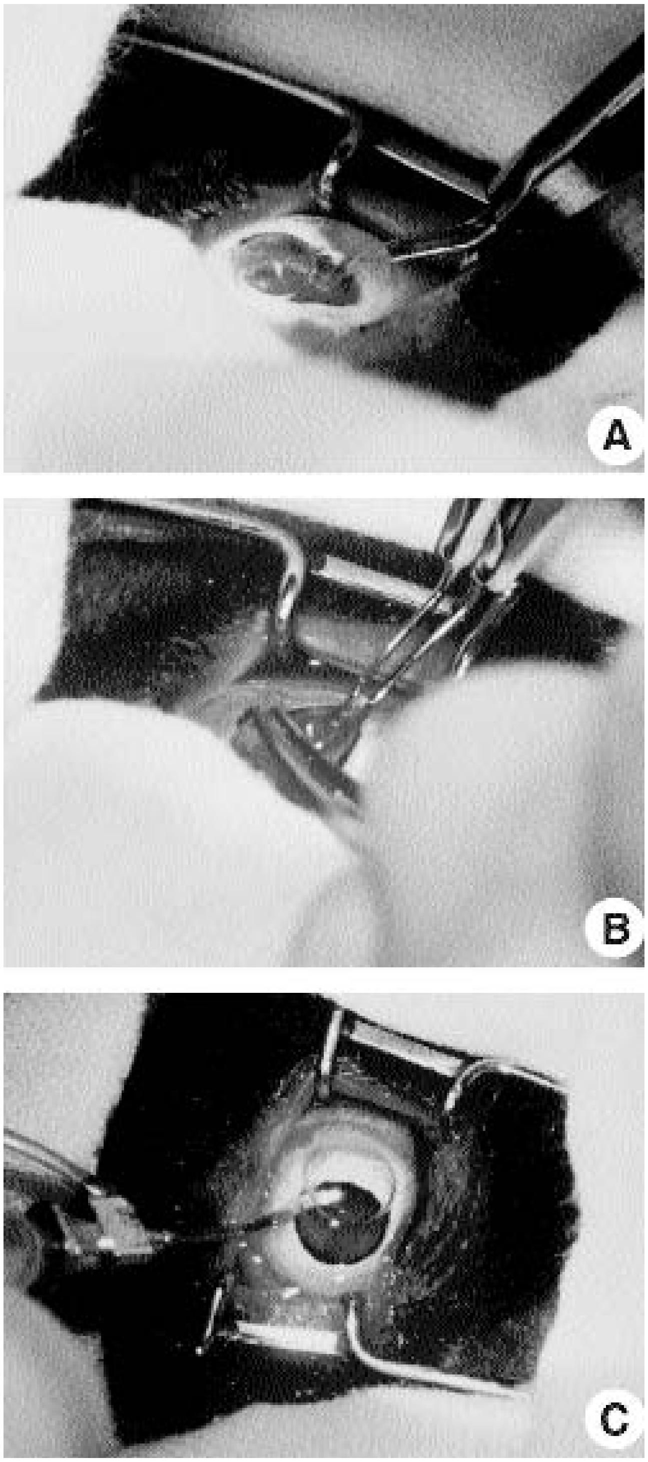

FIGURE 37.10. Extracapsular phacectomy being performed in a king vulture (*Sarcorramphus papa*). **A.** Capsulorhexis being made with a capsulorhexis forceps after corneal incision. **B.** Lens removal. **C.** Continuous lactated Ringer's solution infusion through an aspiration/irrigation cannula and anterior chamber depth restoration.

FIGURE 37.11. Traumatic corneal ulceration stained with fluorescein in a free-living caracara (*Poliborus planchus brasiliensis*).

bulbar conjunctiva near the limbus with 5-0 or 6-0 suture material.[26]

ENUCLEATION In severely compromised eyes, such as those presenting intraocular tumors, severe trauma, severe endophthalmitis, abscesses, or orbital tumors, enucleation may be a necessity. There are two ways one can perform the surgery in birds: using a globe collapsing procedure or enucleating the entire globe using a transaural approach.[25,32]

REPTILES

Ophthalmic Anatomy and Physiology

Reptile patients can be classified in three major groups: the lacertinlians (lizards), the chelonians (turtles), and the ophidians (snakes). The ocular anatomy of snakes differs considerably from that of other reptiles.[8]

The upper and lower eyelids in reptiles are well developed, with the lower being more mobile. Crocodilians possess a bony tarsus in the upper lid. Strong closure of the upper lid in crocodilians often hinders ocular examination.[23] Some lizards have the lower lid variably transparent.[17]

In snakes, ablepharine geckos, and skinks the upper and lower eyelids are fused to form a transparent tertiary spectacle covering the cornea.[23] The spectacle is separated from the cornea by a very narrow epithelial-lined tear-filled space. The anterior layers of the spectacle are shed during normal ecdysis with the rest of the skin. Before ecdysis, the spectacle becomes cloudy because of the thickening and breakdown of skin layers with an accumulation of fluid between the new and old surface layers of the spectacle. During the period in which the spectacle is cloudy, the animal is blind, and many species may become more irritable and aggressive during this time.[17]

Lacrimal secretion runs into the subspectacular space and is drained by the nasolacrimal duct to the roof of oral cavity. The nictitating membrane is absent in snakes.[8] Lizards have a Harder's gland ventromedially placed in the orbit and a lacrimal gland placed dorsotemporally to the globe. Geckos and chameleons lack a lacrimal gland, as snakes do, and in these species the Harder's gland is well developed. Chelonians have large Harder's and lacrimal glands. The Harder's and lacrimal gland secretion of the crocodilian tear film is supplemented by conjunctival accessory glands.[50] The nasolacrimal duct is absent in chelonians.[17]

Reptiles also have poorly developed rectus muscles (except in lizards), well-developed retractor bulbi muscle (except in snakes), and limited rotational movements (except in chameleons). As in birds, hyaline cartilage is present in the sclera of lizards, crocodilians, and chelonians, also with scleral ossicles in lizards and chelonians.[17, 23] These tissues must undergo decalcification if eyes are submitted to histopathology.[23] Snakes lack both scleral ossicles and cartilage, and crocodilians lack scleral ossicles.[17]

Regarding the uveal tissue, iris color, pupil shape, and vascular patterns are variable. Both ciliary and iris sphincter muscles are striated and ciliary processes are present in all reptiles except lizards.[8,17,23]

The reptilian retina is avascular and supplied by the choroid. In some reptiles, there is a vascular conus papillaris that derives from the hyaloid vasculature and projects from the optic nerve head into the vitreous. Snakes lack a conus papillaris; instead the retina is covered by the membrana vasculosa, a fine network of capillaries overlying but not penetrating the surface of the retina.[8] This structure complements choriocapillaris blood supply to the retina.[17]

Some reptiles have a so-called third eye, the pineal or median eye; some have only the vestige of this median eye, the pineal gland. It is best developed in lampreys and some reptiles such as the green iguana (*Iguana iguana*) (Figure 37.12). The pineal eye is part of a complex of pineal organs, the pineal, parapineal, and paraphysis, which develop from the diencephalon, and could function for detecting food, predators above the animal, or for sensing circadian light/dark cycles and day length. The most specialized pineal eyes (of lizards) have a miniature cornea, lens, and even retina, but these probably detect only the presence/absence of light.[9]

Common Eye Diseases

Hypovitaminosis A. Vitamin A deficiency is commonly recognized in reptiles.[37] The disease is most frequently diagnosed among young, rapid-growing chelonians such as terrapins fed diets with high protein content, such as meat and/or insects and no vegetables, thus deficient in either beta carotene or preformed vitamin A, as retinol esters. This deficiency renders dyskeratotic changes caused by squamous metaplasia of the orbital glands and their ducts. The glands begin to swell as the desquamated cells block their ducts. Orbital glands are most developed in chelonians, which

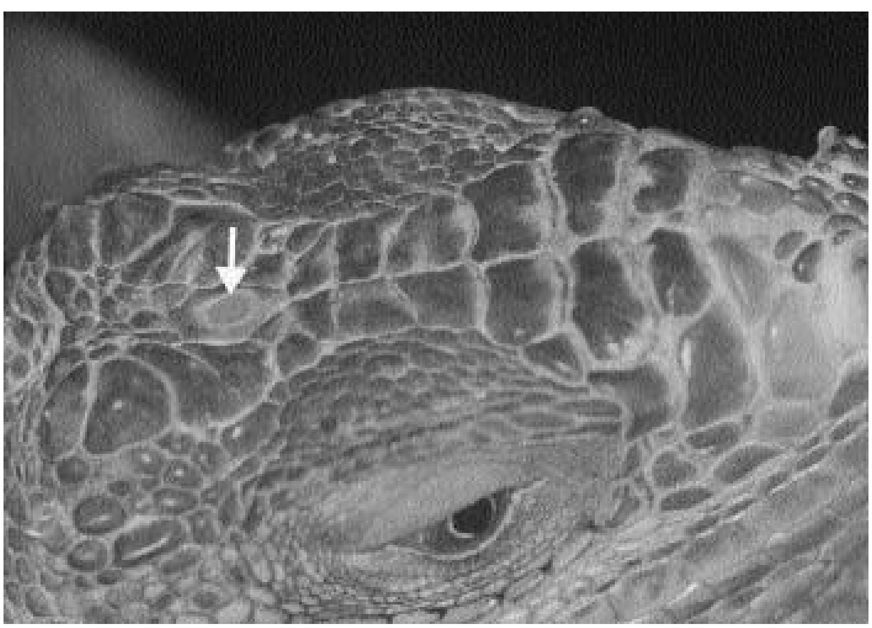

FIGURE 37.12. The so-called third eye, or pineal eye, in a green iguana (*Iguana iguana*). Note the semitransparent scale (arrow). A small cranial aperture is located right beneath the specialized scale.

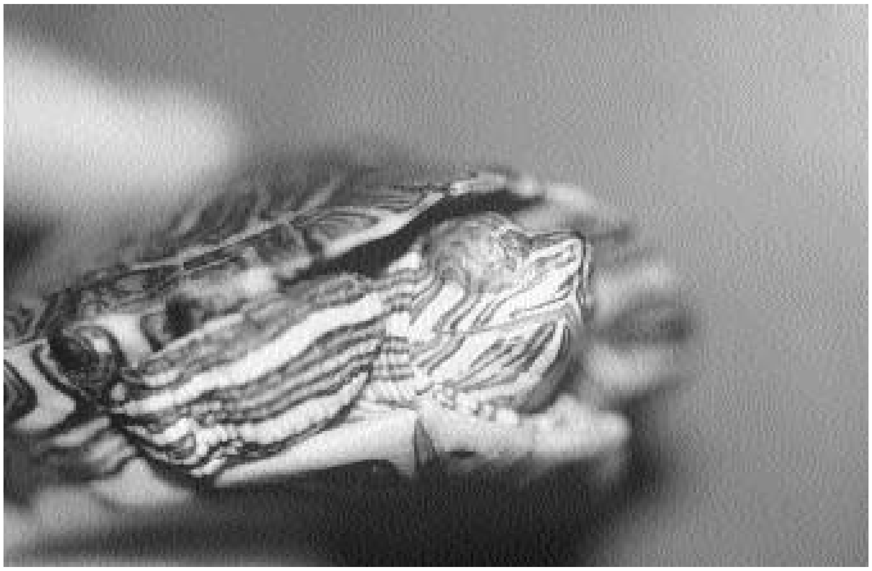

FIGURE 37.13. Mild eyelid edema in a young slider with hypovitaminosis A. (Courtesy of Dr. J. Ricardo Pachaly.)

explains the prevalence of hypovitaminosis A–induced ophthalmic disease in this order.[23] The early ocular changes of the disease are confined to mild eyelid edema and secondary bacterial blepharoconjunctivitis (Figure 37.13).

Subsequently, the animal becomes blind as the palpebral fissure becomes closed. Concomitantly, squamous metaplasia of the renal, pancreatic, gastrointestinal, and respiratory epithelia may progress, leading to miscellaneous clinical signs and a fatal consequence.[12, 50]

During the early stages of the disease, when the animal is still eating, the emphasis must be on the correction of the diet. Balanced chelonian diets commercially available are indicated. Additionally, a recipe is usually given to the client who is instructed to mix meat, dark green vegetables, and calcium carbonate, forming a homemade pellet that should be given to the animal in the water. Routinely, ciprofloxacin 0.3% ophthalmic ointment is prescribed to be used three times a day. The use of an ophthalmic ointment formulation aids in lubricating the ocular tissues and stays on the eye longer than the eye-drop formulations in aquatic environments. Cyclosporine A ophthalmic ointment has been used with success in these cases, probably because of its B- and T-lymphocyte immunosuppressant activity and maybe as a result of its improvement of tear production. However, none of this has been proved in reptiles. Cyclosporine 1% has been used as an ointment being administered twice a day for 21 days. Actually, 21 days is usually the time required for recovery in moderate to severe cases of hypovitaminosis A.

Water-soluble vitamin A, 1000 to 5000 IU, is given weekly by intramuscular injection until recovery is complete. In patients also presenting respiratory disease and/or multiorganic involvement, amikacin 5 mg/kg should be administered by intramuscular injection every 48 hours until recovery is complete.

Retained Spectacles. Improper shedding of the skin or dysecdysis is a common problem among snakes, and it often causes spectacular retention. Several generalized integumentary diseases, inappropriate environment (particularly with low humidity), systemic illness, and mite or tick infestation are common causes of dysecdysis,[50] therefore are common causes of retained spectacles as well. Direct local injuries to the spectacles also may cause the disease. In animals presenting dysecdysis, pieces of skin left attached may harbor many types of disease-causing organisms such as bacteria, fungi, and mites. The best method to remove the attached skin is to soak the patient in a diluted iodine solution (iodine/water, 1:50) in a container deep enough to cover its body. If this method doesn't work, the spectacle must be removed surgically.

Subspectacular Abscess. There is some overlap between dysecdysis and subspectacular abscesses, because one may complicate the other. Subspectacular abscess may occur as a manifestation of septicemia, ascending infection of the oral cavity (such as ulcerative stomatitis) through the nasolacrimal duct, or as an extension of local periocular infection. Several agents have been isolated from this form of infection such as *Proteus* spp., *Providencia* spp., *Aspergillus* spp., and *Geotrichum* spp.[27]

The best treatment for subspectacular abscess must be directly against the causative agent, implicating culture and cytology of the subspectacular exudate. Surgical excision of the affected spectacle following flushing of the space with an antiseptic solution must be performed as well.

FIGURE 37.14. Exophthalmos due to retrobulbar abscess formation in the right eye of a Brazilian freshwater turtle (*Phrynops* sp.). The globe was subsequently enucleated. (Courtesy of Dr. J. Ricardo Pachaly.)

Whether or not the case is dysecdysis or a subspectacular abscess, one has to bear in mind that the cornea underlying the spectacle is very thin and fragile and may be perforated easily.

Bacterial Infections. Bacterial infections causing abscessation involving the cornea and/or conjunctiva, the periorbit, and the retrobulbar area are commonly seen, mainly as the result of poor husbandry. Several cases in wild free-living animals are frequently diagnosed as well. Some infections may even lead to panophthalmitis. Curiously, gram-negative organisms such as *Proteus* spp., *Pseudomonas* spp., and *Aeromonas* spp. are present in almost the totality of the cases. Treatment of these infections includes systemic and topical antibiotics. Antibiotics, such as amikacin 5 mg/kg should be administered every 48 hours intramuscularly as the first choice and enrofloxacin at 10 mg/kg every 48 hours intramuscularly as the second choice. Drainage and spectacle removal must be planned for snakes, since the spectacle is impermeable to topically applied medications. After spectacle removal, ciprofloxacin 0.3% ophthalmic solution or tobramycin 0.3% ophthalmic solution can be used every 8 hours for 10–14 days. In some severe cases, the retrobulbar abscessation has irreversibly compromised the eye function rendering enucleation as the only choice (Figure 37.14).

Other Ocular Diseases. Cataracts and lenticular sclerosis occur in snakes[8] (Figure 37.15). A glaucoma case has been reported in a South American caiman (*Caiman latirostris*) (Figure 37.16).

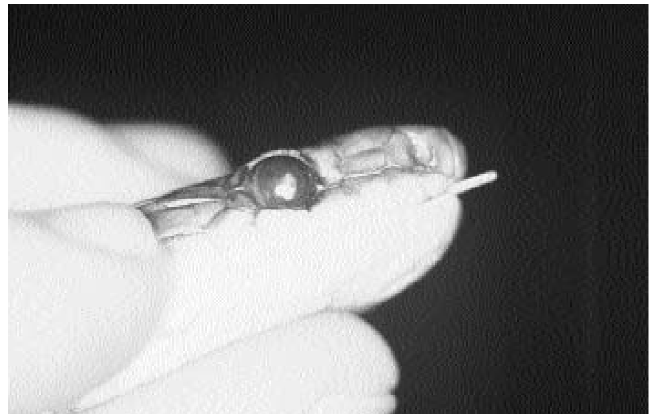

FIGURE 37.15. Mature cataract of unknown origin in a free-living snake (*Philodryas olfersii*).

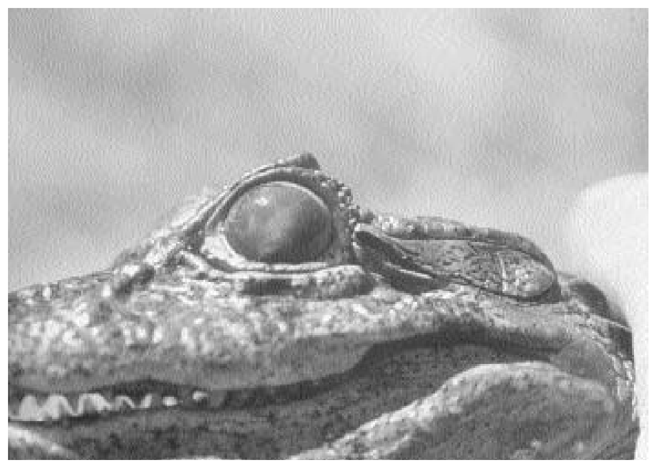

FIGURE 37.16. Glaucoma and buphthalmos in a captive caiman (*Caiman latirostris*). (Courtesy of Dr. Daniel Herrera.)

MAMMALS

The eye of most wild mammals differs little from the domestic phylogenetically related counterparts. These features include a spherically shaped globe protected by an upper, a lower, and a third eyelid. The upper eyelid is the most mobile; the action of the third eyelid is entirely passive, sweeping over the cornea as the globe is retracted into the orbit. Also, there are two tear-producing glands: a lacrimal gland situated in the temporodorsal aspect of the orbit and an accessory lacrimal gland associated with the third eyelid.[8]

Lagomorphs have only one nasolacrimal punctum for each eye. It is located in the conjunctiva between the lower lid and the nictitans, and it is quite large (2-4 mm in diameter).[4] Members of the order Lagomorpha (rabbits and hares) are prone to inflammatory and infectious diseases of the conjunctiva and nasolacrimal drainage system. Many agents have been isolated: *Pasteurella multocida, Staphylococcus aureus,* poxvirus, myxomatosis virus, and allergies.[19] Canids and some large felids have round pupils, but smaller felids have vertical elliptical pupils. Alpacas, llamas, and vicuñas have an oval in a horizontal plane–shaped pupil, with extensions of the posterior pigmented epithelium called corpora nigra. Most rodents and rabbits have round pupils; ferrets have horizontally oval pupils that contract to small slits.[8] Curiously, however, chinchillas have vertical elliptical pupils that contract to small vertical slits. Different than birds and reptiles, iridal musculature is nonstriated, allowing pharmacologic blockage of its action by parasympatholytic agents, such as tropicamide. Additionally, there is no pecten or conus papillaris. The pattern of retinal vascularization varies according to the species. Felids, canids, and rodents have holangiotic retinas. Guinea pigs have anangiotic retinas, whereas rabbits have merangiotic retinas.[51] In some mammals, the choroid contains a layer of reflective tissue called the tapetum lucidum. The tapetum is roughly triangular in shape when viewed funduscopically, and it varies in color. It reflects light that has passed through the retina and, thus, restimulates the photoreceptor cells. The tapetum is responsible for the "eye shine" seen at night when animals face a light source and for the background color of the fundus (Figure 37.17). Alpacas seem to show a direct relationship between iris color, fundus pigmentation, and coat color[13] (Figure 37.18).

The tapetal layer is composed of collagenous fibers in herbivores (Figure 37.18) and iridocytes containing reflecting crystals in carnivores (Figure 37.17) but there are exceptions to this rule. The tapetum lucidum of the paca (*Cuniculuc paca*) is cellular.[41] Despite great variation in the diameter of the eye in mammals, there is comparatively little variation in thickness of retina. The largest mammalian eyes, for example, are about 50 times the diameter of the smallest, and yet the thickness of the retinas never varies by more than a ratio of two to one.[14]

Reported Ophthalmic Malformations

Some ophthalmic malformations have been reported in South American wild mammals. Distichiasis and entropion were surgically corrected in two collared peccaries.[17] Cyclopia and limb deformity of unknown cause was also reported in a collared peccary.[16]

A lens coloboma and optic nerve colobomas were reported in the guanaco and alpaca, respectively.[3,13] Several other congenital ophthalmic disorders have been reported in llamas and, infrequently, in alpacas, including cyclopia, agenesis of the distal portion of the nasolacrimal duct, entropion, ectropion, focal eyelid agenesis, microphthalmia, heterocromia irides, cataracts, and persistent hyperplastic primary vitreous.[13]

Miscellaneous Ophthalmic Diseases and Conditions

Wild felids presenting metabolic osteodystrophies may show strabismus as a result of orbital deformations and interference with the action of extraocular muscles.

A pediculated conjunctival graft in the cornea of a Bactrian camel (*Camelus bactrianus*) was performed recently. During the surgery, intense chemosis was

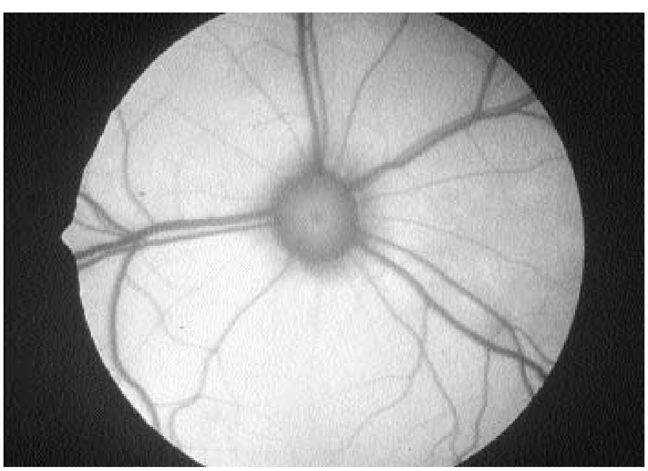

FIGURE 37.17. Ocular fundus of an adult jaguar (*Pantera onca*). Note the holangiotic retina over an extensive tapetal area. (Courtesy of Dr. D.E. Brooks.)

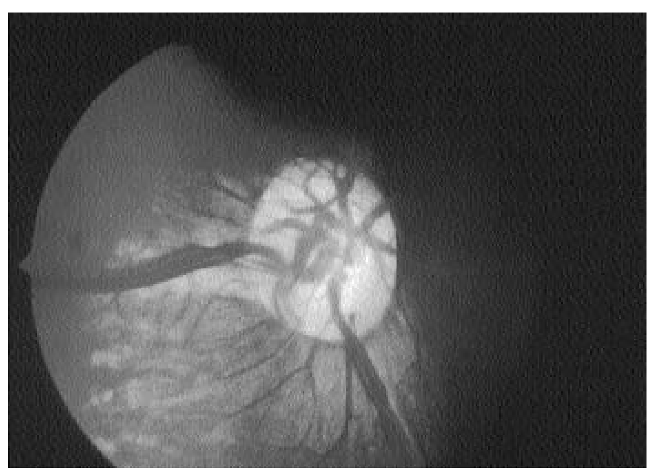

FIGURE 37.18. Ocular fundus of a 3-year-old male alpaca. Note the numerous blood vessels emerging from the center and periphery of the optic nerve head. (Courtesy of Dr. D.E. Brooks.)

observed in response to the surgical insult. Chemosis was resolved a few days after the surgery. Camels are closely related to South American camelids (llama, alpaca, guanaco and vicuñas). However, it is not known if the same happens with those animals.

Corneal diseases in wild mammals follow the same treatment protocols used in domestic small animal ophthalmology. Keratitis is frequently reported in the captive Malyan tapir (*Tapirus indicus*). Affected eyes show corneal opacity, corneal ulceration, and conjunctivitis. This lesion probably is caused by physical trauma and aggravated by excessive exposure to sunlight, because in nature this animal lives in dense tropical jungles that protect its eyes from exposure to direct sunlight.[40] Many pacas (*Agouti paca*) and tapirs (*Tapirus terrestris*) have been seen with lesions that might fit the same description. Curiously, both these species live in dense tropical jungles usually protected from sunlight. Virtually all individuals affected were living in zoos and exhibited in areas without any protection from sunlight. This data gives support to the implication of excess exposure to sunlight as a cause of this disorder. Before the treatment protocol is initiated, the eyes should be swabbed for culture and cytology. Besides ophthalmic topical medication, third eyelid flap or tarsorrhaphy may also help corneal healing. It is worth mentioning that pacas do not have third eyelids, therefore temporary tarsorrhaphy is the only choice. Care should be taken to prevent contact of the suture material with the cornea, which may aggravate the disease. The best option is performing a noninvasive tarsorrhaphy, suturing silicon tubes along with the lid margins and subsequently tying them up, always passing the suture material through the tubes (Figure 37.19).

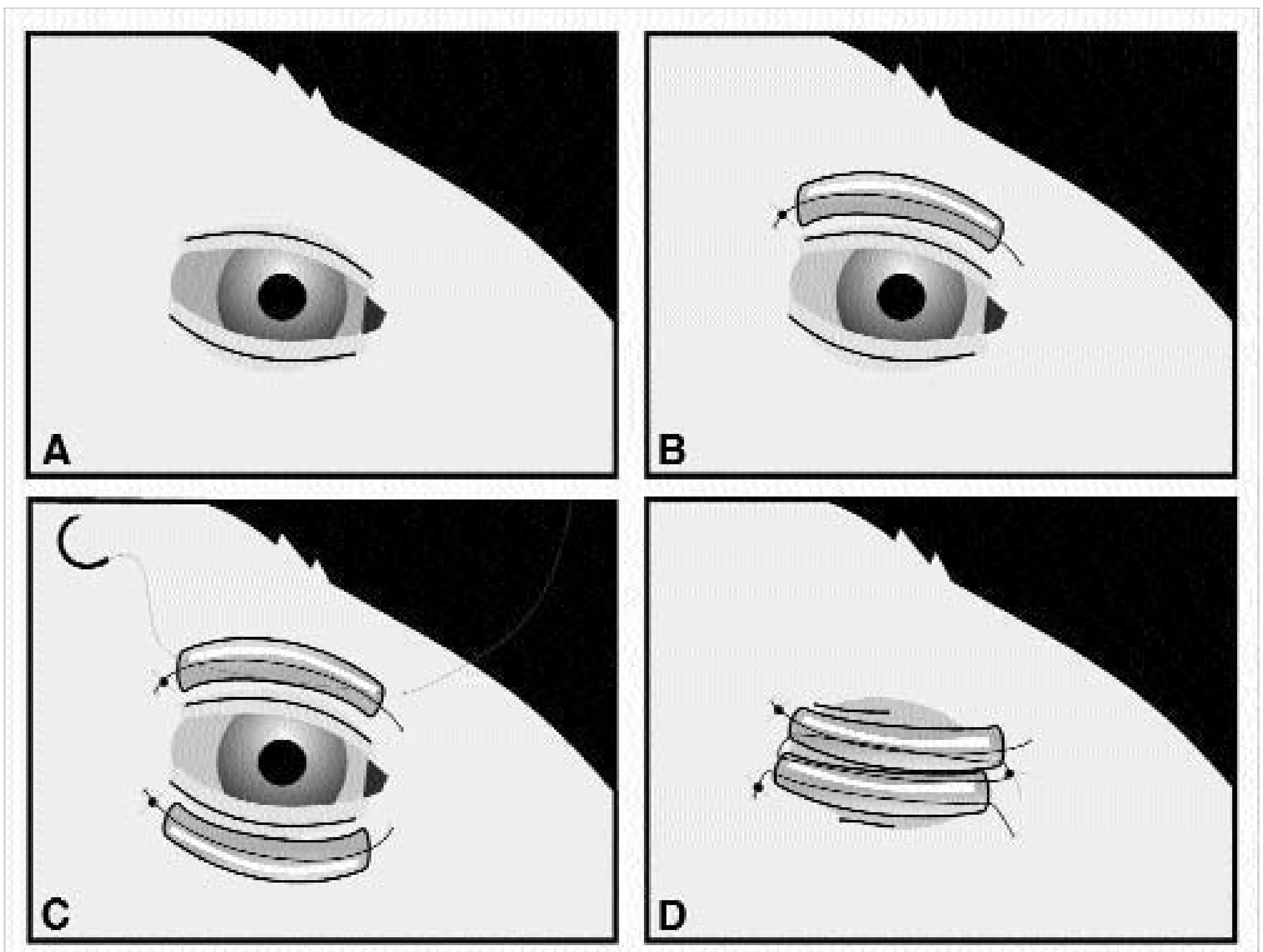

FIGURE 37.19. Noninvasive tarsorrhaphy indicated for mammals with no third eyelid. **A.** The animal is anesthetized and placed in lateral recumbency. The area is prepared for aseptic surgery. **B.** A small silicone tube is cut to a size slightly shorter than the length of the lid margins. The tube is sutured to the lid parallel to the upper lid margin with 3-0 nonabsorbable suture material passing through the tube. **C.** The same procedure is done in the lower lid. **D.** Another line is then passed through both tubes and tied together promoting close apposition of the lid margins.

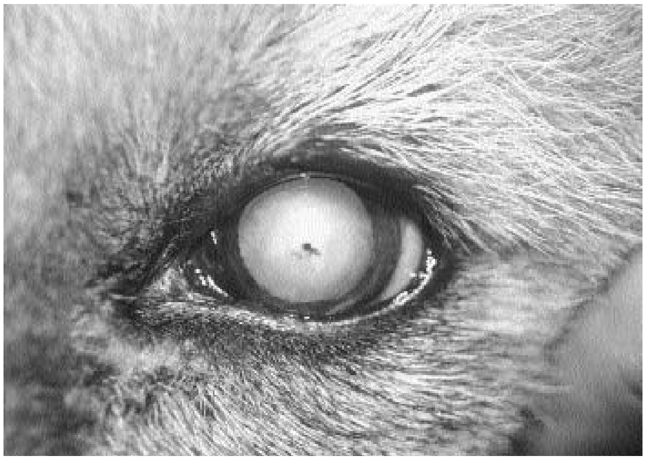

FIGURE 37.20. Hypermature cataract in a maned wolf (*Chrysocyon brachyurus*). Note the presence of uveal pigment adhered to the anterior lens capsule surface (former posterior synechia). (Courtesy of Dr. J. Ricardo Pachaly.)

Keratoconjunctivitis in llamas has been associated with *Moraxella liquefaciens*. Blindness and encephalitis develop in alpacas and llamas in association with equine herpesvirus.[13]

Lagomorphs and rodents are prone to corneal ulcers.[19] Most ulcers do not cause craters or pits as are typical in dogs or cats; they more closely resemble stromal abscesses. Numerous chinchillas are presented with this condition. The stromal abscesses may be septic or sterile. In most septic cases *Staphylococcus* spp. or *Pasteurella* spp. were isolated from the infection site.

Wild felids are susceptible to infection with feline herpesvirus and calicivirus.[11] These upper respiratory pathogens may cause paroxysmal sneezing episodes, serous or mucopurulent nasal discharge, conjunctivitis, oral ulceration, and ulcerative keratitis. The presence of these clinical signs in wild felids, particularly in young patients, suggests feline viral upper respiratory infection. However, much work must be done to establish a true percentage of upper respiratory infections attributable to these viruses. The treatment protocol is based on oral antibiotics such as amoxicillin–clavulanic acid, rehydration therapy, and trifluridine applied topically to the eye.

Cataracts seem to be common in almost every mammal. Several species of captive wild mammals have been diagnosed with cataracts of presumably known origin, such as traumatic or secondary uveitis (Figure 37.20) but several animals probably show genetic bilateral cataracts of unknown origin, with no vestige of other concurrent ocular problem. One agouti (*Dasyprocta azarae*) with diabetes mellitus showed polyuria, polydipsia, hyperglycemia, glycosuria, and immature cataracts[24] (Figure 37.21). In Brazil, cataracts also are commonly diagnosed in wild and captive sirenians and pinnipeds and often are associated with corneal opacities and vascularization. Curiously, the anterior corneal stroma of the adult manatee contains normally sparsely populated blood vessels, visible in optical microscopy.[41]

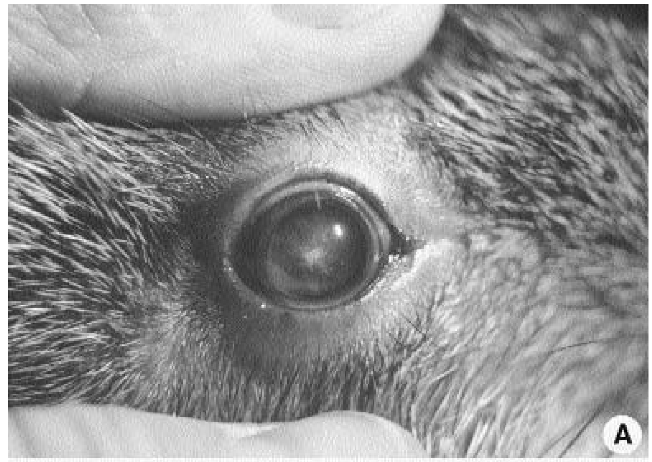

FIGURE 37.21. A and B. Bilateral cataracts in a diabetic agouti (*Dasyprocta azarae*). (Courtesy of Dr. P.R. Werner.)

In Curitiba's Zoological Park a litter of maned wolf pups was hand raised and bottle-fed with cow milk, goat milk, and a commercial canine milk replacer, interchangeably for 45 days. At this time the pups had developed extensive immature cataracts, with diffuse cortical lens opacification in both eyes. It is known that the milk of timber wolves contains high amounts of arginine. When timber wolf pups were fed an arginine-deficient milk, such as a regular canine milk replacer, cataracts developed.[48] The same may have happened with these maned wolves. The arginine content of maned wolf milk is unknown. However, it is logical to believe that cataract development was caused by deficient amounts of arginine received by the pups fed on nonmaned wolf milk.

Just as observed in a small percentage of dogs after vaccination, uveitis and corneal edema have developed in a young maned wolf (*Chrysocyon brachyurus*) vaccinated 14 days previously with a combination containing canine hepatitis virus.[46]

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REFERENCES

1. Allen, D.G.; Pringle, J.K.; and Smith, D.A. 1998. Handbook of Veterinary Drugs, 2nd. Philadelphia, Lippincott-Raven, p. 886.
2. Anderson, G.A.; and Buyukmihci, N. 1983. Phacoanaphylactic endophthalmitis in an owl. Veterinary Pathology 20:776–778.
3. Barrie, K.P.; and Jacobson, E. 1978. Unilateral cataract with lens coloboma and bilateral corneal edema in a guanaco. Journal of the American Veterinary Medical Association 173:1251.
4. Burling, K.; Murphy, C.J.; Curiel, J.S.. 1991. Anatomy of the rabbit nasolacrimal duct and its clinical applications. Prog. Vet. Comp. Ophthalmol. 1:33–40.
5. Bellhorn, R.W. 1973. Ophthalmologic disorders of exotic and laboratory animals. Veterinary Clinics of North America 3(3):345–356.
6. Buyukmihci, N.C. 1985. Lesions in the ocular posterior segment of raptors. Journal of the American Veterinary Medical Association 187(11):1121–1124.
7. Cubas, Z.S. 1993. Natural diseases of free-ranging birds in South America. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 166–171.
8. Davidson, M.G. 1985. Ophthalmology of exotic pets. Compendium on Continuing Education for the Veterinarian Practice 7(9):724–736.
9. Eakin, R.M. 1973. The Third Eye. Berkeley, University of California Press, p. 112.
10. Feenstra, R.P.; and Tseng, S.C. 1992. What is actually stained by rose bengal? Arch. Ophthalmol. 110:984–993.
11. Fowler, M.E. 1986. Felidae. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 831.
12. Frye, F.L. 1991. Reptile Care: An Atlas of Diseases and Treatments, Vol. 2. Neptune City, T.F.H. Publications, p. 637.
13. Gelatt, K.N.; Otzen Martinic, G.B.; Flaneig, J.L. 1995. Results of ophthalmic examinations of 29 alpacas. Journal of the American Veterinary Medical Association 206(8):1204.
14. Glyckstein, M. 1976. The vertebrate eye. In R.B. Masterton, M.E. Bitterman, C.B.G. Campbell, and N. Hotton, eds., Evolution of Brain and Behavior in Vertebrates. Hillsdale, New Jersey, Lawrence Erlbaum Associates, pp. 53–71.
15. Jensen, J.M. 1986. Ratite restraint and handling. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 198–203.
16. Hellgren, E.C.; Lochmiller, R.L.; and Thomas, M.W. 1984. Cyclopia, congenital limb deformity, and osteomyelitis in the collared peccary (*Tayassu tajacu*). Journal of Wildlife Medicine 20:225–227.
17. Kern, T.J. 1999. Exotic animal ophthalmology. In K.N. Gelatt, ed., Veterinary Ophthalmology. Philadelphia, Lippincott Williams & Wilkins, pp. 1273–1305.
18. Kern, T.J.; Murphy, C.J.; and Riis, R.C. 1984. Lens extraction by phacoemulsification in two raptors. Journal of the American Veterinary Medical Association 185(11):1403–1406.
19. Kirschner, S.E. 1997. Ophthalmologic disease in small mammals. In E.V. Hiller and K.E. Quesenberry, eds., Ferrets, Rabbits and Rodents: Clinical Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 339–345.
20. Korbel, R.; Reese, S.; and Hegner, K. 1998. Anatomical and clinical examination of the iridocorneal angle (gonioscopy) and the ciliary body in various bird species. In Proceedings of the European Association of Zoo and Wildlife Veterinarians. pp. 329–341.
21. McDonald, S.E.; Lowenstine, L.J.; and Ardans, A.A. 1983. Avian pox in blue-fronted Amazon parrots. Journal of the American Veterinary Medical Association 183(11):1218.
22. Mikaelian, I.; Paillet, I.; and Williams, D. 1994. Comparative use of various mydriatic drugs in kestrels (*Falco tinnunculus*). American Journal of Veterinary Research 55(2):270–272.
23. Millichamp, N.J. 1995. Reptile ophthalmology. In J.D. Bonagura, ed., Kirk's Current Veterinary Therapy, Vol. 12. Small Animal Practice. Philadelphia, W.B. Saunders, pp. 1361–1365.
24. Montiani-Ferreira, F. 1996. Catarata e diabetes mellitus em *Dasyprocta azarae* [Cataract and diabetes mellitus in *Dasyprocta azarae*]. In Proceedings of the 15° Congresso Panamericano de Ciências Veterinárias. Curitiba, PR, CPGCV, p. 81.
25. Montiani-Ferreira, F.; Mangini, P.R.; Cartelli, R.; Pachaly, J.R.; Teixeira, V.N.; Augusto, A.Q.; and Reifur, L. 1997. Review of ophthalmic surgical interventions performed at the Federal University of Parana's wild animal medicine service between the years of 1996 and 1997. Archives of Veterinary Science 2(suppl.):64.
26. Montiani-Ferreira, F.; Mangini, P.R.; and Pachaly, J.R. 1995. Técnica cirúrgica de recobrimento corneano por membrana nictitante em gavião carcará (*Poliborus planchus brasiliensis*) com ulceração de cornea [Corneal flap technique using nictitating membrane in a caracara (*Poliborus planchus brasiliensis*) presenting deep

corneal ulceration]. In Proceedings of the 2° Simpósio sobre Ciências Médicas e Biológicas-Cinquentenário da Descobertta da Leishmania Enrietti. Curitiba, ANCLIVEPA/PR, p. 26.
27. Montiani-Ferreira, F.M.; Mangini, P.R.; Pachaly, J.R.; and Werner, P.R. 1997. Geotricose cutânea em uma serpente (*Elaphe gutata):* Relato de caso [Cutaneous geotrichosis in a serpent (*Elaphe gutata*):-Case report]. In Proceedings of the 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. p. 23.
28. Montiani-Ferreira, F.; Pachaly, J.R.; and Ciffoni, E.M.G. 1996. Normal values for intraocular pressure in *Felis pardalis*. In Proceedings of the 15° Congresso Panamericano de Ciências Veterinárias. Santa Cruz de La Siera, PANVET, p. 71.
29. Montiani-Ferreira, F.; Pachaly, J.R.; and Ciffoni, E.M.G. 1997. Valores de pressão intra-ocular em cutias (*Dasyprocta azarae*) anestesiadas pela associação cloridrato de xilazina, cloridrato de ketamina e sulfato de atropina [Intra-ocular pressure values in agouti (*Dasyprocta azarae*) anesthetized with xylazine, ketamine and atropine]. In Proceedings of the 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. Curitiba, ANCLIVEPA/PR, p. 23.
30. Montiani-Ferreira, F.; Teixeira, V.N.; Pachaly, J.R.; and Silva, A.S.P. Cataract correction in a king vulture (*Sarcorramphus papa*) using the extracapsular surgical techinique with an irrigation/aspiration device. Archives of Veterinary Science 1(1):45–46.
31. Moore, C.P.; Picket, J.P.; and Beehler, B. 1985. Extracapsular extraction of senile cataract in an Andean condor. Journal of the American Veterinary Medical Association 187(11):1211–1212.
32. Murphy, C.J.; Brooks, D.E.; Kern, T.J.; Quesenberry, K.E.; and Riis, R.C. 1983. Enucleation in birds of prey. Journal of the American Veterinary Medical Association 183(11):1234–1237
33. Murphy, C.J. 1993. Raptor ophthalmology. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 211–221.
34. Murphy, C.J.; and Riis, R. 1984. Lens extraction by phacoemulsification in two raptors. Journal of the American Veterinary Medical Association 185(11):1403–1406.
35. Oliphant, L.W.; Johnson, M.R.; Murphy, C. 1983. The musculature and pupillary response of the great horned owl iris. Experimental Eye Research 37:583.
36. Pass, D.A. 1993. Disease of free-ranging birds in Australia. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 172–177.
37. Pachaly, J.R.; Mangini, P.R.; Teixeira, V.N.; Cartelli, R.; Augusto, A.Q.; Montiani-Ferreira, F.; Reifur, L.; Klemz, C.; Sincero, P.C.; Mafra, A.P.; Candido, M.V.; and Ciani, R.B. 1997. Estudo da prevalência de hipovitaminose a em quelônios atendidos no serviço de medicina de animais selvagens e odontologia veterinária do hospital veterinário da ufpr no período de setembro de 1995 a outubro de 1997. Archives of Veterinary Science 2(suppl.):65.
38. Piggins, D.; and Muntz, W.R.A. 1983. Physical and morphological aspects of the eye of the manatee *Trichechus inunguis*. Marine Behavior and Physiology 9:111–129.
39. Ramer, J.C.; Murphy, J.P.; Brunson, D.; and Murphy, C.J.P. 1996. Induction of mydriasis in cockatoos, African gray parrots, and blue-fronted Amazon parrots. Journal of the American Veterinary Medical Association 208(2):227–230.
40. Ramsay, E.C. 1993. Infectious disease of the rhinoceros and tapir. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 459–466.
41. Samuelson, D.A. 1999. Ophthalmic anatomy. In K.N. Gelatt, ed., Veterinary Ophthalmology. Philadelphia, Lippincott Williams & Wilkins, pp. 31–150.
42. Scmidt, R.; and Toft, J.D. 1981. Ophthalmic lesions in animals from a zoologic collection. Journal of Wildlife Diseases 17(2):267–275.
43. Shivaprasad, H.L.P. 1998. A bird's eye view of avian ocular anatomy and pathology. In Proceedings of the Association of Avian Veterinarians. pp. 273–282.
44. Slatter, D.H.; Bradley, S.; Vale, B.; Constable, I.J.; and Cullen, L.K. 1983. Hereditary cataracts in canaries. Journal of the American Veterinary Medical Association 183(8):872–874.
45. Stiles, J.; Buyukmihci, N.C.; and Farver, T.B. 1994. Tonometry of normal eyes in raptors. American Journal of Veterinary Research 55(4):477–479.
46. Thomas-Baker, B. 1985. Vaccination-induced distemper in maned wolves and vaccination-induced corneal opacity in a maned wolf. In B. Thomas-Baker, ed., Proceedings of the American Association of Zoologists and Veterinarians. p. 53.
47. Tully, T.N.; and Harrison, G.J. 1994. Pneumology. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine Principles and Application. Lake Worth, Florida, Wingers Publishing, pp. 556–581.
48. Vainisi, S.J.; Edelhauser, H.F.; Wolf, E.D.; Cotlier, E.; and Reeser, F. 1981. Nutritional cataracts in timber wolves. Journal of the American Veterinary Medical Association 179(11):1175–1180.
49. Williams, D.L. 1994. Ophthalmology. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine Principles and Application. Lake Worth, Florida, Wingers Publishing, pp. 673–694.
50. Williams, D.L. 1996. Ophthalmology. In D.R. Mader, ed., Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 175–185.
51. Williams, D.L. 1999. Laboratory animal ophthalmology. In K.N. Gelatt, ed., Veterinary Ophthalmology. Philadelphia, Lippincott Williams & Wilkins, pp. 1209–1236.

38
The Oral Cavity

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INTRODUCTION

Examination of the oral cavity for early detection and correction of problems should be a part of the general health care program in any institution that deals with wild animals. Zoo veterinarians should adopt and follow a strict routine of physical examination of the oral cavity whenever any animal is chemically immobilized. Furthermore, handlers should be trained to recognize early signs suggestive of oral diseases.

Preventing medical problems of the oral cavity preserves the efficiency of the digestive processes. This, in turn, contributes to maintenance of health, improves reproductive ability, increases life expectation, and substantially improves the patient's quality of life.

Zoo veterinarians deal with several taxonomic groups and need to be prepared to manage oral problems in a wide range of species. It is important to acquire scientific knowledge of the anatomical and physiological aspects of the oral cavity of all probable patients.

BASIC ANATOMY OF THE TOOTH

Following is a brief description of the dental organ of carnivores and primates, which includes the tooth itself and its supportive structures, the periodontium. The tooth is composed of enamel (97% inorganic matter), dentine (75–80% inorganic matter) and cementum (50% inorganic matter). Enamel is produced by cells called ameloblast*s*, which die just after the production ends. The dentine, the largest volume of the tooth, is produced by the odontoblasts, inside the pulp cavity. Cementum covers the tooth root, but in some species, like horses and ruminants, cementum is also visible on the crown. In normal carnivores and primates, only the enamel is visible, in the crown.

The tooth is fixed in the mandible and maxilla by four structures that comprise the periodontium: (gingiva, periodontal ligament, cementum, and alveolar bone). The gingiva covers the other three supportive periodontal structures. The periodontal ligament fixes the tooth in the alveolus through fibers that extend from the cementum to the bone. The space where the ligament lies is 0.1–0.3 mm wide. The gingiva attaches to both the bone and the tooth. A junctional epithelium is attached to the tooth by desmosomes, and apically gingival connective fibers attach to the periodontal ligament. Above the junctional epithelium (coronally) there is a virtual space called the gingival sulcus. In the gingiva, a complex of immune system cells and substances are constantly inhibiting invaders (dental or bacterial plaque). Thus, there is a constant production of gingival fluid by the gingival tissue into the sulcus. The anatomy is similar for many species, but one expects to find less cancellous bone in the alveoli and stronger attachment in the periodontium of carnivores than in other species. The depth of the gingival sulcus varies according to the species, and studies are needed in order to accurately establish the differences in these anatomic features.

Inside the tooth is the pulp, a soft supportive tissue with nerves, blood and lymphatic vessels, immune system cells, and an important layer of cells, the odontoblasts, close to the dentinal wall. Their function is the production of dentine throughout the animal's life. The first dentine produced by odontoblasts in the young animal or fetus is the primary dentine. When the tooth erupts in the mouth of a carnivore, its root is not yet fully formed, and the dentinal wall is very thin. After a few months, the root is formed, and the tip of the root (the apex) is closed (apexogenesis). The time of closure for many species remains to be established. In domestic cats, the apex closes between 9 and 11 months of age.[5] The pulp is nourished by vessels penetrating through the apex, from the mandibular canal artery and maxillary alveolar arteries. Nerve, veins, and lymphatics reach the root through the apex also.

In domestic carnivores, such as the dog and cat, the apex has dozens of perforations where the vessels and nerves penetrate rather than a single apical opening, which is typical of primates. The apical structure of most wild animals is still unknown. In humans and some primates there are one, two, or three main perforations (foramina).[1] This anatomic characteristic is important in performing a root canal when a tooth is to be preserved. For many species, the anatomy and physiology of the dentinal-pulp complex need to be studied in order to understand its morphology and thus better treat injured teeth.

After the tooth elongates maximally (apical-coronal distance), with apexogenesis complete, the odontoblasts continue to produce dentine, now called secondary dentin. The cells produce dentine around their cytoplasmic extensions, forming a tunnel called odontoblastic tubule. There is an average of 45,000 tubules per square millimeter in the dentine. Extending from the enamel in the crown to the cementum in the root, the cellular body of the odontoblast lies in the pulp, besides exclusive pain-sensitive nerve endings. With aging, dentine becomes thicker and the pulp cavity narrows, thus strengthening the tooth. Because of its inelastic walls, injuries to the pulp results in irreversible inflammation, with edema and compression of cells and vessels. Pain is likely to occur, and the animal refuses to eat.

TYPES OF DENTITION

Following is a description of the basic types of dentitions.[2] Some teeth grow continuously during life, and other teeth reach their maximal length and stop elongating. The elodonts (from Latin *elongare,* elongate and Greek *odon* and odous, tooth) have teeth that grow continuously in "height" (apical-coronal length) as long as the animal lives. Examples are the incisor teeth of rodents and lagomorphs, the tusks (incisors) of the elephants, and the caudal teeth of the aardvark and some xenarthrans. The apex of these teeth are in continuous activity and therefore are always "opened," producing dentinal structure in coronal direction and elongating the teeth. The crown is worn slowly by mastication, which necessitates good occlusion for even physiological wear of the crown. In the presence of any abnormality that interferes with this wear, the tooth may become elongated and injure soft and hard tissues, producing pain, anorexia, and eventually death of the animal.

Anelodont teeth do not continue to grow in height after reaching the maximum genetically coded extension. This is the case in carnivores, primates, and herbivores. There is a clear line dividing crown and root. There are animals with clearly larger crowns than roots, and vice versa. The animals considered hypsodonts (from Greek *hipsos,* height) have high teeth, with a disproportionate ratio between root and crown, since the crowns are long. Equines and ruminants have this kind of teeth. It is important to highlight that these teeth are in constant eruption; that is, after reaching maximum height they continue to erupt toward the table surface of the tooth, but do not increase in height because they are worn off. In older animals the tooth may be lost since it no longer has a root attachment.

Animal teeth may also be separated into homodonts and heterodonts. The teeth of homodonts (from Greek *homos,* equal or similar) are all similar. This group can be divided into two main subgroups, according to the eruption of the sets of teeth: monophyodonts and polyphyodonts. The toothed whales, dolphins, and porpoises (Odontoceti) are monophyodonts, that is, once a tooth is lost, there is no replacement of it. Polyphyodonts have an endless succession of teeth. When a tooth is lost, another one takes its place. Sharks, crocodilians, and snakes are included in this group.

The heterodonts (from Greek *heteros,* different) have anatomically different teeth in the same set of dentition. There is a clear distinction between incisors, canines, premolars, and molars. In terms of dental eruption, some heterodonts have grouped eruptions (carnivores, primates, herbivores, and ruminants), where the eruption occurs in a lateral pattern (side by side), and others have sequential eruptions (elephants, in their posterior teeth, and macropods), where eruption occurs in a caudal-rostral pattern (horizontally). Both ways of eruption may occur in the same species. All elodonts and anelodonts are included in the heterodont group.

ORAL PROBLEMS IN CAPTIVE REPTILES

The main diseases observed in the oral cavity of reptiles are ulcerative stomatitis and hypovitaminosis A.

ULCERATIVE STOMATITIS Snakes are most often affected by ulcerative stomatitis, but the problem may also be observed in lizards and chelonians. The most common etiologic agents are gram-negative bacteria, with a predominance of *Aeromonas hydrophilia* (*Pseudomonas* and *Klebsiella* are less frequently identified).

Among the most common predisposing factors are chronic stress, malnutrition (calcium, protein, and vitamin A and C deficiencies), and trauma as a result of management problems (capture, transport, inadequate enclosures, and forced feeding).

Warning signs include inappetence and salivation. Hyperemia, ulceration, and necrosis in the oral mucosa, as well as hemorrhage and presence of caseous exudate (typical of reptiles), are seen in the mouth. In the majority of snakes the normal oral mucosa has a white or whitish pink color, making it easy to identify hyperemia.

Pathophysiologically, the ulcerative lesions of the oral mucosa become necrotic, with gingival destruction, ultimately spreading to osseous tissues. It causes osteomyelitis and subsequent dental exfoliation. Cases of ulcerative stomatitis that are not appropriately treated generally progress to septicemia and lead to death.

The main method of avoiding ulcerative stomatitis in captive reptiles is to establish judicious prophylactic protocols, aiming at eliminating the predisposing factors. Affected animals should be isolated and housed under optimal conditions of environmental temperature, improving the action of organic mechanisms of defense as well as increasing efficiency of the medical treatment. The oral lesions should be debrided (removal of exudates and necrotic material) and cleaned with a solution of organic iodine (PVPI) which may be mixed in equal parts with 3% or 5% hydrogen peroxide. Another important factor for the patient's recovery is to establish correct antibiotic therapy, based on sensitivity of the pathogen. Traditionally, recommended drugs are gentamicin and chloramphenicol, and the authors have had good results with the use of enrofloxacin. Forced feeding may become necessary for snakes.

HYPOVITAMINOSIS A Hypovitaminosis A is frequently observed in turtles. The characteristic clinical signs are conjunctivitis, blepharitis, and palpebral edema due to hyperplasia of the mucin-producing glands. Additional signs include dyspnea, oronasal discharge, and increased predisposition to respiratory infections, as well as excessive growth of the horny tissue of the beak.

Prevention is essential, by supplying sufficient vitamin A in the diet. Besides diet correction, affected reptiles should be medicated with parenteral vitamin A. Excessive growth of the beak may be corrected with dental burs, disks, or saws.

ORAL PROBLEMS IN CAPTIVE WILD BIRDS

The beak is an osseous structure, covered by horny tissue. In addition to bacterial diseases, the oral cavity of birds may be affected by several other diseases including trauma, nutritional disturbances, and the action of parasites, viruses, and fungi. The main problems that directly or indirectly affect the oral cavity and adnexal structures in birds are beak fractures, beak overgrowth, hypovitaminosis A, oral candidiasis, trichomoniasis, avian pox, and the beak and feather syndrome. See the section on psittacine birds for details on these diseases.

DENTISTRY IN SOUTH AMERICAN WILD MAMMALS

Animal handlers are usually first to notice abnormalities that may be related to dental problems. They should be trained to recognize the early signs, such as any sudden changes in prehension and mastication of food, as well as changes in the general condition and behavior of the animals. Some behavioral changes that are highly suggestive of dental disturbances in wild mammals include anorexia and weight loss, selective appetite (preference for softer food), abnormal aggressive behavior (due to dental pain), anguish, acute reactions to the ingestion of cold water, pawing the ground, pawing or scratching the face, head tilting and rubbing against obstacles, and uncommon manner of drinking and eating.

Among the clinical signs that can be noticed at a distance, the most important are abnormal salivation, dental fractures, oral and/or nasal discharge, malocclusion, dental overgrowth, and swelling on the head.

Once an animal is examined, all the observed changes should be recorded in an appropriate clinical record, and a therapeutic schedule should be planned. The establishment of indicated therapeutic procedures should be the briefest possible.

Oral diseases that affect zoo mammals are usually the result of one or more of the following factors: trauma, inadequate diet, action of pathogenic microorganisms that destroy calcified tissues and cause inflammation in soft tissues, changes in development of calcified tissues, malocclusion, and dental degeneration (attrition, abrasion, erosion, and resorption).

Dental trauma is a common problem in zoo mammals, leading to pulp exposure. Carnivores and primates are most often affected. Common causes are biting metal cage bars, wire netting or concrete posts, fighting with enclosure mates and deleterious human action (teeth clipping, commonly practiced by poachers, illegal animal traders, and personnel of circuses).

Dentistry is a veterinary specialty that has undergone a sound development in the last few years. Among the South American wild animals, rodents, lagomorphs, camelids, primates, and carnivores require special dental care.

Dentistry in South American Rodents and Lagomorphs

Canine teeth are absent in rodents and lagomorphs. The incisor teeth grow continuously and are constantly worn off by counterabrasion, which keeps edges permanently sharp. There is a gap called the diastema between incisors and premolars. The temporomandibular joints allow wide lateral movement of the jaws.

Malocclusion is the most common dental problem in rodents and lagomorphs and its cause may be traumatic, functional, or congenital. One incisor tooth may overgrow because of a fracture of the opposite tooth, preventing the normal wear. The overgrowth of incisor teeth may also be caused by insufficient masticatory exercise (inadequate diet, absence of suitable chewing sites).

Affected teeth must be cut in order to improve alignment. In smaller animals it is possible to use plier cutters to trim part of the crown. In larger animals such as agoutis, pacas, and capybaras, the use of dental burs, disks, or saws is indicated. Sometimes pulp capping and crown restoration may be necessary.

Dentistry in South American Camelids

South American camelids have four canine teeth and two caniniform upper incisors. These six teeth are razor sharp and males use them to inflict severe injuries to other males. Extraction of these teeth is not recommended because of the extensive curved root. Rather, the crown may be amputated using obstetrical wire or a hobby drill saw. Malocclusion of the incisors with the dental pad results in elongation of the incisors, which may require partial amputation.

Dentistry in Wild Carnivores and Primates

In primates the root apex has a single wide apical foramen, whereas carnivore's teeth have many thin foramina.[1] Chemical restraint is essential for the accomplishment of any dentistry procedure in primates or wild carnivores. See appropriate chapters for details of immobilization and anesthesia.

Antibiotic therapy is an important procedure, before and after the majority of dental interventions. It controls primary infection and prevents secondary problems of cellulitis, arthritis, endocarditis, glomerulonephritis, and other infections. Because of the difficulties inherent in sequential administration of oral or injectable medication, some veterinarians prefer to give a single dose of an injectable antibiotic to inhibit the likely bacteremia that occurs during treatment. The preferred option in Brazilian zoos seems to be a combination of procaine penicillin G and benzathine penicillin, commonly used in cattle. Other antibiotics include a combination of metronidazole and amoxicillin/clavulanate, which is recommended against gram-positive agents, and the combination of metronidazole and enrofloxacin for gram-negative organisms.

ORAL DISEASES IN SOUTH AMERICAN PRIMATES

Diseases that may be encountered include fibrous osteodystrophy, herpesvirus infection, traumatic pulp exposure, periodontal disease, and dental caries. See other chapters for details of some of these conditions.

HERPESVIROSIS Human herpesvirus (*Herpesvirus hominis, Herpesvirus simplex*) causes a severe disease in marmosets (*Callithrix* spp.) and owl monkeys (*Aotus trivirgatus*). Transmission occurs by contact with humans carrying active lesions. The patients present ulcerations in the oral mucosa, conjunctivitis, and respiratory disease, which may progress to a severe neurological disorder. Death occurs in 2–5 days. Prevention is important, and the goal is to avoid contact of the monkeys with infected humans.[3,4] Herpesvirus T (*Herpesvirus tamarinus*) causes a similar clinical disease in marmosets (*Callithrix* spp.), owl monkeys (*Aotus trivirgatus*), and titi monkeys (*Callicebus* spp.). Transmission is by contact with contaminated squirrel monkeys (*Saimiri sciureus*) or spider monkeys (*Ateles* spp.), which are the natural hosts.[3]

TRAUMATIC PULP EXPOSURE The common causes of pulp exposure in primates are accidental fractures and intentional deleterious clipping of canine teeth. Bacterial infection of exposed pulp leads to dentoalveolar abscesses. In the upper arcade the process may result in a malar sinusal fistulae with entropion as a sequel. In the lower arcade it may lead to mandibular fistulae.

PERIODONTAL DISEASE Localized periodontal disease is common in almost all species of captive South American monkeys. The generalized form is most frequent in spider monkeys (*Ateles* spp.).

DENTAL CARIES Demineralization of enamel and dentine due to the action of bacteria and its acid metabolites (dental caries) is a common disease, especially in aged monkeys. The high prevalence is a result of inadequate diets and anatomical characteristics of molar and premolar teeth.

ORAL DISEASES IN SOUTH AMERICAN CARNIVORES

The key to minimizing oral disease is an adequate diet. Evaluate diets offered to animals with the goal of duplicating natural diets and dietary habits as far as possible. Capture of prey (predation), food processing (biting, tearing, lacerating, chewing, gnawing), fighting, intimidation, aiding copulation (Felidae), offspring transport, and hair coat cleaning are the most important natural functions of a wild carnivore's teeth, and these need to be preserved in captivity.

PERIODONTAL DISEASE All the taxonomic groups of captive South American carnivores have a high prevalence of periodontal disease. They are predisposed especially by inadequate diets, that do not provide chewing exercise. Prevention of this important clinical problem is a constant challenge to zoo veterinarians, who are responsible for evaluating and correcting diets as well as periodically conducting examinations and treating disorders.

DENTAL CARIES The prevalence of dental caries in wild carnivores is low, when compared with primates. Factors that favor this low occurrence include low carbohydrate diets, low salivary urea levels, low salivary pH, immune factors, and anatomical characteristics.

DENTAL ABRASION Constant, obsessive, and generally aggressive biting of metallic bars or wire netting of enclosures results in dental abrasions. This behavior is caused by psychological problems related to chronic stress (aggression, frustration, or tedium). The normal dental response to abrasion is the production of reparative dentine. If constant abrasion overcomes the ability to repair damage, exposure of the pulp occurs, requiring veterinary restorative procedures (generally including endodontic treatment).

ODONTOCLASTIC RESORPTIVE LESIONS IN FELIDS Noncarious resorptive lesions affect the dental neck of domestic and wild felids. The authors have observed such lesions in jaguars, pumas, and ocelots. Pathogenesis is not well understood. Patients show evident signs of discomfort or pain when eating or drinking (especially cold water), and may become inappetent or anorexic. In the early stages of disease the restoration with glass ionomer cement and composite resins has had a reasonable success rate.

SELECTED DENTISTRY TECHNIQUES IN SOUTH AMERICAN WILD CARNIVORES AND PRIMATES

Veterinary dentistry applied to wild mammals include periodontics, exodontics, endodontics, and restorative dentistry.

Periodontics

Periodontal disease is the most common oral disease in wild carnivores and primates. Microorganism growth is the determining factor in triggering and developing periodontal disease. There are, however, predisposing and modifiing factors that affect the course of periodontal disease, such as inadequate diet, dental and periodontal abnormalities, and immune disturbances. Trauma, malocclusion, persistence of deciduous teeth, dental crowding and rotation, rough dental surfaces, and some systemic diseases may contribute to the accumulation of bacterial plaque. Dental plaque locates primarily in the gingival sulcus and leads to such reactions in oral tissues as gingivitis and/or periodontitis. It causes lesions in periodontal ligaments and alveolar bone. At the same time mineral salts may precipitate, forming dental calculus over the surface of the tooth, supra- and subgingivally. Gingival retraction or hypertrophy may occur. Besides the local infection, bacteremia may cause inflammation in other organs, possibly leading to glomerulonephritis, endocarditis, or meningitis.

A recommended periodontal therapeutic schedule follows:

1. Removal of supragingival calculus.
2. Removal of subgingival calculus and curetting the gingival sulcus, removing necrotic tissues and debris.
3. Scaling and root planing of the root cementum.
4. Gingivectomy (in presence of gingival hypertrophy or hyperplasia).
5. Irrigation with antiseptic solution of 0.12% chlorhexidine.
6. Polishing with prophylactic fluorinated or pumice paste.

Exodontics

It is important to understand that tooth extraction should be avoided. Its necessity is reduced as conservative techniques of maintaining dental health are developed. Tooth extraction causes resorption and remodeling of the osseous support structures, making the cranium more fragile. Whenever possible, dentition should be preserved to maintain the integrity of the masticatory apparatus and assure appropriate food processing. Indications of necessary extraction include severe periodontal disease, periapical abscesses (with or without fistulae), and dental fractures with severe pulpar necrosis. The basic procedures to extract any tooth involve the following steps:

1. Sindesmotomy (separate tooth from gingiva).
2. Luxation (separate tooth from alveolus).
3. Avulsion with forceps.
4. Alveolar curettage, whenever contamination is present (severe periodontitis or apical abscesses).
5. Hemostasis (direct compression, adrenaline, and/or fibrin sponges, when necessary).
6. Suturing of the gingiva should be done only in the absence of contamination (a rare situation).

There are special exodontic conditions that are challenging to clinicians. They are the extraction of canine, carnassial, and ankylosed teeth. In the case of canine and carnassial teeth (fourth premolar), extractions are indicated in some cases of severe periodontal disease, dental fractures with severe pulpar necrosis, and some cases of apical abscesses (with or without fistula). Ankylosis is caused by severe inflammatory reactions that induce alveolar bone sclerosis and consequent fusion between bone and dental root. The occurrence of oronasal fistulae is a frequent complication after extraction of canine teeth. It generally occurs after extracting teeth affected by apical abscesses, when destruction of the osseous wall that separates the dental alveolus and the nasal cavity has occurred. Clinical signs include sneezing and mucopurulent or bloody nasal discharge. The therapeutic schedule includes alveolar curetting, resection of the gingival borders, and repairing the defect by suturing a gingival and/or palatal flap over the cavity. The same surgical techniques indicated for domestic dogs may be used in all species of South American wild carnivores.

Endodontics

The beginning of any endodontic treatment is the partial or complete removal of the dental pulp. Necrosis of pulp tissue may result from dental trauma or degenerative processes. The necrotic process is usually irreversible and imposes the therapeutic removal of the necrotic and infected tissue. The objective of endodontic treatment is to avoid extraction of problematic teeth, preserving the dentition. Don't perform endodontics on fractured deciduous teeth, which should be extracted to avoid complications to permanent dentition. Endodontics is sometimes used to disarm aggressive animals. The indication of endodontic treatment should be based on physical examination of the oral cavity, with careful visual and instrument evaluation. This examination should be followed by radiographic evaluation, which is useful in defining problems and following the treatment results. The most important radiographic evidence of endodontic involvement is the presence of a radiolucent area around the root apex or periapical area, due to bone resorption. The root canal therapeutic schedule follows these steps:

1. Diagnostic radiographs.
2. Access to the pulp and the root canal with spherical bur.
3. Tooth measurement with endodontic rules or files.
4. Pulp or necrotic tissue removal with barbed broaches.
5. Disinfection of the root canal (flushing with antiseptic solution: 1–5% sodium hypochlorite).
6. Root canal instrumentation with endodontic files.
7. Flushing the root canal with antiseptic solution.
8. Drying with paper cones or pipe cleaners and compressed air.
9. Obturation of the root canal (cements and gutta-percha).
10. Crown restoration with the chosen material (preferably silver amalgam, especially for large carnivores).
11. Follow-up radiographs.

Endodontic files and broaches are special tools designed for pulp tissue removal and root canal instrumentation. Human files have a 31 mm maximum length, and the files designed for domestic dogs are 60 mm in length. There are now files 120 mm in length designed for large zoo carnivores. Even so, because of the length or width of the root canal or the impossibility of obtaining appropriate equipment, it may be necessary to improvise endodontic instruments. It is possible to make files from surgical-wire saws [gigli-wire saws]. Actually, any instrument that facilitates the cleaning of the internal walls of the root canal by filling or curetting may be used. During biomechanic preparation, thorough flushing with antiseptic solution such as 1% sodium hypochlorite is recommended. After instrumentation, the root canal should be dried and entirely obturated with sealing paste and gutta-percha cones or sticks. Several sealing medications are commercially available, but the authors' preference is a paste made by mixing liquid eugenol and powdered zinc oxide. The sealing paste is introduced within the root canal through a special blunt needle or a small caliber catheter, connected to a disposable syringe. Better obturation and higher resistance are obtained when using gutta-percha cones or sticks soaked in the sealing paste and pressed within the root canal by using pluggers or spreaders. Gutta-percha is a strong rubberlike glutinous bacteriostatic substance of vegetable origin that can be acquired in the form of cones (same length and caliber as veterinary or human endodontic files) or in sticks measuring 10 cm in length and about 0.4 mm in diameter. These sticks are indicated for larger carnivores.

Restorative Dentistry

The primary objective of restorative dental procedures is to substitute for lost or bur-removed dental tissues. These procedures are suitable for crown restoration after endodontic treatment of fractured teeth, restoration of resorptive lesions, restoration of caries, and restoration of defects of the enamel (erosions, abrasions, hypoplasia). In wild animals rarely is a tooth reconstructed to its original form (cosmetic restoration), rather it is more important to produce a functional repair and protection of the remaining structure. The treatment of deep caries follows the same pattern. There are several restorative materials that can be used, according to commercial availability and to the veterinarian's preference. The most common are silver amalgam, composite resins, and glass ionomer. Silver amalgam is an alloy of metal powder and mercury. It is the most-used restorative material in wild animals, due to its low cost, easy manipulation, biocompatibility, resistance, and durability. Because it does not adhere to the tooth, it is necessary to drill a retentive cavity with dental burs.

With composite resin restorations, acid conditioning (etching) over the enamel is necessary in order to obtain micromechanical retention of the resin to the tooth. The method is to apply acids, in general orthophosphoric acid (that etches the hydroxiapatite prism) over the area to be restored. In addition, before application of resin, a bonding agent, which is a fluid resin capable of penetrating the etched areas, must be applied. There are commercially available autopolymerizing resins (chemical polymerization) and photopolymerizing resins (that require special equipment, but are easily manipulated).

Glass ionomer is a restorative material for repairing osteoclastic resorptive lesions that affect the dental neck of felids. Nevertheless, recent studies indicated that these lesions continue to develop beneath restorations.[6] The advantage of this material is direct bonding to the enamel and dentine, reducing the necessity of retention sculpture or acid etching. It permits fluoride ions to permeate the surrounding dental structure, inhibiting secondary infiltration and reducing painful sensitivity reactions. Glass ionomer is less sensitive than composite resins to inhibition of adherence by moisture, a characteristic that is important when dealing with resorptive neck lesions, where the operative field is frequently wet with blood. Its disadvantages are less resistance than composite resins to abrasion and to occlusion and articulation forces. To improve resistance, some veterinarians combine glass ionomer and composite resins in a sandwich technique, in which a basal layer of glass ionomer is applied directly over the area to be treated, followed by acid etching of the glass ionomer, and finally restoration with composite resin. In this method the glass ionomer links to the dentine, and the composite resin links to the ionomer. It protects the dentine against the corrosive effects of the orthophosphoric acid.

REFERENCES

1. Gioso, M.A. 1994. Avaliação do Método Sinestésico na Obtenção da Extensão do Canal Radicular dos Dentes do Cão [Evaluation of the synesthetic method in measuring the root canal in teeth of dogs]. Masters thesis, Universidade de São Paulo.
2. Kertesz, P. 1993. A Colour Atlas of Veterinary Dentistry and Oral Surgery. London, Wolfe, pp. 215–273.
3. Ott-Joslin, J.E. 1986. Viral diseases in nonhuman primates. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders, pp. 674–697.
4. Pachaly, J.R.; Werner, P.R.; and Diniz, J.M.F. 1991. Infecção natural por *Herpesvirus hominis* em *Callithrix jacchus jacchus* Thomas, 1903—Callithricidae, Primates [Infection by *Herpesvirus hominis* in *Callithrix jacchus jacchus*]. A Hora Veterinária 11(61):11–12.
5. Wilson, G. 1999. Timing of closure of the maxillary canine and mandibular first molar teeth of cats. Journal of Veterinary Dentistry 16(1):19–21.
6. Zetner, K. 1992. The influence of dry food and the development of feline neck lesions. Journal of Veterinary Dentistry 9(2):4–6.

39
Ultrasonography in South American Wild Animals

Alessandra Quaggio Augusto

INTRODUCTION

Ultrasonography in zoo animals has become more important in South America during the last few years. It is a safe and noninvasive method of diagnosis that supplies information about internal anatomy, size, and direction of structures and organs. Ultrasonographic imaging techniques use high-frequency sound waves in the range of 2–10 MHz, which are generated and transmitted by a transducer applied on or into the patient's body. The waves are absorbed and reflected in varying degrees by different organs, being captured again by the transducer. The transducer frequency must be selected by the operator according to the anatomic region to be examined and the animal's size. Higher frequencies provide better definition, but lesser penetration. Thus, high frequency is indicated for small animals and examination of superficial structures.[17,10] Accurate interpretation depends directly on the differentiation of normal and abnormal anatomy and the operator's experience.

Each organ has a specific echo pattern that is dependent on its structure. Basically, ultrasonography can identify changes in an organ's size, parenchymal and vascular structures, and differences between homogeneous liquids and heterogeneous structures. Bones and gas-filled organs reflect the sound waves and impede visualization of underlying structures. In cases of fluid collection in the abdomen, ultrasonography is a diagnostic method of great value because, unlike x-rays, liquid increases the image definition of the abdominal organs (Figure 39.1).

Ultrasonography evaluation may be limited because of the peculiarities of some wild animal species; for example, feathers, shells, plates, fur, avian air sacs, and large body size may block the sound waves. In addition, there are few published works on the ultrasonographic appearance of different organs in a large number of species of wild animals. Reference data in small animal ultrasonography and the experience of the operator aids ultrasound evaluation in wild animals.[13,17,18,20]

For ultrasound examinations the majority of zoo animals require sedation or general anesthesia. A period of fasting is always recommended. However, in some species of birds and reptiles only physical restraint is necessary. Because of the cost and lack of specialization in ultrasonography, the great majority of zoos in South America lack ultrasonography equipment and depend on universities and private clinics for help.

One of the most important applications of ultrasonography in zoos is in reproduction.[10] Ultrasonography provides more accurate determination of gender and sexual maturity than other methods. It is also valuable for evaluation of the reproductive tract and monitoring gestation.[5,10]

This chapter emphasizes several clinical applications and the author's experience in interesting cases with some common animals in South American zoos.

BIRDS

Introduction

The air sacs and the compact intestinal convolution of birds impede transmission of ultrasound waves during transcutaneous ultrasonography. However, in larger avian species with abdominal effusion or increased organ size, ultrasonography provides an excellent opportunity to obtain more information, especially on the internal structure of the organ.

Clinical Application

A great advantage of ultrasonography in birds is for sex identification of monomorphic species. Transcloacal

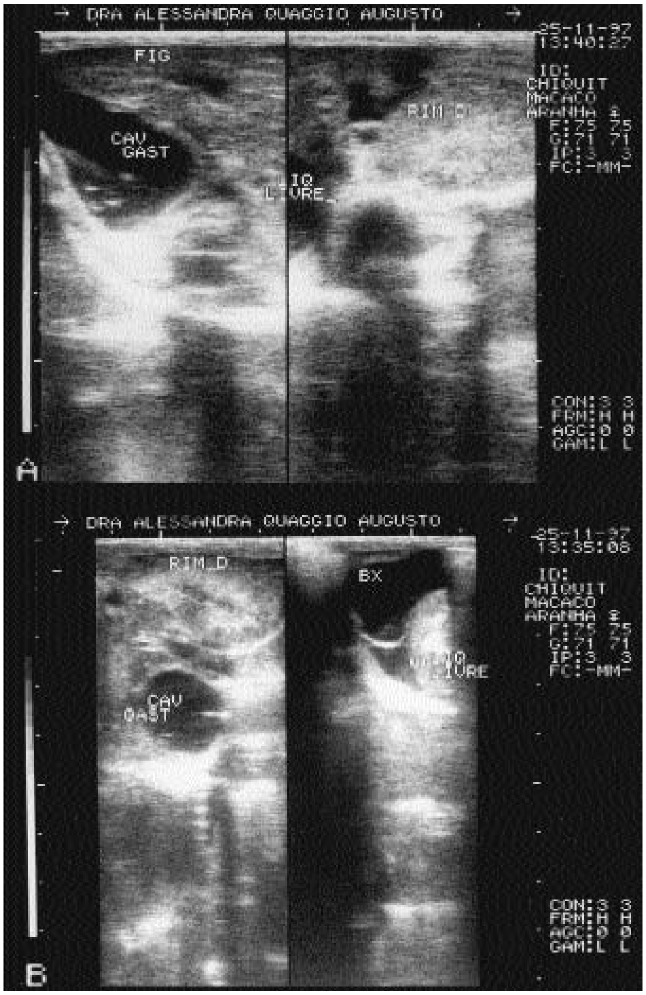

FIGURE 39.1. Sonograms of a spider monkey (*Ateles paniscus*) with ascites. **A and B.** Free abdominal fluid is detected by the presence of anechoic areas (free liquid) separating the various intra-abdominal structures. **A.** Stomach (CAV GAST) containing liquid; liver (FIG). **A and B.** Right kidney (RIM D). **B.** Bladder (BX) is easily recognized.

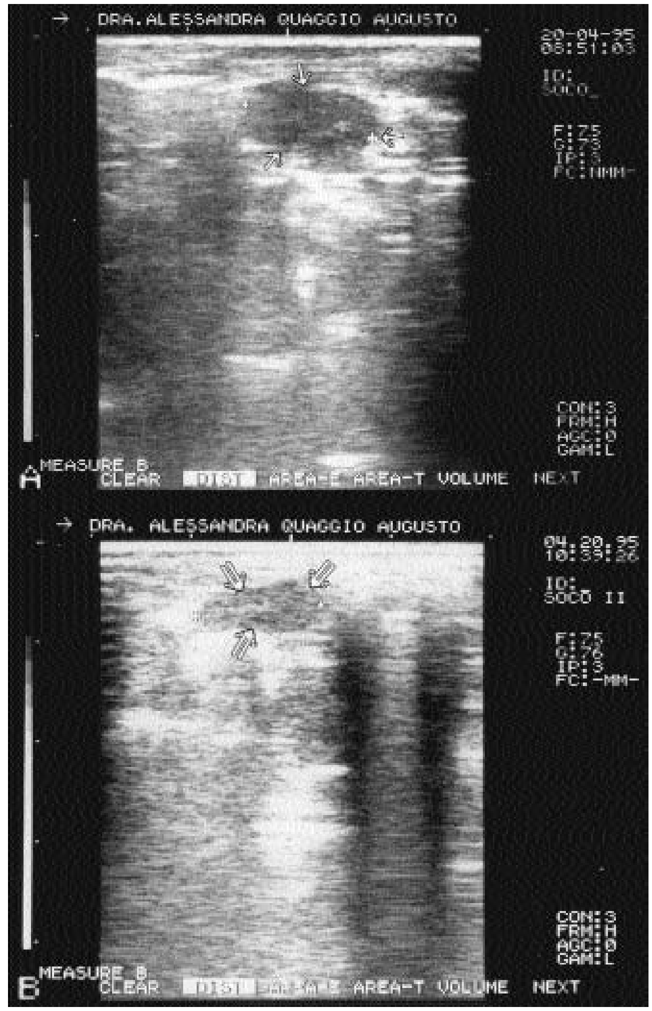

FIGURE 39.2. Sonograms of **(A)** ovary of a female and **(B)** testicles of a male black-crowned night heron (*Nycticorax nycticorax*). **A.** The active ovary (between arrows) has some follicles (small anechoic areas). **B.** The testicles (between arrows) have a homogeneous echo pattern. The diagnosis was confirmed by laparoscopy.

ultrasonography allows good visualization without interference of the air sacs. Unfortunately, this method is seldom used in South America.[11] For some species of birds, using a high-frequency transducer with a small head size, it is possible to find "acoustic windows" for transcutaneous ultrasonographic examination. Differences between ovary and testicles are shown in Figure 39.2.

Ultrasonography is useful for the evaluation of liver diseases in birds, differentiating inflammatory processes from neoplasia. Hepatomegaly in birds is often caused by infectious processes, especially in acute viral infections. The size of the liver varies widely between species. Evidence of hepatomegaly is the presence of liver parenchyma far caudal to the sternum.[20] In Amazon parrots, fatty liver degeneration is often found. The ultrasonographic image is similar to that of mammals and is characterized by increased echogenicity and hepatomegaly. *Aspergillus* spp. are widespread mycotic agents and may cause diffuse liver necrosis. All bird species may be affected, but psittacines are especially susceptible under conditions conducive to stress.[6,8] The ultrasonography examination may show a spotted pattern of the liver parenchyma. Chlamydiosis in parrots may cause severe liver damage.[7] Ultrasonography is of little value in detecting acute or chronic hepatitis, and it is difficult to differentiate between cirrhosis and necrosis.

Examination of the urogenital tract has some limitations, but it is indicated for fertility problem evaluations, abdominal swelling, egg binding, and suspected renal tumors. Normal kidneys are impossible to visualize through transcutaneous ultrasonography because of their position along the vertebral column, and normal ovaries are difficult to identify, especially when they are inactive.[14]

A curiosity of ocular ultrasonography in birds is the ability to visualize the pecten, a small hypoechoic structure emerging from the ocular fundus. Further details may be found in the ophthalmology chapter in this book.[2]

REPTILES

Introduction

Transcutaneous ultrasonography also has limitations in some species of reptiles because of a dense shell or scales. Little data about ultrasonography in South American reptiles are available.

Clinical Application

Ultrasonography is effective in cases of hepatic disease, urinary bladder calculi, coelomic fluid, pregnancy, and cardiac activity in reptiles.[1-16] This method is also valuable for evaluation of reproductive status, pregnancy diagnosis, prediction of parturition date, and identification of prenatal conditions.[15] It is also important for sex determination of monomorphic species of reptiles, however, in some cases the transintestinal method is more effective.

In chelonians it is possible to access some internal organs from the skin between the head and thoracic member and the skin cranial to the pelvic member (mediastinal openings and inguinal openings). The mediastinal and axillary approaches offer good visualization of the heart, liver and gallbladder. The inguinal window allows visualization of the urinary/accessory bladder, kidney, and gonads.[19]

For lizards and snakes, the best ultrasonographic images are obtained from the ventral surface. This examination may reveal all stages of ova development and follicle growth. The gallbladder, in snakes, may be used as a marker. Ovulated follicles and eggs are linearly arranged, occupying almost the entire caudal half of the snake. Examples of ultrasonography in lizards are depicted in Figure 39.3.

The scales of snakes may produce artifacts during ultrasonography examination; however, the use of coupling gel may improve the contact with the transducer and displace air under the scales. Many snakes may be examined without anesthesia. Such an examination may assist in evaluating the coelomic structures, soft tissue masses, cystic structures, coelomic fluid, and assessment of cardiac function.[12] Special care should be taken to avoid mistaking fat bodies for other viscera. Ultrasonography is effective in evaluation of the female reproductive tract.[21]

MAMMALS

Clinical Application

In wild mammals of several species, it is possible to perform ultrasonography as in domestic animals and humans. Abdominal ultrasonography may help detect reproductive disorders, but the anatomic variability of various species of mammals may obstruct recognition of pathological alterations. In some carnivores from South America, *Dioctophyme renale* has been identified in the kidney ultrasonography.

Ovarian cysts may be detected easily by ultrasonography, as may small increases of uterine volume. For example, a little spotted cat (*Leopardus tigrinus)* presented with a vaginal discharge, apathy, and anorexia. During the ultrasound examination, it was possible to observe uterine fluid in moderate quantity, providing the opportunity for early therapy (Figure 39.4).

Ultrasonography may be used to identify fetal death, which is characterized by lack of a fetal heartbeat and loss of fetal integrity. A surgical procedure was indicated in one female mazama deer (*M. gouazoubira)* after an ultrasonography examination indicated a fetal death (Figure 39.5).

Breeding programs may be assisted by ultrasonography for its accuracy in showing the presence of ovarian follicles induced by hormone stimulation in an ocelot (Figure 39.6). Through ultrasonography, it is possible to detect early stages of pregnancy and monitor fetal development, an important diagnostic tool for management and feeding of a pregnant animal. Figure 39.7 shows one example of early pregnancy in an agouti (*Dasyprocta azarae).* In some species it is possible to determine fetal sex; and the stage of pregnancy may be determined by measuring fetal structures and comparing the measurements with those of similar species. There are indirect methods of pregnancy determination, such as estrogen and progesterone metabolites in feces or urine, but they should be specific because of differ-

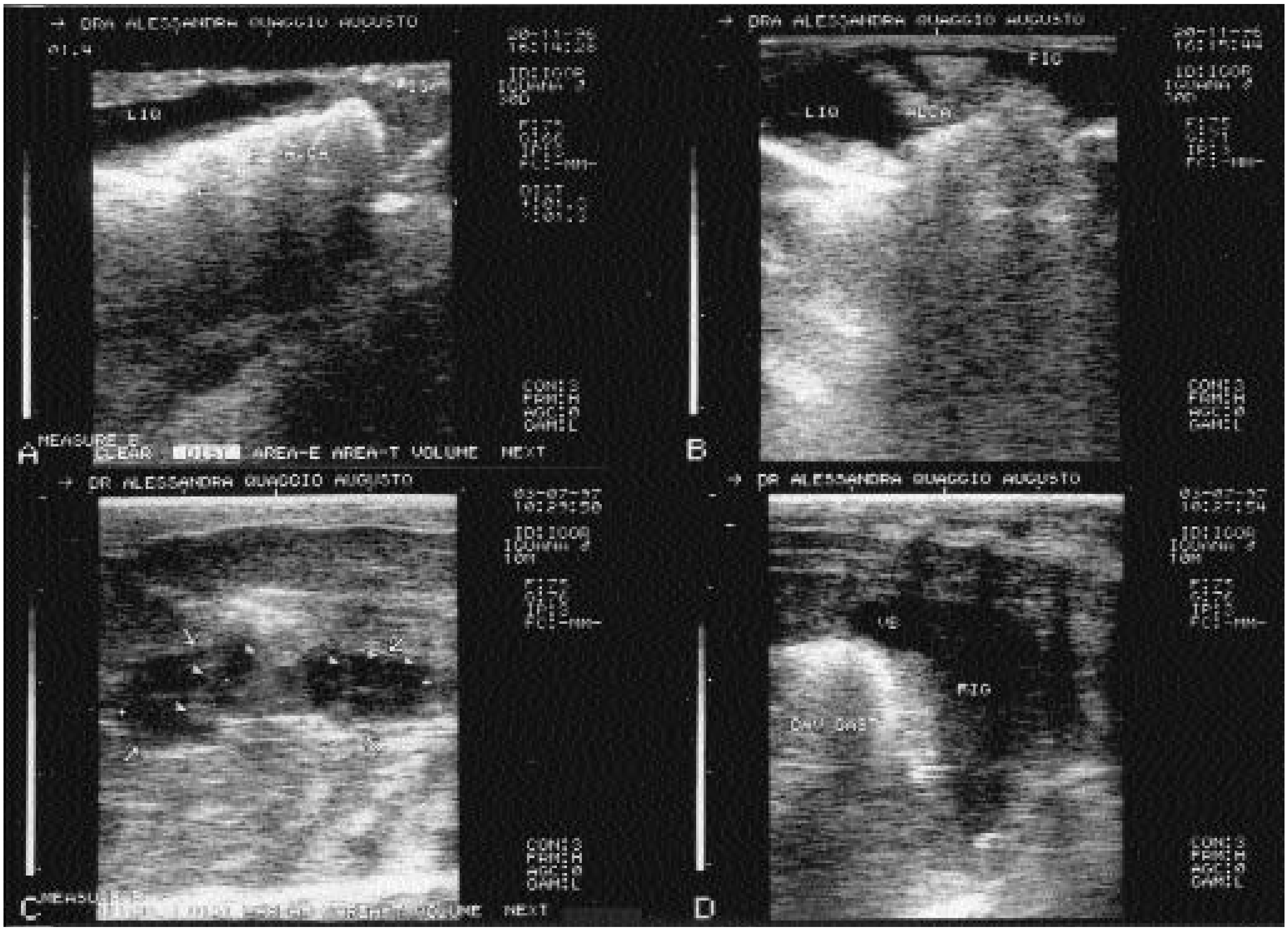

FIGURE 39.3. A and B. Sonograms of the coelomic cavity of a small green iguana (*Iguana iguana*), showing the presence of ascites due to a poor diet: anechoic area (black) is free of coelomic liquid (LIQ); air-filled bowel (ALCA); liver (FIG). **C and D.** Sonograms of a 10-month-old female iguana, showing active ovary: **C.** (between arrows), hypoechoic pattern with several follicles (arrowheads); **D.** stomach (CAV GAST), gallbladder (VB), and liver (FIG).

ences in physiology and metabolism among various species of animals.

In aged mammals, ultrasonography may be helpful in diagnosing abdominal masses. In one 18-year-old cougar (*P. concolor)*, a large cystic mass and few small cysts in the hepatic parenchyma were observed, which proved to be a hepatic adenoma (Figure 39.8).

Another frequent ailment of aged mammals is renal disease, particularly in older zoo felids.[9] Glomerulonephritis and nephrosis have been reported in wild felids. Two examples of renal diseases are shown in Figures 39.9 and 39.10. Diseased kidneys usually show an increasing cortical echotexture, and sometimes hydronephrosis may be present.

Evaluation of endocrine problems and differential diagnosis of distended abdomen are some other applications of ultrasonography examinations. Normal adrenal glands are difficult to visualize by transcutaneous ultrasonography in most zoo and domestic animals. Figure 39.11 shows the slightly enlarged adrenal gland of an ocelot *(Leopardus pardalis)* that was confirmed by fecal hormone analysis. An enlarged abdomen with signs of disease may result from the enlargement of abdominal viscera and intra-abdominal fluid accumulation that is secondary to cardiac, hepatic, or renal diseases, as well as malignant tumors. Some examples are shown in Figures 39.12 and 39.13.

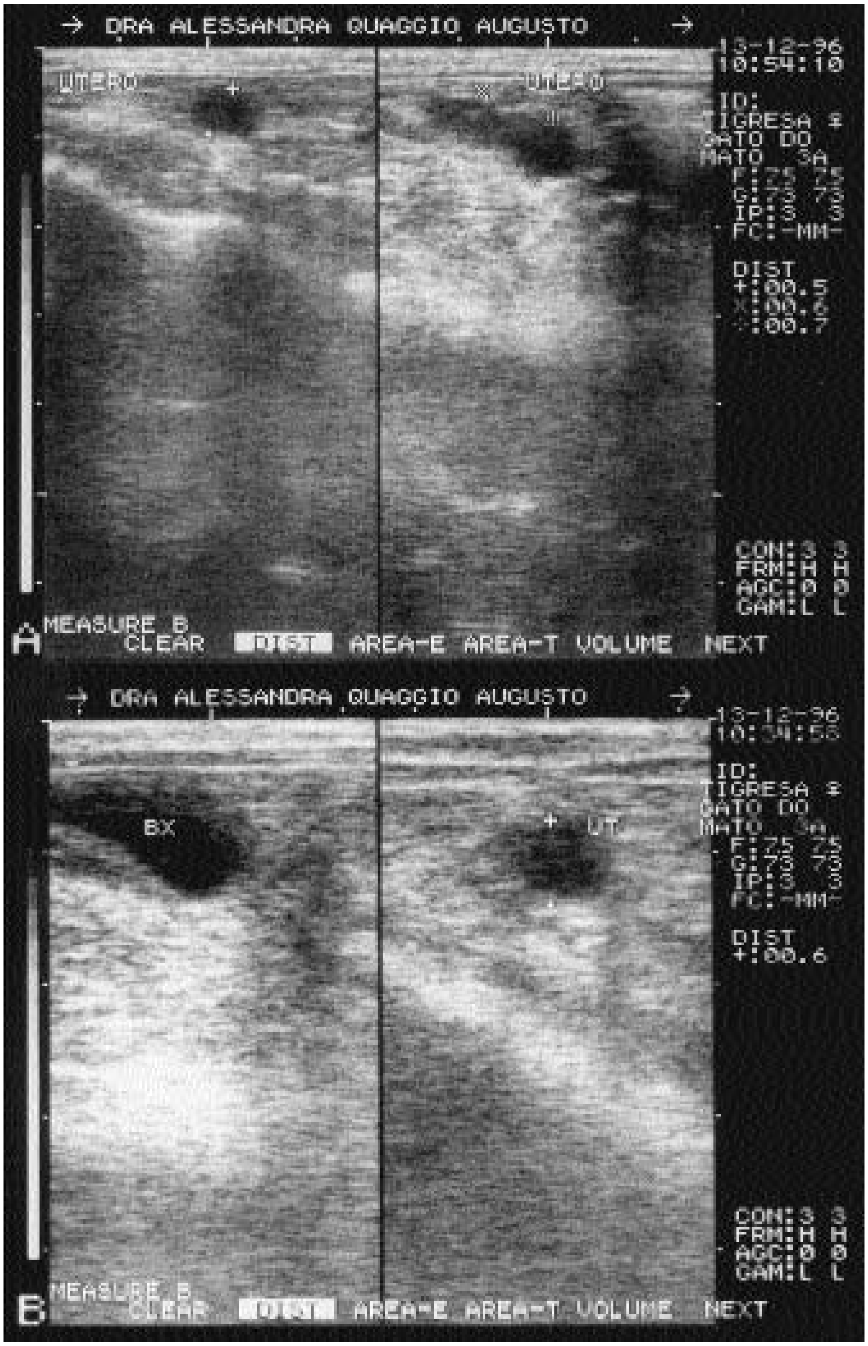

FIGURE 39.4. A and B. Sonograms of the uterus (UTERO/UT) in a little spotted cat (*Leopardus tigrinus*). **A and B.** Slightly enlarged uterine horns containing hypoechoic material and normal bladder (BX). **B.** Clinical symptoms and laboratory exams confirmed pyometra.

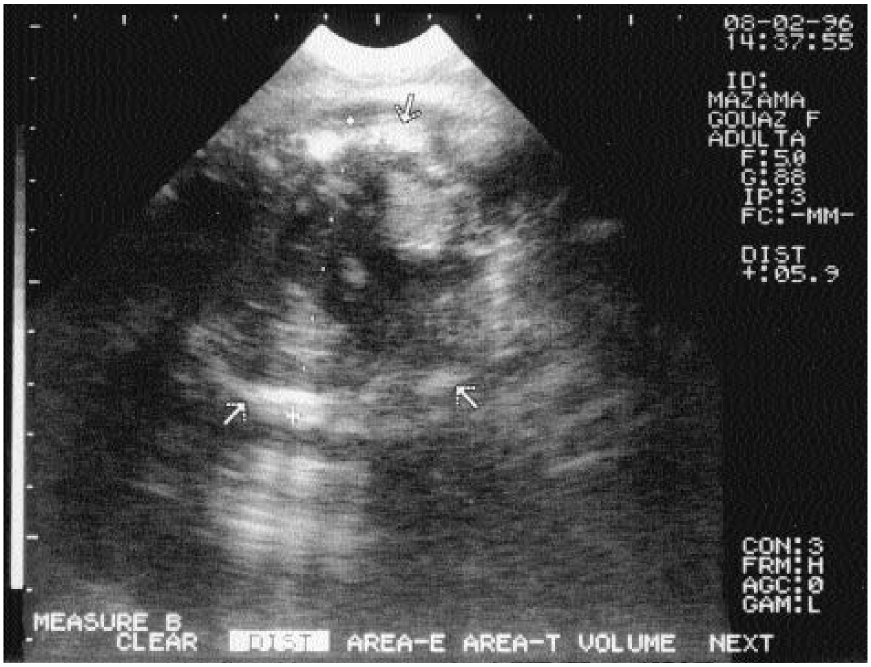

FIGURE 39.5. Sonogram of an adult female mazama deer (*Mazama gouazoubira),* showing fetal death: part of the thorax (between arrows) with loss of fetal integrity.

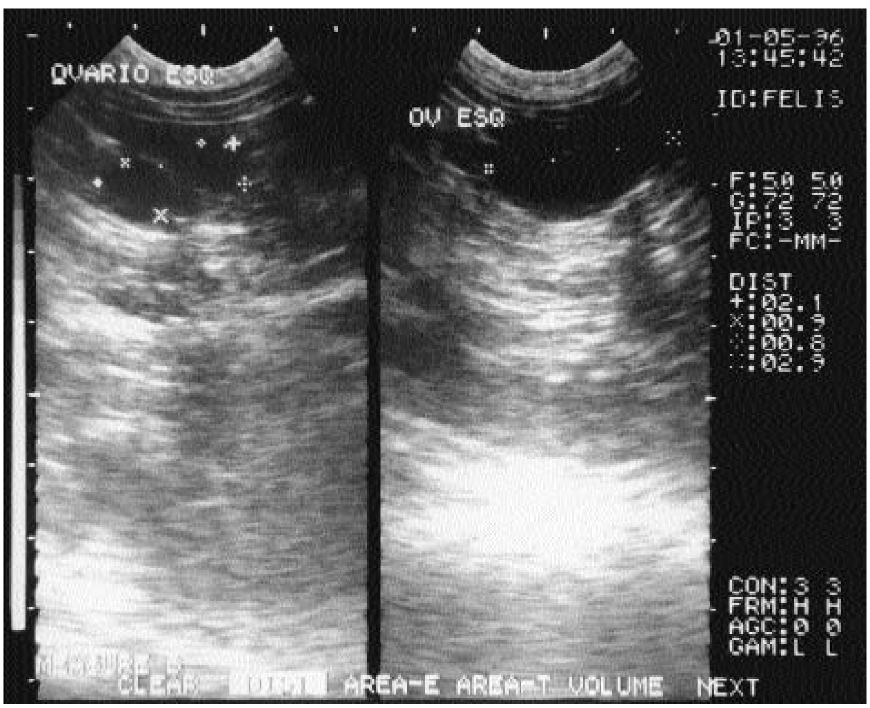

FIGURE 39.6. Sonogram of ovarian follicles induced by hormone stimulation of an adult female ocelot *(Leopardus pardalis):* left ovary (OVARIO ESQ) containing multiple, small anechoic areas (follicles).

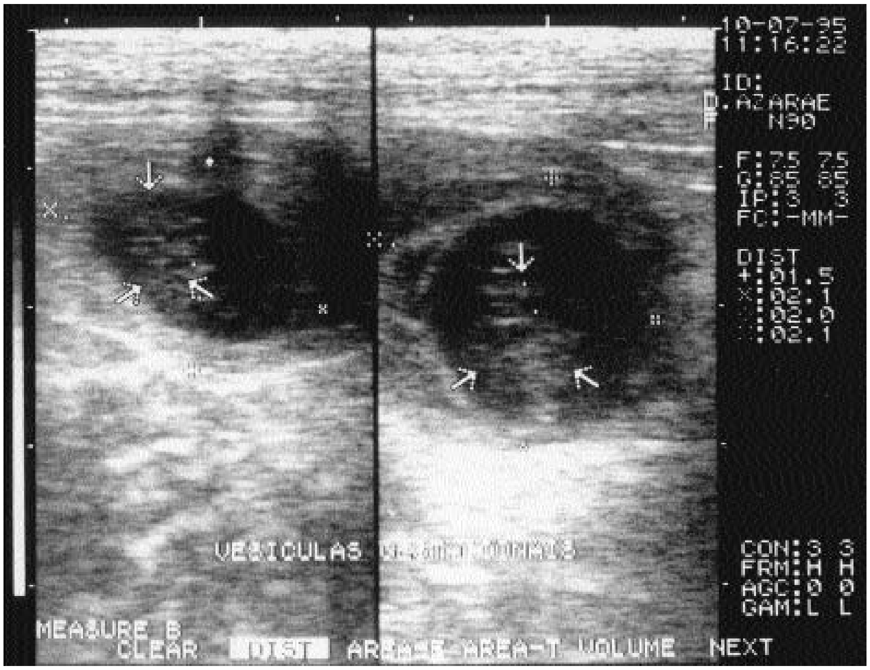

FIGURE 39.7. Sonogram of early pregnancy of an agouti (*Dasyprocta azarae)* by transcutaneous ultrasonography: (between arrows) gestational sac contains the echogenic embryo.

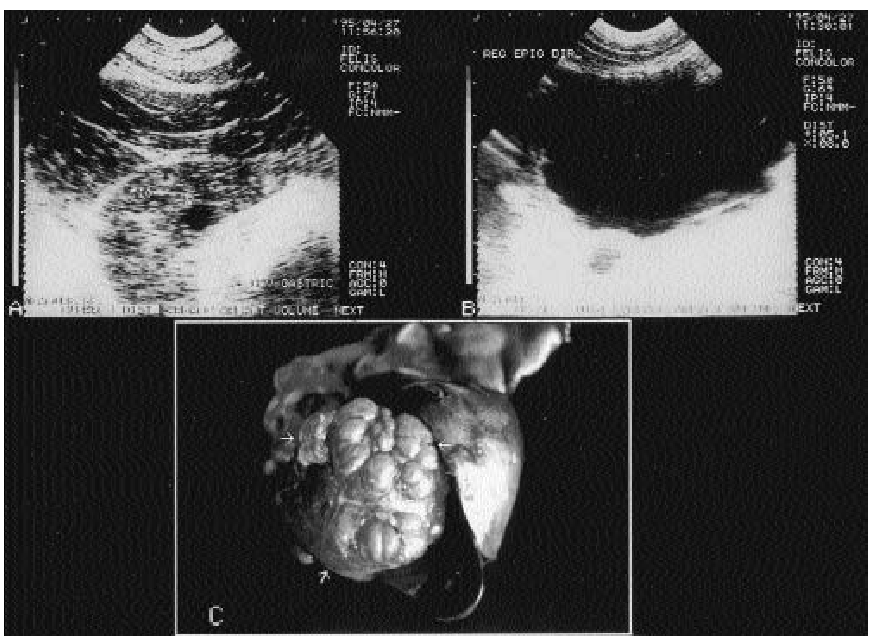

FIGURE 39.8. Hepatic adenoma in an 18-year-old male cougar (*Puma concolor)*. **A.** Small cystic area (white arrow) in one hepatic lobe. **B.** Cystic area (5 cm × 8 cm) with irregular walls and strong distal acoustic enhancement. **C.** Macroscopic appearance of the tumor and the liver parenchyma containing cystic area (between arrows) visualized on **B.**

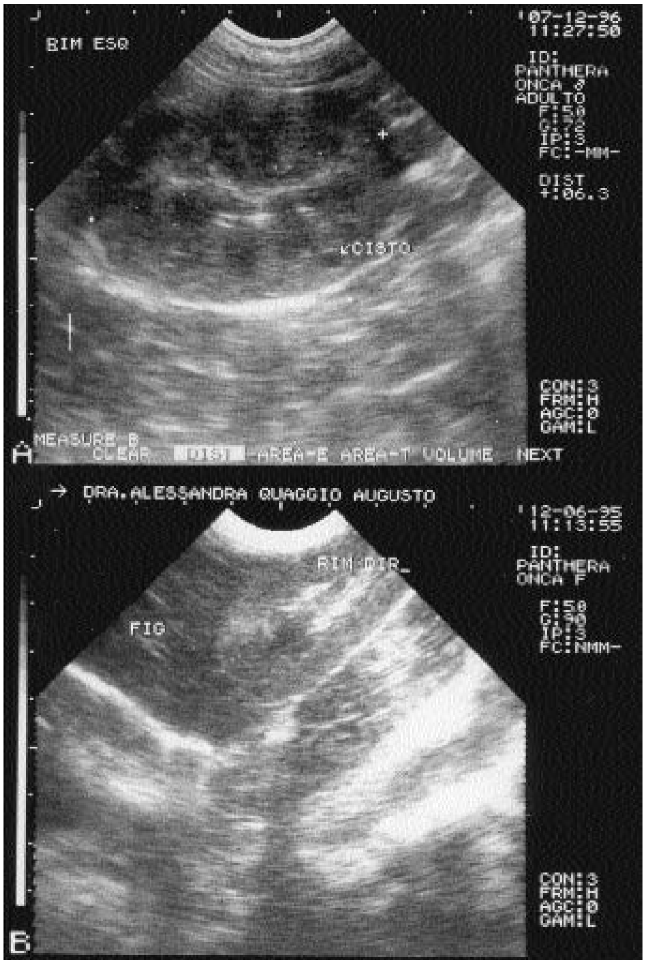

FIGURE 39.9. A and B. Sonomorphologic appearance of the kidneys of two aged jaguars (*Panthera onca).* Increased cortical echogenicity in both kidneys is abnormal, but it is a nonspecific finding. **B.** A slightly increased echogenicity in right kidney (RIM DIR) compared with the liver (FIG) is associated with glomerulonephritis.

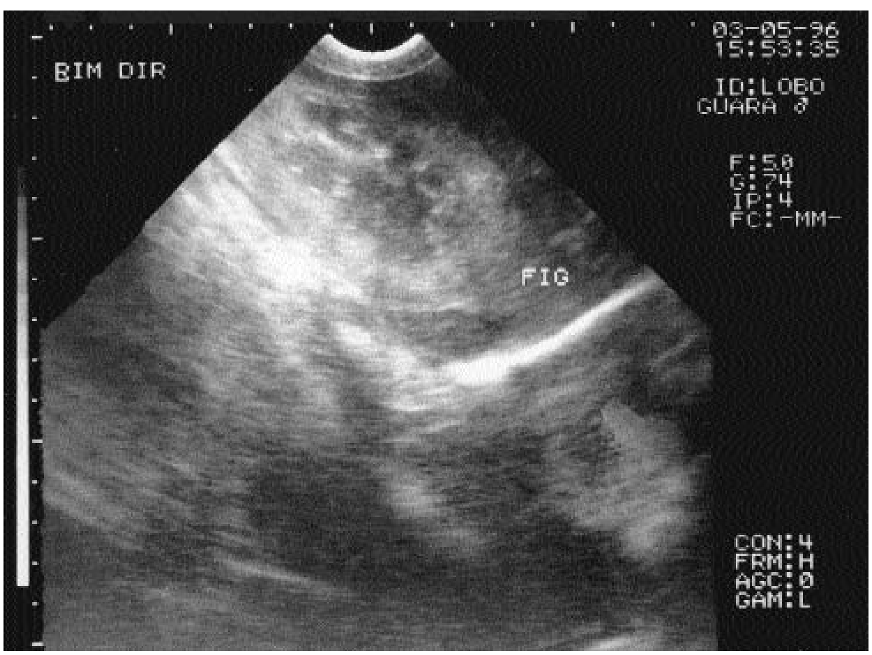

FIGURE 39.10. Sonagram of right kidney (RIM DIR) of a male maned wolf (*Chrysocyon brachyurus),* showing an increased cortical echogenicity. The renal cortical is more echogenic than the parenchyma of normal liver (FIG). Glomerulonephritis was confirmed by laboratory exams.

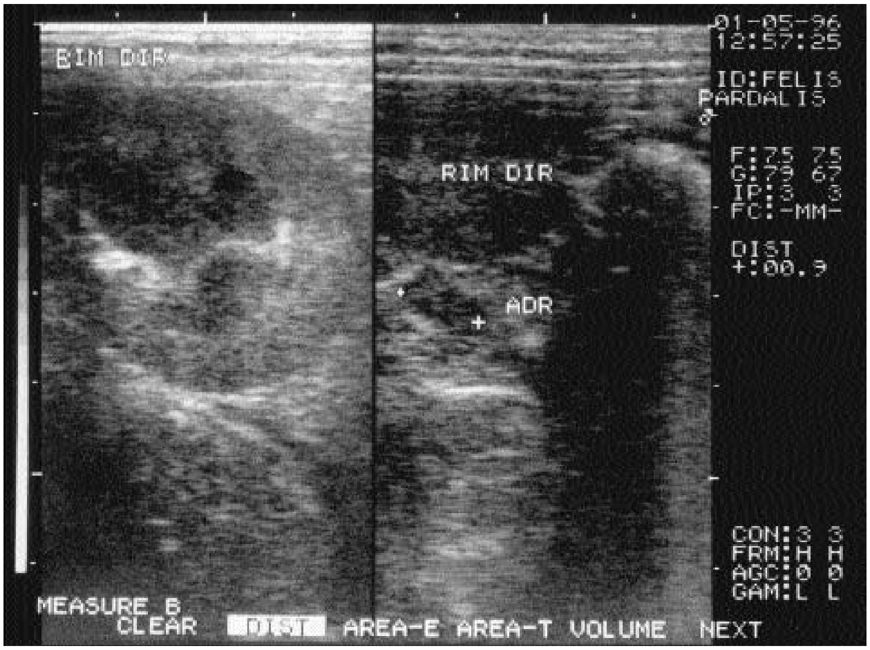

FIGURE 39.11. Sonogram of right kidney (RIM DIR) and right adrenal gland (ADR) of a male ocelot *(Leopardus pardalis)*. Slight enlargement of the adrenal gland was confirmed by fecal hormone analysis.

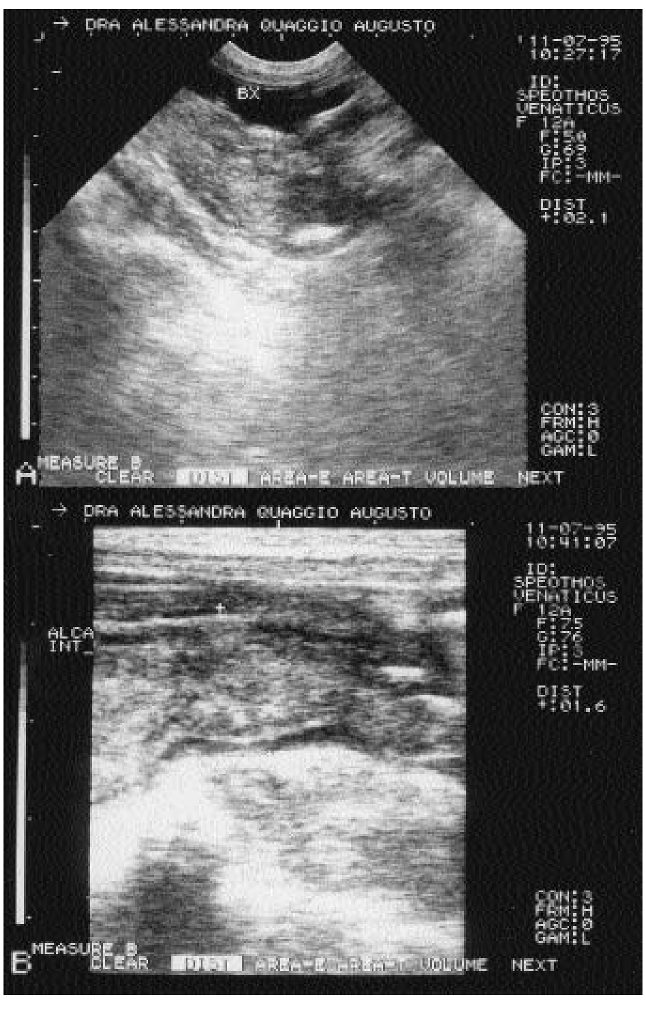

FIGURE 39.12. Sonograms of enlarged abdomen of a bush dog *(Speothos venaticus)*. The animal had symptoms of apathy and inappetance; blood was observed on the floor of the enclosure. The clinical symptoms suggested uterine infection, but ultrasonography detected distended bowel loops and colon. **A.** Colon is replete with fecal material with small dense (hyperechoic area) material (small bone from diet). **B.** Longitudinal image of small bowel with dense fecal material.

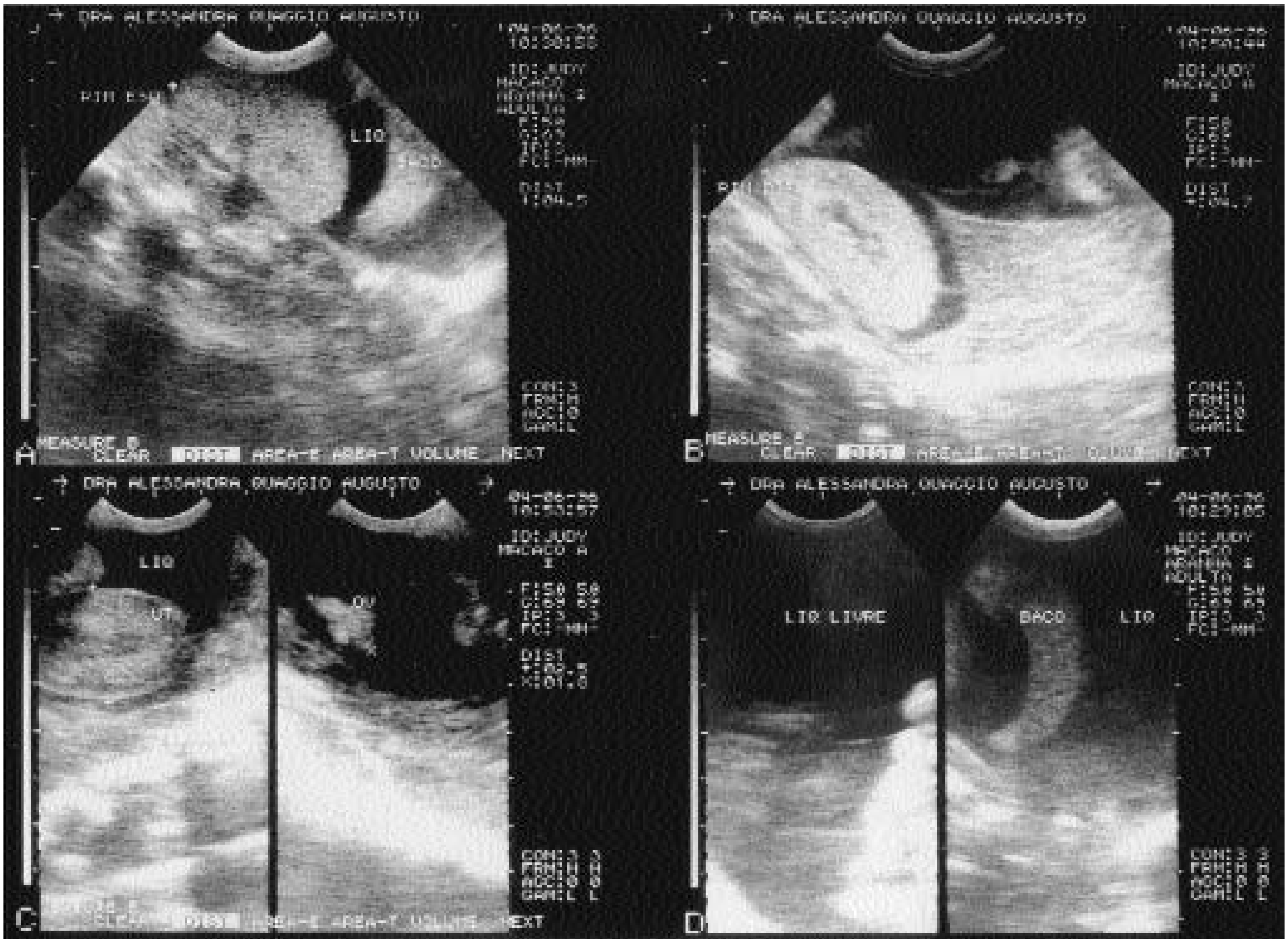

FIGURE 39.13. Sonograms of ascites associated with renal problems of a female spider monkey (*Ateles paniscus).* **A–D.** Excessive free abdominal fluid (LIQ/LIQ LIVRE) in anechoic areas. **A.** Left kidney (RIM ESQ) with increased echogenicity and spleen (BACO). **B.** Right kidney (RIM DIR) with increased echogenicity and liver (FIG) separated by ascites. **C.** Normal uterus (UT) and ovary (OV). **D.** Normal spleen (BACO). The animal died, and glomerulonephritis and chronic interstitial nephritis were diagnosed by histopathological examination.

REFERENCES

1. Anderson, N.L. 1994. Successful treatment of a urolith associated with a fungal cystitis in *Iguana iguana*. In Proceedings Association of Reptilian and Amphibian Veterinarians. Chester Heights, Pennsylvania, Association of Reptilian and Amphibian Veterinarians, pp. 52–57.
2. Augusto, A.Q.; Ferreira, F.M.; Pachaly, J.R.; and Lange, R.R. 1995. Biometria ultra-sonográfica de globos oculares de *Nycticorax nycticorax* [Ocular globe ultrasonographic biometry in *Nycticorax nycticorar*]. In Proceedings of the 2° Simpósio sobre Ciências Médicas e Biológicas. Curitiba-PR, Archives of Veterinary Science, p. 14.
3. Augusto, A.Q.; Ferreira, F.M.; Pachaly, J.R.; Werner, P.R.; Lacerda, O.; Lange, R.R.; and Vilani, R.G.D.'O.C. 1996. Utilização do exame ultra-sonográfico como método diagnóstico para diferenciar ascite em Macaco Aranha (*Ateles paniscus*)—Relato de caso [Application of ultrasonographic exam as a diagnostic method in order to differentiate ascites in spider monkey—Case report]. In Proceedings of the 3° Simpósio sobre Ciências Médicas e Biológicas. Curitiba-PR, Archives of Veterinary Science, p. 46.
4. Augusto, A.Q.; Pachaly, J.R.; Werner, P.R.; and Lange, R.R. 1995. Ultra-sonografia de um adenoma hepático em um puma (*Felis concolor*) [Ultrasonography of hepatic adenoma of a cougar]. In Proceedings of the 4° Congresso Nacional do Associação Brasileira de Animais Selvagens. Curitiba-PR, Archives of Veterinary Science, p. 11.
5. Augusto, A.Q.; Pachaly, J.R.; and Lange, R.R. 1995. Diagnóstico ultra-sonográfico de gestação em Cutia *(Dasyprocta azarae*) [Ultrasonographic diagnosis of pregnancy of an agouti]. In Proceedings of the 4° Congresso Nacional do Associação Brasileira de Animais Selvagens. Curitiba-PR, Archives of Veterinary Science, p. 10.

6. Cubas, S.Z. 1996. Special challenges of maintaining wild animals in captivity in South America. In M.E. Fowler, ed., Wildlife Husbandry and Diseases. Office International des Epizooties Scientific and Technical Review 15(1):267– 288.
7. Dorrestein, G.M.; and Van der Hage, M.H. 1998. Selected disease—Cases in parrots. In Proceedings of the European Association of Zoo and Wildlife Veterinarians. European Association of Zoo and Wildlife Veterinarians, pp. 355–358.
8. Enders, F.; Krautwald-Junghanns, M.; and Dühr, D. 1993. Sonographic evaluation of liver diseases in birds. In Proceedings of the European Association of Avian Veterinarians. Utrecht, European Association of Avian Veterinarians, pp. 155–162.
9. Fowler, M.E. 1986. Felidae. In M.E. Fowler, ed., Zoo and Wildlife Medicine. Philadelphia, W.B. Saunders, pp. 831–841.
10. Hildebrandt, T.B.; and Göritz, F. 1999. Use of ultrasonography in zoo animals. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, Vol. 4. Current Therapy. Philadelphia, W.B. Saunders, pp. 41–54.
11. Hildebrandt, T.; Pitra, C.; Sommer, P.; and Pinkowski, M. 1995. Sex identification in birds of prey by ultrasonography. Journal of Zoo and Wildlife Medicine 26(3):367–376.
12. Jacobson, E.R.; Homer, B.; and Adams, W. 1991. Endocarditis and congestive heart failure in a Burmese python (*Python molurus bivittatus*). Journal of Wildlife Medicine 22:245–248.
13. Karesh, W.B. 1983. The use of diagnostic ultrasonography in zoo medicine. In Proceedings of the Annual Meeting of the American Association of Zoo Veterinarians. Tampa, Florida, American Association of Zoo Veterinarians, pp. 2–4.
14. Krautwald-Junghanns, M.E.; and Enders, F. 1994. Ultrasonography in birds. Seminars in Avian and Exotic Pet Medicine 3(3):140–146.
15. Mader, D.R. 1996. Reptile Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 220–221.
16. Mader, D.R. 1994. Diagnostic techniques in reptile medicine. In Proceedings of the Association of Reptilian and Amphibian Veterinarians. Association of Reptilian and Amphibian Veterinarians, pp. 27(33.
17. Nyland, T.G. 1995. Veterinary Diagnostic Ultrasound. Philadelphia, W.B. Saunders, p. 4.
18. O'Grady, J.P.; Yeager, C.H.; and Thomas, W. 1978. Practical application of real time ultrasound scanning to problems of zoo veterinary medicine. Journal of Zoo Animal Medicine 9:52–56.
19. Penninck, D.G,; Stewart, J.S.; Paul-Murphy, J.; and Pion, P. 1991. Ultrasonography of the California desert tortoise (*Xerobates agassizi*): Anatomy and application. Veterinary Radiology and Ultrasound 32(3):112–116.
20. Ritchie, B.W.; Harrison, G.J.; and Harrison, L.R. 1994. Avian Medicine: Principles and Application. Lake Worth, Florida, Wingers Publishing, p. 325.
21. Stetter, M.D. 1996. Comparison of magnetic resonance imaging, computerized axial tomography, ultrasonography and radiology for reptilian diagnostic imaging. In Proceedings of the American Association of Zoo Veterinarians. Puerto Vallarta, Mexico, American Association of Zoo Veterinarians, pp. 450–453.
22. Stoskopf, M. 1989. Clinical imaging in zoological medicine. Journal of Wildlife Medicine 20:396–412.

40
Interspecific Allometric Scaling

José Ricardo Pachaly
Harald Fernando Vicente de Brito

GENERAL CONSIDERATIONS

Body size is the single most important characteristic of an organism, since it influences the physical environment faced and, the likelihood of finding sufficient food and avoiding predators.[24] Fundamental characteristics of the organisms, including its anatomy, energy expenditure for maintenance, feeding habits, forms of reproduction, and means of locomotion vary with the body size. The relationships between these characteristics and body size vary quantitatively. These relations are often called allometric relationships.[24] Allometric relationships are generally described as a power function of the body mass.

Basal metabolic rate (BMR) also establishes the minimum energy cost (MEC), which is considered the best way of comparing different organisms. Being a fundamental relationship that exists among all the organisms, MEC is the base for the process of allometric scaling.[22,47] Allometric scaling allows the mathematical extrapolation from one vertebrate species to another. By knowing the BMR of both species and with a knowledge of the pharmacokinetics and pharmacodynamics of a given drug for one species, it is possible to determine a suitable dose of the drug for the other. The purpose of the allometric method is to extrapolate doses of drugs between animals of different sizes and/or taxa, facilitating the use of data obtained in a "model animal" (animal for which the drug was developed) for the treatment of a "target animal" (wild or domestic patient).[50]

BASAL METABOLIC RATE

The knowledge now available on the metabolism of vertebrates is based, largely, in the works of Kleiber, accomplished in the 1930s and 1940s. He demonstrated that the relationship between the metabolic rate and the body mass is not linear, but is a function of metabolic weight.

Metabolic weight (MW) is expressed in the formula $MW = M^{0.75}$, where M is the body weight in kilograms.[8,53]

The basal metabolic rate (BMR) is the value measured when an endothermal animal is inactive and fasting, without suffering any kind of stress and maintained within optimal environmental temperatures.[53]

ALLOMETRIC SCALING

Allometry is the study of the way by which a dependent variable, such as the metabolic rate, varies in relation to an independent variable, such as the body mass. The method of allometric scaling involves the study of the relationships of functions and organic systems to the body size.[49] Allometric calculations can be very useful when one needs to determine basic physiologic data of any amniote, of any size[45,46], such as resting heart rate, respiratory rate, and tidal and minute volumes[45,46], anatomical dead space volume, oxygen consumption, blood volume, cardiac output, and size of specific organs such as the heart.[46]

Basal metabolic rate is the basis for allometric scaling. The general formula for calculation of the basal metabolic rate of wild or domestic vertebrates is expressed by the following equation:[7,9,11,12,14,–17,22,23,41,42,45–48,50,51]

$$BMR = K \times M^{0.75}$$

In this formula, M is the body mass in kilograms and K is a theoretical constant of proportionality, taxonomically dependent, based on the mean core body temperature.

Table 40.1 summarizes the data for the K constant, which is absolutely necessary for calculating the basal metabolic rate of mammals and birds.

TABLE 40.1. Values for the K constant in birds and mammals, relating taxonomic groups and their core body temperatures

Taxonomic Groups	Value of the K Constant	Mean Core Body Temperature
Birds		
Passerines	129	42°C
Non-passerines	78	40°C
Mammals[a]		
Placentals	70	37°C
Marsupials	49	35°C

Source: Modified from reference 13.
[a]For Xenarthra (Edentata) and Monotremata, the K value is 49, which is the same for Marsupialia, whose mean body temperature is also 35°C.[10]

The specific metabolic rate of a given animal can be obtained by dividing its basal metabolic rate by its body mass, according to the equation below:

$$SMR = K \times M^{0.75}/M$$

PHYSIOLOGIC CONSIDERATIONS ON THE BIOAVAILABILITY OF DRUGS

Smaller animals, with their higher metabolic rate, differ from larger ones in the same taxonomic group in regard to their biological particularities, including the speed of occurrence of physiologic events. Comparatively, smaller animals have higher mean times of blood circulation and higher heart rates than larger ones.[43] They have a larger body surface area and greater need of oxygen by unit of body mass. They have higher densities of capillary vessels by unit of any given tissue to ensure a larger surface area for diffusion of substances, a larger respiratory gas exchange surface area, a higher glomerular filtration rate, a higher density of hepatocytes, and higher intracellular concentrations of mitochondria and cytochrome-C per unit of body mass than larger animals.[5,43] Schmidt-Nielsen[43] summarized and simplified those facts affirming that "small animals have more metabolic tools than large animals."

Therefore, the physiologic events related to the uptake, distribution, action, and elimination of drugs in smaller animals are evidently different from those observed in larger ones. Smaller animals tend to require higher doses, in relation to body mass, of drugs whose dynamics are influenced by the metabolic rate, because of their higher metabolic rates.[18,22,41,50] In smaller animals, drugs tend to have faster absorption, distribution and excretion than they do in larger ones.[18,19,22,50] This means that to maintain effective serum levels of a given drug, the dosage (in milligrams per kilogram per day) should be higher and the interval of administration (in hours) should be shorter in smaller animals than in larger animals, if all other factors that influence pharmacokinetics are the same.

When the dosage of a given drug is allometrically extrapolated between two morphologic and metabolically identical animals, but one is small and the other is large, a paradigm is verified: To reach the same serum concentration of the drug, the smaller animal should receive the largest dose, and the larger animal should receive the smallest, according to their respective body weight.

ALLOMETRY APPLIED TO CALCULATION OF DOSAGE SCHEDULES FOR ANIMALS

Veterinarians who deal with wild animals are confronted with a wide range of forms, sizes, and metabolic rates in creatures whose weights vary from a few grams to thousands of kilograms. Such variety creates a serious clinical problem, since little pharmacodynamic information on drugs exists for most of those species.[18,47,50] It becomes necessary, therefore, to extrapolate doses of drugs from one animal to another,[18,50] but more than in any other field of medicine, treatments with drugs in wild animal practice present a high potential for incorrect calculations.[17,50] Calculation mistakes may result in either subdosages and therapeutic inefficiency or in excessive dosages and risks of serious toxicity.[17,50] Veterinarians and physicians traditionally calculate the doses of drugs based on the patient's body weight or mass. This is an efficient procedure when the comparison between animals is based on the similarity between their masses and metabolic rates. However, when great differences exist in mass and metabolic rates, as happens when animals with hundreds of kilograms of difference are compared, or even more, when they belong to different energy groups (*K*), the dosages can vary tremendously.[17,47,50] In such situations, to define a rational method for extrapolation of doses becomes an enormous challenge.[47,50] The ideal situation would be to monitor the therapeutic regimens for each drug and for each species and size by analyzing the serum concentration of the drug.[47] Because of the numerous difficulties inherent to this kind of monitoring, especially in routine clinical situations, the more rational alternative is allometric scaling. This method allows the calculation of dosages for animals other than those for which pharmacokinetic studies have been performed, being the calculation of drug dosages based on the energetic needs of the animals, instead of on the body mass.[1–4,7,9,11,12,14–17,22,23,25,26,28–30,33–42,45–52]

Conventionally, doses of drugs are calculated and expressed as amount by unit of body weight (milligrams per kilogram). The method of allometric scaling, however, calculates and expresses doses as amount for energy consumed by a given animal under basal metabolism (milligrams per kilocalorie). Once absorption, distribution and elimination of drugs happen in the function of the basal metabolic rate, a dose in milligrams per kilocalorie can be used for all animal sizes from all species that absorb, metabolize, and distribute the drug in the same way.[50] Therefore, the basal metabolic rate may be used to calculate the dosage of a given drug for a given animal, based on the dosage established for another one, adjusting for the metabolic and taxonomic differences among the two animals.[12,22,50] That is possible because the pharmacokinetic events related to the drugs are essentially the same, regardless of the species, age, or size of the animal.[50]

METHOD OF ALLOMETRIC CALCULATION OF DRUG DOSAGES

The method of calculation for extrapolating drug dosages from one animal to another, in linear regression (milligrams per kilocalorie) was first proposed by Sedgwick and Pokras.[50] With small modifications or adaptations, their indications were used by several other investigators.[7,9,11,12,14–17,22,23,25,41,42,45–48,51] The available data allows a summary of the method for allometric scaling of drug dosages, as follows.

Initially an animal of known body mass for which reliable scientific data about the drug already exists should be defined as a model animal. Such data should correlate the recommended dosages with therapeutic serum levels (in the case of, for example, antibiotics), or with observed clinical responses (in the case of anesthetics or tranquilizers). The equation $BMR = K \times M^{0.75}$ is used to calculate the basal metabolic rate of the model animal and the target animal. The target animal can be any wild or domestic mammal or bird for which no dosage data exists for the drug dose that will be extrapolated. The total daily dose indicated for the model animal (in milligrams) is then divided by the value of its BMR to obtain the dose indicated for that animal in milligrams per kilocalorie, denoted as $\text{Dose}_{BMR\text{-model}}$. The value of BMR of the target animal is then multiplied by the $\text{Dose}_{BMR\text{-model}}$ to obtain the total dose of the drug in milligrams to be administered to the target animal. To determine the dose in milligrams per kilogram, divide the total dose by the body mass of the target animal. Table 40.2 summarizes these steps.

TABLE 40.2. General method of calculation for interspecific allometric scaling of drug doses

1. Calculate the basal metabolic rate (BMR) of the model animal and the target animal.
2. Divide the total dose indicated for the model animal (in milligrams) by its BMR.
3. Multiply the result by the BMR of the target animal.
4. The result is the total dose (in milligrams) for the target animal.
5. Divide the total dose by the weight (in kilograms) of the target animal.
6. The result is the dose for the target animal (in milligrams per kilogram).

TABLE 40.3. Simplified method of calculation for interspecific allometric scaling of doses of drugs for animals of the same taxonomic group

1. Calculate the metabolic weights (MW) of the model animal and the target animal.
2. Divide the total dose indicated for the model animal (in milligrams) by its MW.
3. Multiply the result by the MW of the target animal.
4. The result obtained is the total dose (in milligrams) for the target animal.
5. Divide the total dose by the weight (in kilograms) of the target animal.
6. The result obtained is the dose (in milligrams per kilogram) for the target animal.

When both the model and target animals belong to the same taxonomic group, allometric scaling may be simplified; the dosage may be calculated on the basis of the metabolic weight, using the equation $PM = M^{0.75}$, as presented in Table 40.3.

ALLOMETRIC CALCULATION FOR THE FREQUENCY OF DRUG ADMINISTRATION

Frequency of administration is the recommended interval, in hours, between administration of drugs to an animal. Knowing the recommended interval for the model animal, it is possible to calculate the frequency of administration of the same drug for the target animal.

There exists in the literature references to such calculations based on equations as that proposed by Johnson[17] et al. To make the calculation of the frequency of administration easier, we propose a simplified method, which is based on the specific metabolic rate, as

TABLE 40.4. Using interspecific allometric scaling for calculation of frequency of drug administration

1. Calculate the specific metabolic rate (SMR) of the model animal and the target animal.
2. Multiply the SMR of the model animal by the administration interval of the drug of the model animal (in hours).
3. Divide the result by the SMR of the target animal.
4. The result is the interval of administration of the drug (in hours) for the target animal.

explained in Table 40.4 and represented by the following equation:

$$\text{Interval target animal} = \text{SMR model animal} \times \text{interval model animal/SMR target animal}$$

USING INTERSPECIFIC ALLOMETRIC SCALING IN CHEMICAL RESTRAINT: THE BRAZILIAN EXPERIENCE

Reports of the use of interspecific allometric scaling to calculate drug doses for chemical restraint of wild and domestic animals were published in scientific journals and/or presented in congresses beginning in 1995 in Brazil. Those reports testify to the utility and safety of the method, even when dealing with such potentially problematic and dangerous animals as large carnivores and elephants.

The method was used to calculate dosages of anesthetics, sedatives and/or neuroleptics for chemical restraint or behavior modulation of Neotropical wild animals such as *Panthera onca,*[29,37,40] *Felis concolor,*[4] and *Dasyprocta azarae.*[25,33] It was used for the same purposes in other wild animals such as *Panthera leo,*[30,36,39] *Panthera tigris,*[2,3] *Ursus arctos,*[34] *Loxodonta africana,*[1] *Cervus unicolor,*[38] *Cervus elaphus,*[28] and also in domestic animals such as *Felis cattus,*[6] *Equus caballus,*[27] *Canis familiaris,*[31] and *Ovis aries.*[32]

The work of Pachaly and Brito[33] with *Dasyprocta azarae* is a good example of the efficacy and safety of the method. The authors used interspecific allometric scaling to calculate doses of the α_2 agonist detomidine hydrochloride (HCl) for the restraint of two agoutis (*Dasyprocta azarae*), in association with ketamine HCl and atropine sulfate. Detomidine HCl is a potent sedative indicated for chemical restraint of horses, and there were no previous reports of its use in agoutis or other Neotropical rodents. The calculations were based on using the dose of 75 mg/kg for a 500 kg horse as model. One of the agoutis, weighing 3.28 kg, received a dose of 265.24 mg/kg. The second one weighed 0.78 kg and received a dose of 384.61 mg/kg. The administered doses, disregarding the interspecific allometric scaling, would be considered extremely elevated for a 500-kg horse. The dose administered to the second agouti was 380.76% larger than the maximum recommended dose and could be lethal to a 500-kg horse. In the agouti, however, such a dosage induced excellent sedation and muscular relaxation and provided a safe and uneventful recovery.

COMMENTS

This chapter discussed the use of interspecific allometric scaling and indicates the method as an effective tool when dealing with mammals and birds. There are some references of its employment in reptiles,[16,21–23,41,49] but it seems premature to recommend the method for this taxonomic group because of the enormous diversity observed among reptiles and the lack of metabolic data on the great majority of reptilian species.[16,21]

All the formulas used for the simplified methods proposed in this chapter may be used to set up calculation worksheets in computer programs such as the MS-Excel to facilitate the calculations.[25]

REFERENCES

1. Brito, H.F.V.; Pachaly, J.R.; and Kasecker, G.G. 1995. Emprego da extrapolação alométrica interespecífica no cálculo da dose de cloridrato de xilazina e sulfato de atropina, para a contenção de um elefante africano (*Loxodonta africana*) [Use of interspecific allometric scaling to calculate xylazine HCl and atropine sulfate doses to chemically restraint an African elephant]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pos Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 12.
2. Brito, H.F.V.; Pachaly, J.R.; and Kasecker, G.G. 1995. Emprego do cloridrato de detomidina, em associação a cloridrato de ketamina e sulfato de atropina, para a contenção de um tigre (*Panthera tigris*), com base em extrapolação alométrica interespecífica [Use of interspecific allometric scaling to calculate detomidine HCl, ketamine HCl and atropine sulfate doses to chemically restrain a tiger]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 13.
3. Brito, H.F.V.; Pachaly, J.R.; and Kasecker, G.G. 1995. Emprego do cloridrato de xilazina, em associação a sulfato de atropina, na contenção de um tigre (*Panthera*

tigris), com base em extrapolação alométrica interespecífica [Use of interspecific allometric scaling to calculate xylazine HCl and atropine sulfate doses to chemically restrain a tiger]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 14.
4. Brito, H.F.V.; Pachaly, J.R.; and Lacerda, O. 1995. Emprego de cloridrato de detomidina em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na contenção de um puma (*Felis concolor),* com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate detomidine HCl, titelamine HCl, zolazepam and atropine sulfate doses to chemically restrain a puma]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 2.
5. Calder, W.A. 1984. Size, Function and Life History. Cambridge, Massachusetts, Harvard University Press.
6. Costa, R.C.; Pachaly, J.R.; and Brito, H.F.V. 1995. Uso de um neuroléptico de ação prolongada (decanoato de haloperidol) no tratamento de dermatite psicogênica em gato doméstico [Use of interspecific allometric scaling to calculate haloperidol decanoate doses to treat psychogenic dermatitis in a domestic cat]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 22.
7. Dorrestein, G.M. 1997. Metabolism, pharmacology and therapy. In R.B. Altman, S.L. Clubb, G.M.; Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia: W.B. Saunders, pp. 661–670.
8. Feldman, H.A.; and McMahon, T.A. 1983. The ¾ mass exponent for energy metabolism is not an statistical artifact. Respiration Physiology 52:149–163.
9. Fowler, M.E. 1993. Tópicos em medicina de animais selvagens [Topics in wild animal medicine]. Course handout, Curso de Pós Graduação em Medicina Veterinária da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo.
10. Fowler, M.E. 1993. Pers. commun. São Paulo.
11. Gamble, K.C.; Boothe, D.M.; Jensen, J.M.; Heatley, J.J.; and Helmick, K.E. 1997. Pharmacokinetics of a single enrofloxacin dose in scimitar-horned oryx (*Oryx dammah*). Journal of Zoo and Wildlife Medicine 28(1):36–42.
12. Gibbons, G.; Pokras, M.; and Sedgwick, C. 1988. Allometric scaling in veterinary medicine. Australian Veterinary Practitioner 18(4):160–164.
13. Hainsworth, F.R. 1981. Animal physiology adaptations in function. Reading, Massachusetts, Addison-Wesley, pp. 160–163.
14. Harrison, G.J. 1994. Determination of metabolic scaling. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lake Worth, Florida, Wingers, p. 1351.
15. Heard, D.J. 1997. Anesthesia and analgesia. In R.B. Altman, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds.,. Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 807–827.
16. Jacobson, E.R. 1995. Use of antimicrobial therapy in reptiles. In Antimicrobial therapy in caged birds and exotic pets: Proceedings of the International Symposium at the North American Veterinary Conference. pp. 28–37.
17. Johnson, J.H.; Wolf, A.M.; Johnson, T.L.; and Jensen, J.M. 1993. Gentamicin toxicosis in a North American cougar. Journal of the American Veterinary Medical Association 203(6):854–856.
18. Kirkwood, J.K. 1983. Influence of body size in animals on health and disease. Veterinary Record 113(13):287–290.
19. Kirkwood, J.K. 1984. Dosing exotic species. Veterinary Record 114(18).
20. Kirkwood, J.K.; and Wathes, C.M. 1984. Size, time, ketamine and birds. Veterinary Record 115(14):390–391.
21. Klingenberg, R.K. 1994. Basic principles of therapeutics used in reptile medicine. In Proceedings of the American Association of Zoo Veterinarians Annual Conference. Pittsburgh, American Association of Zoo Veterinarians, pp. 18–26.
22. Mader, D.R. 1991. Metabolic scaling of antibiotic dosages. In F.L. Frye, ed., Reptile Care—An Atlas of Diseases and Treatments, Vol. 2. Neptune City, T.F.H. Publications, pp. 632–633.
23. Martin, J.C.; and Sedgwick, C.J. 1994. A review of allometric scaling with considerations for its application to reptile therapeutics. In Proceedings of the American Association of Zoo Veterinarians Annual Conference. Pittsburgh, American Association of Zoo Veterinarians, pp. 62–65.
24. McNab, B.K. 1988. Complications inherent in scaling the basal rate of metabolism in mammals. The Quarterly Review of Biology 63(1):25–54.
25. Pachaly, J.R. 1998. Contenção da Cutia, *Dasyprocta azarae* Lichtenstein, 1823 (Rodentia: Mammalia), Pela Associação de Cloridrato de Cetamina, Cloridrato de Xilazina e Sulfato de Atropina(Definição de Protocolos Posológicos Individuais com Base em Extrapolação Alométrica Interespecífica [Restraint of the agouti, *Dasyprocta azarae,*Lichtenstein, 1823 (Rodentia: Mammalia), by the association of ketamine hydrochloride, xylazine hydrochloride and atropine sulfate—Individual dosages defined by interspecific allometric scaling]. Doctoral thesis, Universidade Federal do Paraná.
26. Pachaly, J.R.; Deconto, I.; Brito, H.F.V; Domingos, I.T.; Teixeira, V.N.; Cartelli, R.; Silva, A.S.P.F.; Maçaneiro, C.G.; Gois, D.R.; Vilani, R.G.D.C.; and Silva, M.E.P.F. 1995. Emprego de cloridrato de detomidina em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na anestesia de um gnu (*Connochaetes taurinus*) [Use of interspecific allometric scaling to calculate detomidine HCl, titelamine HCl, zolazepam and atropine sulfate doses to chemically restraint a gnu]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas

do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pos Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 56.
27. Pachaly, J.R.; Vilani, R.G.D'O.C.; Brito, H.F.V.; Deconto, I.; and Teixeira, V.N. 1996. Emprego de cloridrato de tiletamina e zolazepam, em associação a cloridrato de romifidina e sulfato de atropina, na anestesia de um cavalo doméstico (*Equus caballus*), com base em cálculos de extrapolação alométrica interespecífica [Use of interspecific allometric scaling to calculate romifidine HCl, tiletamine HCl, zolazepam and atropine sulfate doses to chemically restrain a domestic horse]. Archives of Veterinary Science 1(1):38.
28. Pachaly, J.R.; Brito, H.F.V.; Mangini, P.R.; Ferreira, F.M.; Silva, A.S.P.F.; and Silva, M.E.P.F. 1996. Emprego de decanoato de haloperidol na modulação comportamental de um grupo de cervos nobres (*Cervus elaphus*) [Use of interspecific allometric scaling to calculate haloperidol decanoate doses to behavior modulation of a group of *Cervus elaphus*]. In Abstracts 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 75.
29. Pachaly, J.R.; Brito, H.F.V.; Mangini, P.R.; Ferreira, F.M.; Silva, A.S.P.F.; and Silva, M.E.P.F. 1996. Emprego de decanoato de haloperidol na modulação comportamental de um casal de onças (*Panthera onca*) [Use of interspecific allometric scaling to calculate haloperidol decanoate doses to behavioral modulation of a pair of jaguars]. In: Abstracts 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 75.
30. Pachaly, J.R.; Lange, R.R.; Jaworovski, M.L.; Teixeira, V.N.; Cartelli, R.; Ferreira, F.M.; Mangini, P.R.; and Ciffoni, E.M.G. 1997. Anestesia em leão (*Panthera leo*) pela associação de cloridrato de xilazina, sulfato de atropina, cloridrato de tiletamina e zolazepam em três ocasiões diferentes—Protocolo posológico calculado por meio de extrapolação alométrica interespecífica [Use of interspecific allometric scaling to calculate xylazine HCl, tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of a lion]. In Anais 19° Congresso Brasileiro de Clínicos Veterinários de Pequenos Animais. Campo Grande, Panamerican Association of Veterinary Sciences, p. 21.
31. Pachaly, J.R.; Ciffoni, E.M.G.; Pedroso, F.F.; Silveira, A.P.; and Roehsig, C. 1998. General anesthesia of dogs with allometrically scaled dosage of xylazine HCl, tiletamine HCl, zolazepam and atropine sulfate. In Summary Proceedings of the 16th Panamerican Congress of Veterinary Sciences, Santa Cruz de la Sierra, Panamerican Congress of Veterinary Sciences, p. 153.
32. Pachaly, J.R.; Ciffoni, E.M.G.; Saab, A.B.; Poleze, E.; Lupatini, F.; Altmeyer, J.C.; Bornia, P.A. B.; Brotto, W.G.; Magrinelli, F.A.; and Estelai, A.E. 1998. General anesthesia of sheep with allometrically scaled dosage of xylazine HCl, tiletamine HCl, zolazepam and atropine sulfate. In Summary Proceedings of the 16th Panamerican Congress of Veterinary Sciences. Santa Cruz de la Sierra, Panamerican Congress of Veterinary Sciences, p. 255.
33. Pachaly, J.R.; and Brito, H.F.V. 1995. Emprego de cloridrato de detomidina, em associação a cloridrato de ketamina e sulfato de atropina, na contenção de cutias (*Dasyprocta azarae*), com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate detomidine HCl doses to chemically restrain agoutis, in association to ketamine HCl and atropine sulfate].In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 1.
34. Pachaly, J.R.; and Brito, H.F.V. 1996. Emprego da extrapolação alométrica na definição de protocolos anestesiologicos individuais para animais selvagens [Use of interspecific allometric scaling to calculate individual anesthetic schedules for wild animals]. In Abstracts 15° Congresso Panamericano de Ciências Veterinárias. Campo Grande, Panamerican Association of Veterinary Sciences, p. 75.
35. Pachaly, J.R.; and Brito, H.F.V. 1996. Emprego de cloridrato de detomidina, em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na contenção de urso marrom (*Ursus arctos*), com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of a brown bear]. Informativo da Associação Brasileira de Veterinários de Animais Selvagens 4(17):2.
36. Pachaly, J.R.; Brito, H.F.V.; Lacerda, O. 1995. Emprego de cloridrato de xilazina, em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina na contenção de um leão (*Panthera leo*), com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of a lion]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 1.
37. Pachaly, J.R.; Brito, H.F.V.; and Lacerda, O. 1995. Emprego de cloridrato de detomidina em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na contenção de duas onças (*Panthera onca),* com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate detomidine HCl, tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of two jaguars]. In Anais 1° Jornada de Medicina de Animais Selvagens e de Pequenos Ruminantes do Cone Sul. Curitiba, ABRAVAS/AVEPER, p. 2.
38. Pachaly, J.R.; Brito, H.F.V.; and Lange, R.R. 1995. Emprego de cloridrato de detomidina, em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na anestesia cirúrgica de um cervo sambar (*Cervus unicolor*), com base em extrapolação alométrica interespecífica [Use of interspecific allometric scaling to calculate detomidine HCl, tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of a *Cervus unicolor*]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação

em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 50.

39. Pachaly, J.R.; Brito, H.F.V.; and Mangini, P.R. 1995. Emprego de cloridrato de detomidina em associação a cloridrato de tiletamina, zolazepam e sulfato de atropina, na contenção de duas leoas (*Panthera leo)*, com base em extrapolação alométrica [Use of interspecific allometric scaling to calculate detomidine HCl, tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of two lionesses]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 51.
40. Pachaly, J.R.; Brito, H.F.V.; and Silva, A.S.P.F. 1995. Emprego da extrapolação alométrica interespecífica no cálculo de doses de cloridrato de detomidina, cloridrato de tiletamina, zolazepam e sulfato de atropina, para a contenção de duas onças (*Panthera onca*) [Use of interspecific allometric scaling to calculate detomidine HCl, tiletamine HCl, zolazepam and atropine sulfate doses in the chemical restraint of two jaguars]. In Resumos 2° Simpósio sobre Ciências Médicas e Biológicas do Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná. Curitiba, Curso de Pós Graduação em Ciências Veterinárias da Universidade Federal do Paraná, p. 52.
41. Pokras, M.A.; Sedgwick, C.J.; and Kaufman, G.E. 1992. Therapeutics. In P.H. Beynon, ed., Manual of Reptiles. Gloucestshire, U.K., British Small Animal Veterinary Association, pp. 194–209.
42. Quesenberry, K.E.; and Hillyer, E.V. 1994. Supportive care and emergency therapy. In B.W. Ritchie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lake Worth, Florida, Wingers, pp. 382–416.
43. Schmidt-Nielsen, K. 1984. Scaling: Why Is Animal Size So Important? Cambridge: Cambridge University Press, pp. 90–98.
44. Schmidt-Nielsen, K. 1996. Fisiologia Animal: Adaptação e Meio Ambiente [Animal physiology: Adaptation and environment]. São Paulo, Santos Editora.
45. Sedgwick, C.J. 1988. Anesthetic and chemical restraint techniques for zoo animals and wildlife. In Proceedings of the 55th Annual Meeting of the American Animal Hospitals Association. pp. 162–166.
46. Sedgwick, C.J. 1991. Allometrically scaling the data base for vital sign assessment used in general anesthesia of zoological species. In Proceedings of the American Association of Zoo Veterinarians Annual Conference. pp. 360–369.
47. Sedgwick, C.J. 1993. Allometric scaling and emergency care: The importance of body size. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 3rd Ed. Philadelphia, W.B. Saunders, pp. 34–37.
48. Sedgwick, C.J. 1994. Grisefulvin toxicity in zoo cats. Zoo Vet News 10(3):11–12.
49. Sedgwick, C.J.; and Borkowski, R. 1996. Allometric scaling: Extrapolating treatment regimens for reptiles. In D.R. Mader, ed., Reptile Medicine and Surgery, Philadelphia, W.B. Saunders, pp. 235–240.
50. Sedgwick, C.J.; and Pokras, M.A. 1988. Extrapolating rational drug doses and treatment periods by allometric scaling. In Proceedings of the 55th Annual Meeting of the American Animal Hospitals Association. pp. 156–161.
51. Sedgwick, C.J., Pokras, M.A.; and Kaufman, G. 1990. Metabolic scaling: Using estimated energy costs to extrapolate drug doses between different species and different individuals of diverse body sizes. In Proceedings American Association of Zoo Veterinarians Annual Conference. pp. 249–254.
52. Timm, K.L.; Picton, J.S.; and Tylman, B. 1994. Surface area to volume relationships of snakes support the use of allometric scaling for calculating dosages of pharmaceuticals. Laboratory Animal Science 44(1):60–62.
53. Withers, P.C. 1992. Animal Energetics. Comparative Animal Physiology. Fort Worth, Texas, Saunders, pp. 82–121.

41
Pests and Nuisance Animals in Zoological Parks

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INTRODUCTION

The abundance of food and waste in animal housing areas provides an ideal environment for the proliferation of vertebrate and invertebrate pests. Pests are undesired animals that may place the animal collection at risk, either by predation or disease transmission. Nuisance and pest animals may also carry zoonoses. Pests detract from visitors' experience and the reputation of the institution, because their presence is recognized as a sign of unsanitary conditions.

Diseases may be introduced into an animal collection through pests (see Table 41.1). Exclusion or control of pests depends on the observance of basic rules, strategies, and actions. Although significant animal losses by predation and disease may occur in most of the zoos in South America, few institutions have given the necessary attention to implementation of permanent pest control programs.

Preventive methods should prevail over control procedures, which are usually associated with the use of toxic compounds. Avoiding the access, permanence, and reproduction of pest animals is a wise way to initiate an integrated pest management program. In most situations, this means reducing the availability of food, waste, and shelter. Excessive use of toxic compounds creates unnecessary risk to the animal collection, employees, and visitors and increases operating expenses. Effective pest control programs must be permanent, planned, and supervised, and should involve most of the employees, including maintenance personnel, keepers, biologists, veterinarians, administrators, and, if necessary, consultants with expertise in pest control. In-house staff directly involved in pest control and pesticide application should receive training, and it may be necessary for them to be certified by the country or state, according to local regulations. Pest control companies may eventually be contracted to orient zoo personnel or to do the job, if budget allows doing so.

Decisions regarding the disposition of live-trapped animals should be made in advance in accordance with wildlife agency regulations and local public health agency policy (in the case of domestic animals). Relocation or humane euthanasia of wild animals is only possible if allowed by local wildlife agencies. This subject is usually treated as taboo, and official impediments may discourage the implementation of a trapping program.

The impact of pest and nuisance animals may vary in different parts of the world. This chapter covers general aspects and methods of prevention and control of the most common pest species in South American zoos. The term infestation is used to describe the presence of pests in a specific area. See Table 41.1 for a listing of infectious diseases potentially transmitted by pest species as hosts, carriers, or reservoirs.

PEST MANAGEMENT IN ZOOS

Integrated pest management (IPM) is the term employed to describe a strategy that uses regular monitoring and pest control methods to keep infestation at tolerable levels.[4,11] Diminishing the use of pesticides to avoid harm to the animal collection and to the environment is a goal of IPM. Direct and indirect suppression tactics are employed. Training and involvement of staff in regard to hygiene, preventive procedures, and environmental monitoring are considered important indirect strategies of control. Traps, baits, and other physical methods are useful direct suppression strategies in controlling severe infestations.

The pest control officer is a position that may be created to coordinate and monitor the program. Veterinarians, biologists, and animal curators and zookeepers should provide suggestions and information so that proper management is applied and risk to the animal collection is minimized. Keepers are usually the first to notice the presence of vermin and must communicate this to supervisors or the pest control officer. Personal protection equipment should always be used when applying pesticides, and the recommendations of the manufacturer must be followed.

METHODS OF PREVENTION AND CONTROL

Sanitation

All efforts should be made to maintain a high level of sanitation. Food wastes should be removed daily and, equally important, not be dumped in neighboring areas, which offer an ideal condition for the living and nesting of pests. Ultimately food availability determines the level of infestation. General control measures are applicable to many pest species, such as storing feed in rat- and insect-proof garbage bins with tight lids, which make the contents inaccessible to animals, storing garbage in animal-proof containers, and daily removal of animal and human refuse.

Removal of uneaten food in animal quarters before dark may be a difficult task in a large animal collection. One should also consider that some animals (such as small bird species) must not be deprived of food for a prolonged period of time in order to reduce the food available to nocturnal pest animals. Placing feeders in areas inaccessible to rodents may be more effective than food deprivation at night and dawn periods. Food storage should be built to be pest proof. Feed bags and boxes should be arranged in racks with adequate space for visual inspection and ventilation. The placement of baits in areas with abundant food is unproductive. Aviaries and animal enclosures should be cleaned daily, disinfected regularly, and monitored for the presence of undesired animals.

Habitat Management

Rodents, cockroaches, and other pests tend to live in close proximity to their foraging areas. Therefore, hiding places near enclosures and aviaries should be eliminated. Potential hiding places are logs inside aviaries, piles of stones, concrete slabs, boxes, holes, cracks and crevices, burrows, and pits. During the daytime, opossums and rats may find shelter in roofs, leaving their hiding places at night in search of food. Pruning trees and shrubs may prevent nests and access of small mammals to buildings by means of overhanging branches. Sand substrate and few plants inside aviaries favor a clear view of rat signs such as runs, burrows, and feces. Properly sealed doors may prevent the access of mice and cockroaches. It is important to learn about the biology, behavior, and physical abilities of pest animals in order to adopt effective and permanent methods of prevention and/or control.

Trapping

Traps may be useful in capturing some pest species. Opossums and feral or wild cats may be easily trapped in sliding door cage traps. Snap traps may be useful in controling rodents in areas where poisons are not advisable. Rarely is trapping effective as the only method of control, and better results are achieved with the use of attractive baits. Continuous use of traps may develop shyness in rodents. Coatis learn promptly to avoid traps. In order to increase trapping success, its use should be restricted to short periods, and traps should be correctly placed. Each zoo should establish its own policy for trapped wild animals. Options are removal of the offending animal to neighboring areas, relocation to distant areas, or euthanasia with the authorization of the state wildlife agency. Relocation programs should consider the risk of disease transmission to animals already resident in the relocation area, adaptation and survival of relocated animals in the new surroundings, and their impact on the new area. Felines such as jaguars, cougars, ocelots, and spotted cats may become a problem in facilities situated close to natural forests. The large felines have the habit of returning to their hunting area within a few days and may be trapped.

Physical Methods

Many zoos have experienced the misfortune of animal losses perpetrated by feral dogs. Perimeter fencing is the first physical barrier against dogs. To prevent dogs from squeezing under fences, wire mesh should be firmly fixed into a solid concrete base. Walls may prevent the access of venomous snakes and other ground-dwelling pests. Electric fencing is a useful exclusion method for rats and opossums, but is not foolproof, and eventually some animals may pass over. Electric fences marketed for cattle and horse pens are suitable for the zoo situation.

A blowtorch applied in open-sky areas distant from flammable gas (including sewer and refuse pits) and flammable material has been used effectively to disinfect animal enclosures and to dislodge rats from their burrows and eliminate them from areas where use of rodenticide is hazardous to animals in the collection. The use of protective equipment is necessary, such as protective glasses. One must remember that rats may cause considerable bird losses in one single night and urgent control action is sometimes necessary. Harassment on a regular basis may be effective to discourage the entry of such diurnal animals as coatis and vultures.

Poisoning

Zoos may have different policies regarding the use of poison in their area. Some zoos prefer to make use of pesticides as the last resort, when infestation has become so critical it puts the animal collection at risk. Others consider baiting a prophylactic method of control that prevents the proliferation of resident pest populations. Baiting is undertaken mainly to control rodents and invertebrates. Wild animals should not be poisoned because of legal and ethical constraints.

Only trained people wearing protective clothes and equipment should apply toxic substances. Necessary precautions to reduce the risk of intoxication to animals in the collection and nontarget species should always be taken. The least toxic methods should be tried first. Direct exposure of nontarget animals to bait may be prevented, but exposure to poisoned pest animals can be more difficult, and secondary intoxication may eventually occur. Special attention should be given when placing pesticides near enclosures of carnivores and insectivores, such as owls, hawks, toucans, and small cats. Rodents poisoned by tracking powders ingest a higher concentration of the active ingredient than is contained in food baits, so those carcasses represent a higher risk to animals in the collection. Thus this method of poisoning is not recommended in proximity to carnivore animals premises.

VERTEBRATES

Rodents

Rats and mice cause huge losses to agriculture worldwide. Rodents are also responsible for transmission of diseases to animals and humans (Table 41.1). In the zoo setting, the roof or black rat (*Rattus rattus*) and the Norway or sewer rat (*Rattus norvegicus*) prey on bird chicks, eggs, and small adult passerines. Rodents may destroy stored food and telephone cables and may damage buildings. Mice (*Mus musculus*) proliferate quickly under unsanitary conditions and abundant food. Because they are small, the primary harm to the animal collection is the risk of introduced diseases. Of the three species mentioned, the roof rat is the most noxious to the animals, because it is a voracious predator on small

TABLE 41.1. Infectious diseases potentially transmitted by pest species as hosts, carriers, or reservoirs

Pest Species	Diseases (agents)	Affected Species
Rats and mice	*Leptospira* spp.	Carnivores (otters, wolves, foxes), Primates
	Salmonella spp.	Mammals and birds
	Pasteurella spp.	Birds and mammals
	Yersinia pseudotuberculosis	Birds, callithrichids
	Lymphocytic choriomeningitis virus (callithrichid hepatites)	Callitrhichids
	Encephalomyocarditis virus	Monkeys, hoofed animals
Opossum	*Sarcocystis falcatula*	Old World psittacines
		Passeriformes, toucans
Bats	*Salmonella* spp.	Reptiles, birds, mammals
	Shigella spp.	Nonhuman primates
	Trypanosoma evansi (surra)	Equidae, Bovidae, Camelidae
	Venezuelan equine encephalitis (Alphavirus)	Equidae, Canidae, humans
	Rabies (Rhabdoviridae)	Mammals
Pigeons, sparrows	*Salmonella typhimurium*	Birds and mammals
	Salmonella enteritidis	
	Yersinia pseudotuberculosis	
	Yersinia enterocolitica	
	Chlamydiosis	
	Trichomoniasis	Pigeons and doves, raptors
Cockroaches	Salmonellosis	Reptiles, birds, mammals
	Amoebiasis	
	Giardiasis	
	Sarcocystis falcatula	Birds
	Pterygodermatites nycticebi (Spirurid nematode)	Callithrichidae
	Worms	Birds
Feral dogs and cats	Canine distemper, enteroviruses, toxoplasmosis	Carnivores

birds and can climb and reach the most inaccessible places, such as nests and roofs. Although larger than the roof rats, Norway rats tend to inhabit the floor, making their nests on the ground. As a result, locating nests and applying a rodenticide is easier. Anatomic differences among the three species may be found in the references.[9]

Rodents are extremely prolific, thus control measures must be rational and continuous. The elimination of rodents from an area is realistically unlikely, because neighboring populations will probably migrate to the "clean" area. It is important to know rodents' abilities, habits, and biology in order to control them successfully (Table 41.2). It is unlikely that it will be possible to eliminate food sources in a zoological park, but an effort should be made to reduce the amount of food and shelter available to pests. Sound hygiene practices and correct use of food may certainly reduce food availability. Keepers are usually the first to recognize signs of rodent infestation, including droppings, runs, burrows, gnawing marks, urine odors, smudge marks, tracks, and even visual sighting. Signs are useful to differentiate which rodent species are present (Table 41.3). Rats are nocturnal, and it is estimated that for every rat seen at night, 10 others are present. However more reliable methods of estimating rat populations are possible using rat traps, measuring food consumption, or by quantitative assessment of their signs.[9] Estimation of rat numbers is an important information to guide the use of a rodenticide. Effective rodent control requires an integrated management strategy, which includes prevention of rodent access, elimination of hiding places and nesting sites, reduction of food availability and control of the existing population with rodenticides.

TABLE 41.2. Some skills and habits of rodents

Skills	Use of pipes of 4–8 cm to climb.
	Young rats and mice may pass through mesh wire and openings of 5-mm diameter.
	The roof rat is a good climber, climbing along telephone cables, pipes, and branches of trees.
	Rats can jump obstacles 90 cm high and up to 1.2 m distant.
	Rats can fall safely from 15 m high.
	The Norway rat is good swimmer and has an interdigital membrane. It can stay submerged up to 3 minutes. This species lives close to sewers and constructs nests in below-ground burrows, usually with escape holes.
Habits	Nocturnal.
	Rats tend to use the same routes habitually.
	Range from the nest: sewer rats up to 45 m; black rats up to 60 m; mice up to 9 m.
	Rodents live in colonies.
	Norway rats may kill and rid the area of mice and roof rats.
	Rodents gnaw rigid materials.

Source: From reference 1.

PREVENTIVE MEASURES Whenever possible, buildings should be constructed so as to prevent entry ways for rodents. Cracks and gaps under doors and holes in the walls are potential ways of entry and should be covered with metal sheets or wired mesh smaller than 6 mm.[2] Vegetation should be eradicated for 1 m around buildings, and potential nest sites, such as logs and stones inside aviaries, weeds, refuse dumps, and open sanitary sewers should be eliminated. Norway rats are good swimmers and may inhabit domestic sewage. Whereas adult roof rats can squeeze through openings of 12 mm, mice and young roof rats need openings of only 6 mm to gain access to enclosures. Thus, most of the wire mesh used in zoos offers no protection against the entry of rodents.

Perimeter walls of at least 1.2 m high and below-ground fencing may prevent rodents from entering aviaries. Electric fences installed around aviaries are

TABLE 41.3. Rodent signs

Sign	Norway Rats	Roof Rats	Mice
Nests	Burrow system below ground or beneath piles of boards. Burrow entrance of 5 × 8 cm.	Burrow system below ground or in higher places, e.g., trees, bushes, or roofs.	Indoor or small holes on the ground. Mice nest in any hole or space: furniture, under slabs, holes in the wall, and next to animal feeders.
Feces	Like olive-stone of 6 × 16 mm.	Similar, but smaller: 5 × 10 mm.	Form and size similar to a rice grain.
Runs	Runs lead to the nest.	Runs on the ground and smudge marks in higher places. Fur leaves a greasy film.	Usually along walls.
Gnawing marks and rodent odors	Yes	Yes	Yes

useful to protect small birds against rodent predation. Catch trays may be placed under bird feeders to minimize food spillage onto the floor. Concrete slabs are ideal places for tunnels and nests and should be avoided or inspected regularly. Keepers and maintenance employees should be trained to monitor rodents' presence and inform the pest control officer.

RODENTICIDES AND PHYSICAL METHODS OF CONTROL Trapping may be effective when done properly and in association with other methods of control. Snap traps and wire-mesh cage traps have been used for many years. Rats are wary of new objects and may be trapped only after 4 or 5 days of exposure to the traps. For this reason rat traps should be placed in an area for 5 days before they are set. Another strategy is prebaiting trapping areas for several days before setting the traps, so that rats become used to the new objects. Mice are less shy and can be caught easily in snap traps baited with peanut butter. Rats may prefer fatty food items, such as bacon, hot dogs, and meat. After a few days of use, most rodents learn to avoid traps, so to minimize learning it is recommended that many traps be placed for a short period of time. Another suggestion is to increase effectiveness by placing double sets of snap traps (one next to the other) in the rodent's natural runway (usually close to a wall) with the trigger facing the wall.

The use of rodenticide is inevitable in most situations. Single-dose acute poisons, such as strychnine and arsenic, are banned in Brazil. Anticoagulants have been the preferred rodenticides and can be multiple dose or single dose. Broadifacoum and bromadiolone are potent single-dose rodenticides (death ensues usually after 4 days after a single feeding). This group of rodenticides presents some risk to nontarget animals, and their use in zoos should be monitored carefully.

Rodenticides may be formulated or can be purchased as dry baits, paraffin blocks, and tracking powders. Rats prefer high-quality foods and will reject baits of inferior food items. Grains (oats, corn, wheat or barley), nuts, meat, fish, bacon, fruits, and vegetables are the preferred food items. Grain-based pelleted baits packed in plastic bags are easy to place and last longer than home-formulated baits, because they are protected from humidity. Paraffin or wax bait blocks are useful for damp locations, such as sewers and pipelines. However, if more desirable food is plentiful in the area, rats may not accept paraffin baits. Bait stations may be made of cardboard (for indoor), wood, or polyvinylchloride (PVC) pipes, which has the advantage of protecting bait from moisture, providing a protected place for rodents to feed, protecting other animals from ingesting bait, and allowing baiting in locations hazardous to the animal collection. Bait stations should have two small openings on opposite sides and be labeled with warnings and signals. Tracking powders have a higher concentration of the active ingredient and are useful in places where food is plentiful. A patch of powder is applied along rats' runway, and as they walk over the dust they pick some of it up on their feet and ingest it while grooming. Tracking powder can also be pumped into rat burrows. Areas must be treated under dry conditions. Rodenticides should be used only by trained and licensed personnel using protective personal equipment in accordance with local rules. Baits containing vitamin D_3 are proclaimed to cause lethal intoxication in rodents and be much safer to nontarget species. Methods that offer the least risk to the collection animals should be chosen first.

Opossums

Opossums are marsupials ranging from Ontario, Canada, to the south of Argentina and occurring in a variety of habitats from grasslands to forests. Species that inhabit the three Americas are the Virginia or common opossum (*Didelphis virginiana*), southern opossum (*Didelphis aurita* or *D. marsupialis*), and white-eared opossum (*Didelphis albiventris*). The last two species occur only in South America. Weight varies from 2.5 to 5 kg and head–body length is within 35–55 cm. Opossums are nocturnal and terrestrial animals; however they are good climbers. Opportunistic feeders, their diet varies according to food availability. Fruits, insects, meat, small vertebrates, and food remains are some of their preferred food items. They are undoubtedly one of the best examples of wild species adaptable to urban areas, existing as pests in virtually all zoos. Roofs, holes, and shady places offer ideal conditions for hiding during the daytime. Nest boxes and burrows may also serve as diurnal hiding places in zoological parks. At night, solitary animals leave their shelter in search of food. Young animals may find small holes to enter aviaries and prey upon small birds, eggs, or chicks. However, the main threat to animals is the spreading of *Sarcocystis falcatula* oocysts. Though this protozoan is nonfatal and usually asymptomatic in opossums, it may become devastating in an Old World psittacine collection.[8] Deaths may also occur in New World psittacines.

Exclusion of opossums is the most practical method of control. Trapping near aviaries is effective, but a large number of traps are necessary to achieve rewarding results. Chicken meat or canned dog or cat food can be used as bait. Trapped opossums must be relocated to another area or humanely euthanized, if permission is granted by the official wildlife agency. Opossum feces found near aviaries should be promptly removed and destroyed, and the site should be burned or disinfected to prevent oocysts from remaining infective in the environment. Electric fences with a timer mechanism are useful to prevent opossums from climbing aviaries. Roofs and dark enclosures must be inspected regularly. Opossum urine odor may be easily recognized in their hiding places. Microscopic analysis of opossum intestines is needed to assess *Sarcocystis* prevalence within the resident population and the potential risk to birds in the collection.

Feral Dogs and Cats

Feral or domestic dogs and cats are common pests in zoos and should be controlled. They may cause animal deaths due to injuries or by disease transmission. Many zoos have experienced animal losses from their collection, mainly hoofstock and birds chased by dogs that enter enclosures at night. Although some animals are found with severe traumatic lesions, most of the deaths are results of stress syndrome: cardiovascular failure, shock, and in some cases exertion myopathy. Perimeter fencing is the first barrier against feral dogs, and if wire mesh is the chosen material, the fence should be firmly fixed to a concrete foundation, blocking any possible way of entry. Perimeter and enclosure fences should be regularly inspected for holes and signs of feral dogs, such as tracks, feces, and burrowing.

Trapping is recommended in areas of the zoo where potential predators have been spotted regularly. The services of the local animal control officer should be requested whenever necessary. Canine distemper disease outbreaks have been reported in maned wolves and other canids maintained in zoos in South America.[5] In some cases street dogs were incriminated as the source of infection. Wild canids and felines are susceptible to almost all of the infectious diseases that affect the domestic dog and cat, as well as to their helminthes and ectoparasites. For this reason, dogs and cats maintained as pets should not be allowed to enter zoos with visitors.

Cats may prey on caged birds if they can gain access, but usually prefer to wander around in search of mice. Despite the benefits attributed to domestic cats in controlling the mouse population, they should be banned from zoos exhibiting exotic felines because of the risk of introducing infectious diseases, such as viral rhinotracheitis, feline calicivirus, feline leukemia virus, panleukopenia, toxoplasmosis, and chlamydiosis. New World monkeys are particularly susceptible to toxoplasmosis, and clinical disease with high mortality (up to 100% in callithrichids) has been documented (P. H. Cubas, unpublished).

Cats may be easily caught in cage traps in areas where food availability is reduced. Hiding places, such as burrows under concrete slabs, dark shelters, and piled building materials should be regularly inspected and blocked to access or removed.

Wild Carnivores

Zoos and other animal facilities situated close to natural forests may be visited frequently by wild carnivores, such as coatis, raccoons, spotted cats, ocelots, cougars, and even jaguars. Felines normally attack at night and may kill a number of animals during a single visit. Predators have distinctive feeding patterns, which may help to identify the attacking species. Signs, such as feces, tracks, and animal fur may also be indicative of the aggressive species. Exclusion methods include secure housing at night: trapping and nocturnal vigilance by contracted watchmen. In areas where cougar incursions are frequent, electric fences are useful as a first line of defense. Free-living coati populations flourish in zoos situated close to forests, and bands may enter animal enclosures to steal food. They are indigenous and protected animals and should not be poisoned or trapped for euthanasia. Designating keepers or employees to permanently frighten bands of coatis out of the zoo area is one of the few practical measures to get rid of them.

Bats

The order Chiroptera is significant in South America. Brazil has approximately 140 species in eight families. More information about bats in South America can be found in the literature[1,2] and in Chapter 22. Fruit-eating, nectar-drinking, and insect-eating bats are the groups better adapted to urban areas, where they find great availability of food and shelter. Night lighting of streets, parks, and residences attracts flying insects and consequently insectivore bats. Frugiferous and nectariferous bats are attracted to areas where fruit trees are grown. Plants that serve as food for bats are in the references.[2]

Hematophagous bats (vampire bats) prefer rural areas where they may find their prey more easily (domestic and wild birds and mammals). There are three species on the continent: the common vampire (*Desmodus rotundus*), the hairy-legged vampire (*Dyphylla ecaudata*), and the white-winged vampire (*Diaemus youngii*) bat. The common vampire bat is the species of most concern to domestic animals and wild animals in captivity. Its distribution ranges from northern Argentina to northern Mexico.

Bats rest in dark areas during the day, preferring sites protected from rain, with ideal conditions of temperature and humidity. This place is known as their diurnal shelter and may be in caves, cracks in rocks, tree hollows, plants, or buildings. Night shelters or roosting sites are places where bats take their food to eat and where food remains and feces and urine are usually dropped. The mess they cause is probably the main complaint about their presence, besides the general popular belief that all bats are vampires. If roosting sites are close to aviaries or animal enclosures, the risk of disease transmission must be considered (Table 41.1).

Any bat found in zoos (independent of its food habit) that presents strange behavior, such as foraging during daytime, or nervous signs, or other abnormal behavior, is suspect for harboring rabies.

It was reported that the common vampire bat was feeding on the blood of wild animals in the Rio de Janeiro Zoo.[2] Trained personnel from a local agricultural agency usually carry out vampire bat control using anticoagulant vampirecide paste. Mist nets of 2-cm fine mesh may be used to catch fruit or insect eating bats.

Control of bats in an area demands reduction of their food supply and elimination of diurnal resting places. Buildings should be inspected regularly, watching for such signs as feces, smell, and squeaking. Entry holes to roofs may be in ridge beams or the edges of eaves. Other possible resting places are chimneys, attics, hollows in trees, or any place that is dark and calm. After finding resting areas, ways of entry should be sealed with proper material, making sure that all the resident bats are out of the site. Glass tiles and repellents such as formaldehyde and naphthalene may aid in dislodging bats from their hiding places. Ultrasound equipment seems to be ineffective.

Although attractive to the free-flying birds, fruit trees may also attract bats and pruning may be necessary to reduce fruit availability. When possible, complete removal of the fruits of such trees as palm and papaya is recommended. Ultimately, consideration should be given to the benefits that bats bring to the environment as pollinating agents and insect controllers. Any action of control causing the death of healthy bats should be considered only as a last resort.

Black Vultures

The black vulture (*Coragypis atratus*) occurs from the United States to southern Argentina. This species is particularly adapted to altered areas, often being found in urban areas where food is plentiful. In some zoos, hundreds of black vultures are usually seen near animal enclosures, detracting from the visitors' experience. These nuisance birds steal food from animals in the collection and may injure or kill small- and medium-sized species, harass pregnant females, or kill infants during labor or immediately after birth. Even though studies are lacking, black vultures are widely considered as potential vectors of diseases, such as salmonellosis and tuberculosis.

It is assumed that there is an overpopulation of black vultures around urban areas, explained by the fact that this species is of no economic interest and offers no harm to livestock, consequently have not been persecuted or hunted. They have few natural enemies and are resistant to diseases. Moreover, both rural and urban areas provide an abundance of food, such as animal carcasses, abattoir wastes, and open refuse dumps. Zoos are also preferred places for foraging because of the abundance of and easy access to food. When hungry, black vultures may learn to consume pelleted food or even chopped fruits and vegetables.

Many methods of vulture control have been tried in Brazilian zoos, but few have been successful. Restricting access to food is the best approach. Preventive measures include placement of food only under covered areas inaccessible to vultures, change of feeding times, and implementing a trapping and release program. Trapping is possible using large wire cages with a funnel-shaped entry baited with pieces of a carcass. A baiting wire cage measuring 3 m long, 3 m wide, 2 m high, with an entrance of 60 cm, was used successfully for many years in a zoo in Brazil (L.R. Francisco, personal communication, 1999). Trapped birds were translocated to an area at least 200 km away. Laws in many countries protect the black vulture, and poisoning and euthanasia are not allowed without permission of the local wildlife agency. Local communities recognize that black vultures are useful to the environment and die-offs by poisoning are not accepted. An effective measure of exclusion is to designate one or more employees to constantly chase vultures away from the zoo area, as part of a permanent program of control.

Herons and Egrets

The great egret (*Ardea alba*) and black-crowned night heron (*Nycticorax nycticorax*) are frequent predators of ducklings and other nidifugous species, fish, and small reptiles such as baby caimans and turtles. The accumulation of feces and urine under nesting areas is another inconvenience of their presence in public areas. Egrets and herons may also transmit pathogens, such as *Salmonella* spp., *Edwardsiella* spp., and parasites to birds (A. L. V. Nunes, personal communication, 1999).

The best control approach is the use of exclusion methods. Zoo animals that eat fish and meat should be fed in closed areas, or food should be placed in feeding devices inaccessible to free-ranging birds. In areas of natural lakes and ponds, food may be plentiful and populations of egrets may flourish. Nonresident birds may travel long distances to feed in the zoo area. Shade cloth suspended in the margins of tanks may protect young fish against wading birds, especially during the reproductive season. Devices to prevent egrets from roosting near the margins of ponds are a helpful preventive measure. Removal of nests and eggs near the zoo area is an effective measure to control resident populations. Unfortunately, nests are usually built in areas of difficult access and in the tops of high trees, making their removal a risky procedure for operators.

Pigeons and Sparrows

Pigeons (*Columba livia*) are common pests in urban areas worldwide. They often carry important pathogens, some of which are of zoonotic importance, such as salmonellosis, colibacillosis, chlamydiosis, histoplasmosis, and cryptococcosis. Other diseases that may be transmitted to the collection include coccidiosis, intestinal worms, and avian tuberculosis. It is difficult to rid the zoo of pigeons and sparrows, but a permanent control program may keep population size at an acceptable level. Although the chemical 4-aminopyridine (Avitrol in USA) has been reported as being useful in reducing populations of pigeons and gulls,[11] poisoning may cause an adverse reaction from the local community and animal welfare associations. The risk to nontarget species must also be taken into account when toxic chemicals are considered for use.

Continuous trapping in nonpublic areas may be effective in decreasing pigeon and sparrow populations. Large wire-cage traps with a narrowing funnel entry are preferred. Top-opening or sliding-door traps are able to catch only a few birds per set and need continual rechecking. Decoy birds left in or next to the trap increase trap efficiency. It may be necessary to change trapping sites periodically as birds learn to avoid them. Trapping efficiency reduces considerably within subsequent days. Pest control firms offer a complete assortment of mechanical devices and bird repellents, some of which promise a final solution to the problem of nuisance bird infestation. However, only combined methods of control and planned strategies of exclusion can reduce the nuisance bird population in zoos. Mechanical methods of exclusion are netting at roosting sites and holes that give access to shelter areas under roofs. Metal spikes may be either purchased or made and may be placed along roosting areas, such as ledges, eaves, terraces, windowsills, gutters, ornamental copings, protruding beams, and cornices. Fishing line and metal wire stretched over these structures are alternative materials to prevent roosting. Bird repellents advertised as being made of nontoxic, nonlethal, and noncorrosive substances may be used effectively to control pigeons. Manufacturers may be found locally or through the Internet. Reputable products may be worth a try if proven to be effective. These repellents are products that cause discomfort, itching, warmth, and irritation when in contact with feet. Repellents may be applied in roosting places. Some products are guaranteed for as long as 2 years when applied in outside areas. A bird repellent is produced from the native Brazilian plant *Loasa parviflora* and is guaranteed for 3 years (M. B. Ferreira, personal communication, 1999). Birds readily become accustomed to scare-eye balloons and ultrasonic sound devices.

It is almost impossible to stop nuisance birds from feeding in zoos. Even with all efforts to reduce availability of food and access to animal feeders, there will always be the problem of visitors who feed free-ranging pigeons in or near the area. But, as much as possible, food availability should be reduced. Food availability ultimately determines the size of the resident nuisance bird population. Periodic destruction of nests (every 10 to 14 days) is also helpful to control bird population.[11] Long poles may be used to remove nests in the lower branches of trees, but nests made in the highest branches of trees can be removed only with the assistance of trained personnel or a pest control firm.

Snakes and Tegus

Snakes may enter zoos and breeding areas in search of food; rodents and birds mainly. Nonvenomous snakes may prey on small birds, but this is rarely a problem in most zoos. Venomous snakes in search of rodents may enter aviaries and kill incautious ground birds, such as tinamous that inadvertently approach the snake, and small birds may eventually also become their prey. That they are risky to visitors and personnel is another important aspect to consider. Envenomation by *Bothrops* spp. has been documented in zoos. The best prevention is the implementation of an efficient rodent control program in the zoo and regular inspection along paths and other areas of visitors' access. Piling up construction materials and boxes correctly reduces shelters for snakes in proximity to buildings and aviaries. Extra attention should be paid during days of higher temperatures, as venomous snakes become more active (even during daytime) and the risk of a snake-human encounter is higher. Keepers should wear leather boots in areas of high incidence of venomous snakes. Cleaning of dumps, gardening, and other outdoor activities have a higher risk of exposure. Staff should be kept informed about snake-bite protocol, and in case of accident, take the victim to a hospital immediately.

The tegu lizard *Tupinambis merianae* is the most common lizard in Brazil.[7] In areas of natural occurrence it may become a problem during warmer periods of the year, when it becomes most active, and may enter enclosures in search of food. Tegus are omnivorous and eat eggs laid on the ground (pheasants, quails, tinamous, vulturines, and curassows). Larger tegus may attack small birds. A fine wire mesh at least 1 m high is recommended for aviaries housing small birds. Any hole must be blocked or sealed to prevent entry. Trapping may help to reduce the resident population of tegus.

INVERTEBRATES

Cockroaches

The presence of cockroaches causes embarrassment to zoo personnel in any situation. A negative stigma is attached to this insect, and the common conception is that cockroaches live in areas of poor sanitation and disease. In fact, what regulates the infestation level is the availability of food, water, and shelter. Cockroaches may be vectors of important diseases, such as salmonellosis, amoebiasis, and giardiasis.

Roaches may act as mechanical carriers of *Sarcocystis falcatula,* by eating opossum feces contaminated with sporocysts and subsequently defecating in psittacines' food.[3]

Cockroach anatomy consists of a head with antennae, a thorax with a plat-like structure (pronotum), and abdomen. The most common species are the German cockroach (*Blatella germanica*) and American cockroach (*Periplaneta americana*). German cockroaches are small (approximately 1.6 cm), light brown to brownish-yellow in color, and have two distinct longitudinal stripes on the pronotum. The adult American cockroach has reddish-brown wings, pale toward the margins, and the wings are longer than the abdomen, 2.5–3.8 cm in length.

The life cycle of the cockroach consists of three distinct phases: eggs, which are incubated in tough cases (the ootheca), nymphs or immature cockroaches, and adults. Nymphs molt as they grow; the number varies according to the species.

The German cockroach produces an egg case containing 30–45 eggs, which is carried by the female for approximately 3 weeks until the day of hatching. Nymphs molt 5–7 times in about 60 days before reaching adult stage. Whereas one female German cockroach produces 4–8 oothecas during her lifetime, the American cockroach produces in a single period of summer 12–24 oothecas with 10–14 eggs each. Unlike the German species, the American cockroach hides the egg case (ootheca) in cracks and crevices. Mature insects may live more than 1 year.[10]

Cockroaches congregate in dark areas near water and food. They prefer porous material for shelter, such as paper, cardboard boxes, paper bags, sacks, and wood. Living sites are marked with feces and pheromones. Cracks and crevices in kitchens, pipes and sinks in toilets, food deposit areas, and heated rooms, such as animal hospital areas, provide ideal condition for cockroach reproduction. Cannibalism of dead or injured cockroaches, egg cases, or nymphs occurs in cockroach populations. The new generation of cockroach baits takes advantage of this feeding practice. After feeding on baits at bait stations, roaches return to their hiding places to die. The bodies are eaten by other cockroaches that are secondarily poisoned and die.

PREVENTION The least toxic approach is always preferred. Hygiene and exclusion methods are essential for a long-term control. Various insecticides currently marketed are effective, but individuals may become resistant. There is also a risk to nontarget species, such as free-flying birds and insectivorous animals. Localization of infested and critical areas is the first approach in a good control plan. Keepers are usually the first to identify problem areas. Inspections should concentrate on areas that offer the resources described above. Signs include dead or live roaches, shed skins, egg cases, and feces (differentiate from mice feces). Check such holes of entry as sewer pipes, around drainpipes, and conduits for electricity. Avian nest boxes are preferred hiding places for cockroaches during the daytime. Almost all enclosures in zoos offer appropriate conditions for infestation. Removal of food residues as practiced for all pests should be instituted.

CONTROL There are substances and chemicals of low risk to humans and animals that may give variable results in controlling cockroaches. Under certain conditions, as in a low level of infestation, these techniques may be employed. Diatomaceous earth and silica aerogel are nontoxic substances used as desiccant agents. In contact with the insect they destroy the waxy protective layer of the insect body. Desiccants may be formulated with insecticides and can be used only in dry areas. Insect growth regulators (IGRs) are mildly toxic synthetic biochemicals that alter growth or body development, either killing the insect or preventing reproduction.[10] Hydroprene and fenoxycarb are two of the IGRs in use. The antiparasitic drugs ivermectin and abamectin, can be mixed into food and used as baits to reduce small infestations.

Insecticides are recommended when infestation is high.

Bees and Wasps

European honey bees (EHB) *Apis mellifera mellifera* and Africanized honey bees (AHB) *Apis mellifera scutellata* belong to the same species, but are of different subspecies. They are nearly identical physically and microscopically. Genetic examination is required to distinguish between the two subspecies. The well-recognized and feared aggressive behavior of Africanized bees is its most obvious trait. The Africanized honey bee resulted from the interbreeding between European bees and wild African bees. They were introduced accidentally into South America in 1957. Finding ideal environmental conditions to thrive, the AHB colonies have spread at the rate of 400 to 500 km per year, reaching the state of Texas in 1990 and southern California in 1994. Its southern limit is 34°S latitude in Argentina. With mild weather conditions and food availability, colonies are able to temporarily expand their southern limit to 40°S latitude. It is not yet known how far the AHB can extend their northern distribution, but colder temperatures are a limiting factor.

Africanized honey bees are more aggressive and may attack and cause death of people and confined animals. Some zoos in South America have reported losses of birds and small animals to bee attacks. Although there is no difference in the toxin potency between the two subspecies, Africanized bees attack in larger numbers, defend their hives more vigorously when disturbed, swarm more frequently, and pursue victims over a greater distance. It is accepted that 500 stings can kill an adult nonsensitized man. Making a simple correlation of body size, a parrot weighting 300 g might need only 2 stings to be at risk of death. Bee toxin may cause multisystemic disorders in humans, such as tubular necrosis, intravascular hemolysis, rhabdomyolysis, and cardiac and respiratory failure.[6] As an emergency procedure, stings should be removed quickly to reduce venom injection, ideally within seconds after the victim is stung, no matter if stings are scraped or pinched.[12] The Africanized subspecies is less demanding than the European bees for nesting sites. They can establish colonies in small cavities up high or near the ground, such as in flower pots, hollow trees, shrubs, cavities in walls, old tires, and even in bird nest boxes. Another characteristic is that the AHB abandon their hives (abscond) if disturbed and may fly long distances looking for nectar and pollen and a new place to establish the colony. These biological traits make the Africanized subspecies more likely to cause accidents and attack animals in captivity. Instructing personnel about the biology of AHBs and early recognition of bee presence is probably the best prevention against attacks.

Bees may nest close to buildings, so removal of dumps and debris that may provide shelter is recommended. Caulking holes and occluding cavities is another preventive measure. Trash receptacles should be kept closed and frequent removal of garbage around restaurants and other visitor eating areas are necessary to prevent stinging, especially during spring and summer when bee populations may proliferate. To control honeybees in picnic areas, trash receptacles and trash itself may be sprayed weekly with organophosphate and pyrethroid insecticides. To reduce honeybee attraction around picnic areas, serving soft drinks (colas) in cans may be preferred, rather than in disposable glasses, once soft drink machines have attracted honeybees. Trained specialized personnel, wearing protective clothing and using appropriate equipment, should remove the colonies. Beekeepers should be contacted for assistance.

Wasps will attack if disturbed close to their nest. Common nesting sites are low branches and eaves. Pyrethroids sprayed in the nest are an effective method of control. *Myschocyttarus drewseni* and *Polybia paulista* are some of the species that occur in Brazil.

Venomous Caterpillars

Caterpillars of the genus *Lonomia,* also known as the "killer Brazilian caterpillar," became a public health problem in southern Brazil during the spring and summer of 1989, when hundreds of cases were documented. From 1982 to 1996, 800 cases of envenomation were reported in people. One keeper and visitors were envenomed in a zoological park of Brazil. A few cases have also been reported in Venezuela. Caterpillar poisoning of animals has not been reported. Skin contact with *Lonomia* caterpillars cause fibrinolysis and a hemorrhagic syndrome. Clinical signs in humans include headache, fever, nausea, vomiting, hematomas, ecchymosis, hematuria, gingival bleeding, and, in severe cases, hypovolemic shock, acute renal failure, and cerebral hemorrhage. In 1993 the Instituto Butantan developed a *Lonomia* antivenom, reducing fatal accidents.

Feeding plants include native species and fruit trees. The insects feed at night and congregate on the trunk during daytime, taking advantage of the camouflage. Careful inspection of tree trunks located near public areas is recommended. Inspection should begin by the end of October, when caterpillars may start to appear. It is suspected that environmental disturbances created optimal conditions for a *Lonomia* population explosion.

Ants

Fire ants may become a problem in the zoo area. The author has seen psittacine chicks killed by fire ants while in the nest. To eradicate a colony of fire ants, it is necessary to kill the queen. The Audubon Institute (New Orleans, Louisiana, USA) has maintained an effective fire ant program (Roberto Aguilar, personal communication, 1999). Sugar ant and fire ant infestations may be reduced with a bait of 10 mg of ivermectin in 30 g of commercial peanut butter. This mix has the advantage of low toxicity to other animals (it may be used safely in butterfly exhibits), and it eliminates the whole colony since workers carry the bait to the nest, killing the queen (or queens) and larvae. Fire ants may have many breeding queens. Methods of control consist of locating nest sites, which are sprayed with pyrethroids, the ivermectin bait, and commercial ant baits that are broadcast throughout the area to be carried to the nests. Early spring is the preferred time to begin ant control in temperate zones. In tropical areas, the problem may persist throughout the year. The frequency of baiting and insecticide spraying should be determined for each region and according to the level of infestation.

REFERENCES

1. Barquez, R.M.; Giannini, N.P.; and Mares, M.A. 1993. Guide to the Bats of Argentina. Norman, Oklahoma, Oklahoma Museum of Natural History.
2. Bredt, A.; Araújo, F.A.A.; Caetano, J. Jr.; Rodrigues, M.G.R.; Yoshizawa, M.; Silva, M.M.S.; Harmani, N.M.S.; Massunaga, P.N.T.; Bürer, S.P.; Orto, V.A.R.; and Uida, W. 1996. Morcegos em Áreas Urbanas e Rurais: Manual de Manejo e Controle [Bats in urban and rural areas: Manual of control and management]. Brasília, DF, Fundação Nacional de Saúde, pp. 66–70.
3. Clubb, S.L. 1992. Sarcocystosis in psittacine birds. In R.M. Schubot, K.J. Clubb, and S.L. Clubb, eds., Psittacine Aviculture, Perspectives, Techniques and Research. Loxahatchee, Florida, Avicultural Breeding and Research Center, pp. 20-1–20-8.
4. Collins, D.; and Powell, D. 1996. Applied pest control at Woodland Park Zoological Gardens. In Proceedings of the Annual Meeting of the American Association of Zoo Veterinarians. Puerto Vallarta, Mexico, American Association of Zoo Veterinarians, pp. 290–295,
5. Cubas, Z.S. 1996. Special challenge of maintaining wild animals in captivity in South America. Revue Scientifique et Technique de L'Office International des Épizooties (OIE) 15(1):267–287.
6. Franca, F.O.S.; Benvenuti, L.A.; Fan, H.W.; et al. 1994. Severe and fatal mass attack by "killer" bees (Africanized honey bees, *Apis mellifera scutellata*) in Brazil: Clinocopathological studies with measurement of serum venom concentrations. Quarterly Journal of Medicine 87:269–282.
7. Francisco, L.R. 1997. Répteis do Brasil–Manutenção em Cativeiro. São José dos Pinhais, PR, Brazil, Gráfica e Editora Amaro, p. 134.
8. Godoy, S.N.; Cubas, Z.S.; De Paula, C.D.; Croukamp, A.A.; and Catão-Dias, J.L. 1999. Ocorrência de *Sarcocystis* sp. em pstiacideos mantidos em cativeiro [*Sarcocystis* sp. kept in captivity in Brazil]. In Proceedings of the 3rd Congresso e 8th Encontro da Associação Brasileira de Veterináro de Animais Selvagens (ABRAVAS) [3rd Congress and 8th Annual Meeting of the Brasizlian Association of Wildlife Veterinarians], Sao Pedro-SP, pp. 22.
9. Nagy, T. 1993. Normas Operacionais de Centros de Controle de Zoonoses(Procedimentos para o Controle de Roedores [Operating norms for centers of zoonosis control—Procedures for rodent control—Brasília-DF, Fundação Nacional de Saúde (National Health Foundation].
10. Ogg, B.; Ferraro, D.; and Ogg, C. 1995. Cockroach Control Manual. Lincoln, Nebraska, University of Nebraska Press.
11. Spelman, L.H. 1999. Vermin control. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, Vol. 4. Current Therapy. Philadelphia, W.B. Saunders, pp. 114–120.
12. Visscher, P.K.; Vetter, R.S.; and Camazine, S. 1996. Removing bee stings. Lancet 348(9023):301–302.

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Wild Animals and Public Health

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INTRODUCTION

Human interaction with the environment provides more information and knowledge about various ecosystems and about biodiversity. When this human–nature relationship is not adequately guided, it may lead to ecological changes with disastrous consequences for the environment, humans, and biodiversity. Climate or meteorological events also contribute to environmental degradation.[14]

High population density of humans and domestic animals and a highly developed agriculture enable more contact with wild species. These contacts allow infectious and parasitic agents to find new hosts and new environments where infection, maintenance, replication, and transmission are possible. As a consequence, zoonoses occur with epidemic expansion of hosts and geographical spreading.[9]

Knowledge about the epidemiology of these diseases becomes vital in preserving human and animal health. Certain risk factors associated with the environment trigger modified physiological responses and interference in the genetic expressions of humans and wild animals. Expansion of ecotourism and human occupation of forests have led to new diseases, many of which are considered to be emergent and high risk, such as hantaviruses, lyssaviruses, and morbilliviruses.[38]

To maintain different species of animals living in the wild, in zoos, in colonies for scientific or experimental purposes, or in households as pets, it is necessary to understand their biological and physiological behavior and their capacity to adapt to the environment. Knowledge about infectious and parasitic diseases related to these species is also important, particularly the diseases with potential to become a zoonosis, as well as knowledge about wild species that act as symptomatic or non-symptomatic reservoirs, transmitting diseases to domestic animals as well as to humans.[2,18,24]

When submitted to stressful situations, wild animals are more susceptible to disease-causing agents and may in turn become infection sources for animals of the same or other species. Major disease outbreaks may occur when animals are moved and quarantine procedures are inadequate.[25] In this situation, diseases with zoonotic characteristics become particularly relevant when dealing with wild animals involved in public health issues.

In Brazil, in spite of restrictions imposed by IBAMA (Brazilian Institute of Environment and Renewable Natural Resources), there is an increasing number of individual enthusiasts who insist on keeping birds, reptiles, and wild mammals in private captivity, especially in large urban centers. It is common for veterinary practices to receive these animals, exposing personnel to potential zoonoses.

ZOONOSES

Rabies

Rabies may affect any mammals and is a zoonosis characterized by a fatal acute encephalitis, resulting from the transmission of rabies virus through the bite of an infected animal. It is caused by an RNA virus, family *Rhabdoviridae,* genus *Lyssavirus,* with several serotypes: serotype 1, classic or wild; serotype 2, Lagos bat; serotype 3, Mokola; serotype 4, Duvenhage; serotype 5, European bat *Lyssavirus* 1 (EBL1); and serotype 6, European bat *Lyssavirus* 2 (EBL2). The disease occurs in most countries, either as an endemic or epidemic disease, with urban and wild cycles. Most human cases in South America result from rabid-dog bites. Rabies transmitted by vampire bats is a problem limited to Latin America and is responsible for losses in cattle herds.[2,56]

Wild species are usually involved in maintaining the rabies virus in nature. Besides bats, the following animals have been reported as being reservoirs in the Americas: skunk *Mephitis mephitis,* foxes *Vulpes fulva, Urocyon cinereoargenteus,* raccoon *Procyon lotor,* paca *Agouti paca,* coatimundi *Nasua nasua,* and mongoose *Herpestes auropunctatus*. Many other species may acquire the virus from a reservoir host and expose other animals. Vampire *Desmosdus rotundus* and insectivorous bats serve as reservoirs.[2,3]

The disease in animals may be the furious or the paralytic (dumb) forms. Rabies occurs in many carnivore and other wild mammals. The most sensitive are foxes, wolves, coyotes, and jackals. Opossums, raccoons, bats, and mongooses have the lowest sensitivity. Clinical signs are similar to that in domestic animals with exacerbated excitement, anorexia, pruritus at the bite site, aggressiveness, abundant salivation, motor incoordination and muscle paralysis, and death. Presence of antibodies against rabies virus has been reported in several wild animal species, such as foxes, raccoons, mongooses, and insectivorous bats, suggesting that infection does not always lead to disease and death.[2]

Wild rabies is maintained in nature in a manner similar to urban rabies. One or two species of mammals, especially carnivores and bats, are responsible for perpetuating the rabies virus in a certain ecosystem. In several areas different animal species maintain more or less independent epizootics. When population density is high, rabies assumes epizootic proportions, and a large number of animals die. On the other hand, when the population numbers are small, rabies may become enzootic and tends to disappear with time. When there is a new generation of susceptible animals, a new epizootic occurs.[2]

The primary transmission route of wild and urban rabies is through the bite of a rabid animal. The airborne and digestive routes are less important.[2] There are few references in Brazil about rabies occurrence in wild animals. In Rio de Janeiro,[44] from 1965 to 1974, rabies was diagnosed in 7 marmosets, 5 monkeys, 4 deer, 2 rats, and 1 squirrel kept in captivity or considered as pets. In São Paulo,[27] from 1971 to 1978, rabies was reported in 6 free-ranging marmosets, 8 monkeys, 1 ocelot, 1 crab-eating fox (*Dusicyon thos*), 1 little spotted cat (*Leopardus tigrina*), and 1 deer. In Brazil,[46] rabies cases were reported in the period from 1979 to 1982 in 15 monkeys, 1 crab-eating raccoon (*Procyon carnivorous*), 1 capybara (*Hydrochoerus hydrochaeris*), 1 jaguar (*Pantera onca*), 7 savannah fox (*Dusicyon thos*), 1 coatimundi (*Nasua nasua*), 1 deer (*Mazama americana*), and 1 otter (*Lutra* sp.). From 1989 to 1998, rabies was reported in Ceará state,[41] in foxes from Serra de Ibiapaba, in callithricidae, as well as in humans with cases transmitted by a crab-eating raccoon (*Procyon carnivorous*) in 1997 and by a marmoset (*Callithrix* sp.) in 1998.

According to the Brazilian National Rabies Control Program (National Coordination for the Control of Zoonosis and Poisonous Animals, National Epidemiology Center, National Health Foundation, Ministry of Health), such wild animals as monkeys, foxes, opossums, wild cats, and peccaries were responsible for human rabies transmission in 11, 16, 1, 3, and 1 cases, respectively, in the period from 1980 to 1996.

When Neotropical primates were immunized with killed rabies vaccines for dogs and cats, given by subcutaneous or intramuscular injection, an immune response was induced, with production of neutralizing antibodies in captive *Callithrix* sp,[8,12] *Cebus apella,*[43] and *Saimiri* sp.[29] The use of rabies vaccine with live modified virus should never be used for wild animals, as rabies induced by vaccination has been reported in *Saimiri sciureus*[49] and *Saguinus nigricollis.*[1]

Control of wild rabies is based on reducing the population of the species most involved in virus transmission and responsible for maintaining the transmission cycle, with consequent decrease of infection cases. The methods used may be poisoning, hunting with firearms, immunization with oral vaccines in free-living populations (foxes, wolves, raccoons) and captive populations in areas with endemic rabies. In rabies-free countries, entrance of animals from enzootic areas should be forbidden or a 6-month quarantine should be established for newly acquired animals, as well as immunization of the animals with inactivated rabies vaccines.[2]

High-risk groups such as veterinarians, naturalists, biologists, animal caretakers, and nurses, among others, should receive prophylactic vaccines against rabies before being exposed to bites.

Leptospirosis

Leptospirosis is a worldwide zoonosis. It is highly prevalent in tropical and underdeveloped countries. In humans it is considered as an occupational disease, affecting veterinarians, animal caretakers, farmers, sugarcane harvesters, slaughterhouse workers, and other professionals. It also occurs in domestic and wild animals.[2,6] Leptospira are classified as spirochetids, belonging to the order Spirochaetales, family Leptospiraceae, genus *Leptospira. Leptospira interrogans* is the pathogenic species and *Leptospira biflexa* the free-ranging species. Spirochetids survive in water and moist soils with neutral pH.[2]

The infection is common in rodents, which are reservoirs that exhibit no clinical signs or lesions. Leptospira locate in their kidneys and are eliminated in the urine, contaminating the environment and foods, a major factor in the transmission cycle of the disease, in addition to contact with infected animals. In several areas of the world, studies in wild animals have demonstrated the presence of leptospira in many rodents, edentata, and carnivore species, and they may act as infection sources.[2]

In exotic fauna, leptospirosis plays a major role in clinical disease causing low fertility rates, birth of weak offspring, abortions, and eye disorders. It is important to know the reservoirs in the wild fauna and obtain more information on the epizootiology of this disease.[6]

Among South American animals, marsupials such as opossums (*Didelphis marsupialis*) have different serovars, such as *ballum, bataviae, szwajizam, icterohaemorrhagiae,* and *grippotyphosa,*[51] and edentata, such as nine-banded armadillo (*Dasypus novemcinctus*), have *grippotyphosa, cynopteri,* and *hebdomadis* serovars.[36] In primates, *Leptospira* sp. was isolated from *Saguinus midas midas, Pithecia monachus,* and *Saguinus midas niger.*[37]

Reptiles are considered to be reservoirs, because leptospira may be found in clinically normal reptiles. Studies have shown positive serology in South American species of venomous and nonvenomous serpents, suggesting that infected rodents on which they have preyed could have caused the infection. The reported serovars were *andamana, ballum, icterohaemorrhagiae, biflexa, grippotyphosa,* and *pomona.*[32,52]

Salmonellosis

Salmonellosis is a classic zoonosis caused by various *Salmonella* sp., with food contamination being the main transmission route. It may cause enterocolitis, common in exotic animals, especially under stress situations. This agent is also present in ectothermic animals.[40] Human infection may be related to exposure to reptiles kept as exotic pets, and indirect contamination is frequent (contaminated hands and food).[2]

Salmonellosis has been reported from a large variety of wild mammals, including cervids, rodents, edentata, marsupials,[4] and felines.[15] Both New World and Old World primates may exhibit the infection, which is a major cause of mortality in these animals.[4,5,11,30,53] *Salmonella* sp. was isolated from capybara feces (*Hydrochoerus hydrochaeris hydrochaeris*).[42]

Campylobacteriosis

The *Campylobacter* genus contains several species involved in public and animal health, *Campylobacter jejuni/coli* being responsible for major gastroenteric diseases in humans and animals. Human contamination occurs by indirect routes, exposure to animals with diarrhea, or water and food contamination.[2] Severe diarrhea in primates is associated with this agent, and findings have been reported in *Callithrix* and *Saguinus* kept in captivity.[47] In South American rodents,[42] presence of *Campylobacter jejuni* has been reported in feces of capybaras (*Hydrochoerus hydrochaeris hydrochaeris*).

Shigellosis

Shigella sp. is the cause of severe enteric disease in animals and humans, particularly primates, causing diarrhea difficult to control and eradicate.[11] Both in humans and animals, transmission occurs by indirect route, by contact with carriers and contaminated objects.[2] When compared with other primate species, Neotropical primates of the *Callithrix* and *Saguinus* genera are more sensitive to infection by *Shigella.*[47]

Chlamydiosis

Chlamydia is an obligatory intracellular organism, classified in the order Chlamydiales, family Chlamydiaceae, with only one genus, *Chlamydia,* and four species *Chlamydia psittaci, Chlamydia trachomatis, Chlamydia pneumoniae,* and *Chlamydia pecorum.* It is considered to be a major zoonosis because it causes alterations in the respiratory system also known as psittacosis or ornithosis. It is present mainly in wild birds, causing major gastroenteric, urinary, reproductive, neurological, and respiratory diseases.[28] Airborne transmission to humans occurs in contaminated environments, and the disease is transmitted to birds by airborne and digestive routes.[2]

C. psittaci was reported in *Amazona* genus parrots[48] without any clinical manifestation, characterizing a nonapparent carrier in this species. As to South American mammals, it has been reported in llamas with abortion and birth of weak offspring.[34] It has also been reported in reptiles[33] and amphibians.[17]

Tuberculosis

The etiological agents of tuberculosis in mammals are *Mycobacterium tuberculosis* (main agent of human tuberculosis), *Mycobacterium bovis* (cattle tuberculosis), and *Mycobacterium africanum* (human tuberculosis in Africa). Zoonotic tuberculosis is caused by *Mycobacterium bovis*. The disease has a worldwide distribution, with major variations in different regions and countries.[2] The prevalence of human tuberculosis originated from animals has decreased, but zoo caretakers are exposed to different agents of animal tuberculosis and may develop a positive reaction to the disease.[20,39,54]

Humans may transmit the human-type bacillus to several animal species, mainly primates and dogs. Many domestic and wild mammal species are susceptible to tuberculosis agents.[2,31] In general, animals living in the wild, far from humans and domestic animals, do not have tuberculosis. However, animals kept in captivity in zoos, commercial breeding and research centers, or in households, may be exposed to the etiological agent and become infected through cattle meat or viscera or birds with tuberculosis. Primates are susceptible to *M. tuberculosis, M. bovis* and *Mycobacterium avium* and become infected by airborne or digestive routes. Infection may spread from one primate to another and become a severe problem in colonies maintained by scientific institutions and zoos. These animals may also retransmit the disease to humans. Nonhuman primates with tuberculosis are a risk for public health.[2] *M. tuberculosis* was isolated from a captive capuchin monkey (*Cebus apella*) that exhibited all the signs. After euthanasia, tissue samples were collected for isolation, and the agent was found in lymph nodes and lungs.[26]

In nonhuman primates, the diagnosis is made by tuberculin testing, with intradermal inoculation of mammal tuberculin (purified protein derivative; PPD) on the eyelid and by radiological examinations. Control of the disease is based upon repeated tuberculin testing and euthanasia of animals with positive reactions, avoiding consumption of animal products such as milk, meat, and their by-products without hygiene inspection (Federal Inspection Service), and preventing people with tuberculosis from working with animals (periodical radiographs are required).[2]

Toxoplasmosis

The etiological agent of toxoplasmosis is *Toxoplasma gondii,* protozoan of the order Coccidia, and an obligatory intracellular parasite, with a very complex life cycle: enteroepithelial in the definite host (domestic and wild felines) and extraenteric in intermediate hosts. The disease has worldwide distribution and is one of the most widely spread zoonoses. It is estimated that one-third of the world's population has antibodies against the parasite. Subclinical infection is most common, and the congenital or acquired clinical disease is rare.[2]

Domestic and wild felines of *Felis* and *Lynx* genera are definitive hosts of this parasite and may become infected by tachyzoite, bradyzoite, and oocyst forms present in raw meat, mice, or birds with cysts. There is a sexual cycle in the intestine of felines, and oocysts are eliminated through the feces, contaminating the environment, soil, and pastures for a 3–15 day period, until immunity is developed. They also have the extraenteric, asexual, or tissular parasite cycle. Intermediate hosts are infected when they ingest meat containing cysts or oocysts. Toxoplasmosis has been detected in animals by serological tests or isolation and has been reported in 200 domestic and wild mammal species. Many bird species also harbor the parasite. Most infections are inapparent; clinical presentations may occur sporadically, similar to that of humans.[2]

Several wild species are susceptible to infection by *Toxoplasma gondii:* felines, cervidae, marsupials, and primates.[1] There are reports in South American primates: squirrel monkey (*Saimiri sciureus*),[7,19,21] golden lion marmoset (*Leontopithecus rosália*),[35] lion-headed golden marmoset (*Leontopithecus chrysopygus*),[23] golden-headed lion marmoset (*Leontopithecus chrysomelas*),[22] Spix's moustached tamarin (*Saguinus imperator*),[22] *Saguinus,* and *Callithrix,*[10,47] and in Cebideae,[10,13] night monkey (*Aotus* sp.), howler monkey (*Alouatta* sp.), spider monkey (*Ateles* sp.), and woolly monkey (*Lagothrix* sp.).[10] Clinical signs involve lethargy and digestive and nervous disorders, but death from the disease may ensue without evident clinical signs being observed.[21] Toxoplasmosis has also been reported in birds, causing skeletal muscle and brain lesions.

Disease control is based on supervision of feline feeding, avoiding the use of raw or poorly cooked meat, adequate disposal of excreta, and decreasing the feline population in breeding facilities. As to prevention of human toxoplasmosis, pregnant women and immunodepressed patients should receive special attention, avoiding contact with felines eliminating oocysts with the feces, consumption of raw or poorly cooked meat, and handling raw meat and feline feces. Hygiene is very important during food handling and feline management. Populations of arthropods that can propagate oocysts should be controlled.

REFERENCES

1. Aaron, E.; Kamei, I.; Bayer, E.V.; Emmons, R.W.; and Chin, J. 1975. Probable vaccine-induced rabies in a pet marmoset, California. MMWR Morbidity and Mortality Weekly Report 24:99.
2. Acha, P.N.; and Szyfres, B. 1986. Zoonosis y Enfermidades Transmisibles Comunes al Hombre y a los Animales. Organizacion Panamericana de la Salud Publicación Científica No. 503. Washington, D.C., Organizacion Panamericana de la Salud, p. 989.
3. Acha, P.N.; and Arambulo, P.V. 1985. Rabies in the tropics: History and current status. In E. Kuwert, C. Mérieux, H. Koprowski, and K. Bögel, eds., Rabies in the Tropics. Berlin, Springer-Verlag, pp. 343–359.
4. Adesiyun, A.A.; Caesar, K.; and Inder, L. 1998. Prevalence of salmonella and campylobacter species in animals at Emperor Valley Zoo, Trinidad. Journal of Zoo Wildlife Medicine 29(2):237–239.
5. Adesiyun, A.A.; Seepersadsingh, N.; Inder. L.; and Caesar, K. 1998. Some bacterial enteropathogens in wildlife and racing pigeons from Trinidad. Journal of Wildlife Diseases 34(1):73–80.

6. Alvarez, M.A.L.; Cervantes, L.P.M.; Barranca, J.I.T.; and Still, F.G. 1996. Investigacion serológica de leptospirosis en fauna silvestre mantenida en cautiveiro en el Zoológico de Chapultepec de la Ciudad de México. Veterinaria México 27(3):229–234.
7. Anderson, D.C.; and McClure, M.H. 1982. Acute disseminated fatal toxoplasmosis in a squirrel monkey. Journal of the American Veterinary Medical Association 181:1363– 1366.
8. Andrade, M.C.R. 1997. Avaliação da resposta imunológica produzida por vacinas anti-rábicas em primatas não humanos (*Callithrichidae*) [Evaluation of immune response produced by rabies vaccines in non human primates]. Masters thesis, Instituto Oswaldo Cruz, Rio de Janeiro, p. 128.
9. Barlett, P.C.; and Judge, L.J. 1997. The role of epidemiology in public health. Office International des Epizooties Scientific and Technical Review 16(2):331–336.
10. Bouer, A.; and Werther, K. 1999. Levantamento sorológico para toxoplasmose (*Toxoplasma gondii*) na população de primatas do Parque Zoológico Municipal “Quinzinho de Barros”—Sorocaba/SP [Serological survey for toxoplasmosis in the primate collection of Quinzinho de Barros County Zoo Park]. In Anais 3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens. São Pedro-SP, ABRAVAS, p. 21.
11. Brack, M. 1987. Agents Transmissible from Simians to Man. Berlin, Springer-Verlag, p. 454.
12. Cabrera, M.A.A.; Figueiredo, M.J.; Silva, W.C.; Ferrer, D.; Teixeira, R.H.; Cavalheiro, M.L.J.; Andrade, M.C.R.; Fernandes, R.C.; and Moura, W.C. 1991. Antibody response to a inactivated rabies vaccine in some species of Brazilian monkeys. In Abstracts 24th World Veterinary Congress. p. 322.
13. Cadavid, A.P.; Canas, L.; Estrada, J.J.; and Ramirez, L.E. 1991. Prevalence of anti-*Toxoplasma gondii* antibodies in *Cebus* spp. in the Santa Fe Zoological Park of Medellin, Colombia. Journal of Medical Primatology 20(5):259–261.
14. Childs, J.; Shope, R.E.; Fish, D.; Meslin, F.X.; Peters, C.J.; Johnson, K.; Debess, E.; Dennis, D.; and Jenkins, S. 1998. Emerging zoonoses. Emerging Infectious Diseases 4(3):453–454.
15. Clyde, V.L.; Ramsay, E.C.; and Bemis, D.A. 1997. Fecal shedding of salmonella in exotic felids. Journal of Zoo Wildlife Medicine 28(2):148–152.
16. Cosivi, O.; Grange, J.M.; Daborn, C.J.; Raviglione, M.C.; Fujikura, T.; Cousins, D.; Robinson, R.A.; Huchzermeyer, H.F.A.K.; de Kantor, I.; and Meslin, F.-X. 1998. Zoonotic tuberculosis due to *Mycobacterium bovis* in developing countries. Emerging Infectious Diseases 4(1):59–70.
17. Crawshaw, G.J. 1993. Amphibian medicine. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 3rd Ed. Philadelphia, W.B. Saunders, pp. 131–139.
18. Cubas, Z.S. 1996. Special challenges of maintaining wild animals in captivity in South America. Office International des Epizooties Scientific and Technical Review 15(1):267–287.
19. Cunningham, A.A.; Buxton, D.; and Thomson, K.M. 1992. An epidemic of toxoplasmosis in a captive colony of squirrel monkeys (*Saimiri sciureus*). Journal of Comparative Pathology 107(2):207– 219.
20. Dalovisio, J.R.; Stetter, M.; and Mikota-Wells, S. 1992. Rhinoceros’ rhinorrhea: Cause of an outbreak of infection due to airborne *Mycobacterium bovis* in zookeepers. Clinical Infectious Diseases 15(4):598–600.
21. Dreesen, D.W. 1990. *Toxoplasma gondii* infections in wildlife. Journal of the American Veterinary Medical Association Jan 15 196(2):274–276.
22. Epiphanio, S.; Dias, J.L.C.; Guimarães, M.A.B.V.; Corrêa, S.H.R.; and Fedullo, J.D.L. 1997. Ocorrência de toxoplasmose em *Saguinus imperator* e *Leontopithecus chrysomellas* mantidos em cativeiro [Occurrence of toxoplasmosis in captive *Sanguinus imperator* and *Leontopithecus chrysomellas*]. In Proceedings of the 2° Congresso Medicina Veterinária do Cone Sul. Gramado-RS, Sociedade de Veterinaria do Rio Grande do Sul and Sociedade Brasileira de Medicina Veterinária.
23. Epiphanio, S.; Marques de Sá, L.R.M.; Teixeira, R.H.; and Dias, J.L.C. 1999 Toxoplasmose em mico-leão-preto (*Leontopithecus chrysopygus*): Relato de caso [Toxoplasmosis in black lion tamarin *Leontopithecus chrysopygus:* A case report]. In Anais 3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens. São Pedro-SP, ABRAVAS, p. 24
24. Fowler, M.E. Ed. 1986. Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders.
25. Fowler, M.E. 1996. An overview of wildlife husbandry and diseases in captivity. Office International des Epizooties Scientific and Technical Review 15(1):15–42.
26. Freitas, J. De A.; Ueki, S.Y.M.; Curcio, M.; and Tury, E. 1998. *Mycobacterium tuberculosis* associado a tuberculose em macacos *Cebus apella* mantidos em cativeiro [Tuberculosis associated with *Mycobacterium tuberculosis* in captive capuchin monkeys *Cebus apella*]. In Anais 3° Seminário Nacional de Zoonoses e Animais Peçonhentos. Guarapari-ES, Secretaria de Estado da Saúde do Espirito Santo—Coordenadoria de Epidemiologia—Coordenação do Risco Ambiental/Zoonoses, p. 140.
27. Gambeta, W.R.; Chamelet, E.L.B.; Souza, L.T.M.; and Azevedo, M.P. 1979. Instituto Pasteur de São Paulo, 75 Anos de Atividade (1903(1978): Análise Histórica de Sua Atuação Técnica e Científica na Profilaxia da Raiva [Pasteur Institute of São Paulo, a 75-year review (1903–1978): Historical analyses of technical and scientific actions in rabies prophylaxis]. São Paulo, Instituto Pasteur/Coordenadoria de Serviços Técnicos Especializados da Secretaria de Estado da Sa£de de São Paulo.
28. Gerlach, H. 1994. Chlamydia. In B.W. Richie, G.J. Harrison, and L.R. Harrison, eds., Avian Medicine: Principles and Application. Lake Worth, Florida, Wingers Publishing, pp. 984–996.
29. Haigh, J.C.; and Field, M.F. 1981. Rabies vaccination in a small zoo: Antibody titers studies. Journal of Zoo Animal Medicine 12:17–20.

30. Hunt, R.D.; Anderson, M.P.; and Chalifoux, L.V. 1978. Spontaneous infections of marmosets. Primate Medicine 10:239– 253.
31. Hunter, D.L. 1996. Tuberculosis in free-ranging, semi free-ranging and captive cervids. Office International des Epizooties Scientific and Technical Review 15(1):171–181.
32. Hyakutake, S.; Biase, P.; Santa Rosa, C.A.; and Belluomoni, H.E. 1976. Contribuição ao estudo epidemiologico das leptospiras em serpentes do Brasil. Contribution to epidemiological studies of leptospiras in Brazilian snakes. Revista do Instituto de Medicena Tropical São Paulo 18(1)10–16.
33. Jacobson, E.J. 1993. Blood collection techniques in reptiles: Laboratory investigations. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, 3rd Ed. Philadelphia, W.B Saunders, pp. 144–151.
34. Johnson, L.W. 1993. Abortion in llamas. In M.E. Fowler, ed., Zoo and Wild Animal Medicine. Philadelphia, W.B. Saunders, pp. 541–544.
35. Juan-Salles, C.; Prats, N.; Marco, A.J.; Ramos-Vara, J.A.; Borras, D.; and Fernandez, J. 1998. Fatal acute toxoplasmosis in three golden lion tamarins (*Leontopithecus rosalia*). Journal of Zoo Wildlife Medicine 29(1):55–60.
36. Lins, Z.C.; and Lopes, M.L. 1984. Isolation of leptospira from wild forest animals in Amazonian Brazil. Transactions of the Royal Society of Tropical Medicine and Hygiene 78:124–126.
37. Marques de Sá, L.R.; Teixeira, R.H.F.; Di Loreto, C.; and Dias, J.L.C. 1999. Leptospirose em primatas neotropicais [Leptospirosis in neotropical primates]. In Anais 3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens. São Pedro-SP, ABRAVAS, p. 7.
38. Meslin, F.X. 1997. Global aspects of emerging and potential zoonoses: A WHO perspective. Emerging Infectious Diseases 3(2):223–228.
39. Michalak, K.; Austin, C.; Diesel, S.; Maichle Bacon, J.; Zimmerman, P.; and Maslow, J.N. 1998. Mycobacterium tuberculosis infection as an zoonotic disease: Transmission between humans and elephants. Emerging Infectious Diseases 4(2):283–287.
40. Minette, H.P. 1984. Epidemiologic aspects of salmonellosis in reptiles, amphibians, mollusks and crustaceans—A review. International Journal of Zoonoses 11(1):95–104.
41. Morais, N.B.; Silva, L.M.; and Rolim, B.N. 1998. Raiva humana—Relato de um caso de transmissão por sagui [Human rabies: A case report of transmission by a marmoset]. In Anais 3° Seminário Nacional de Zoonoses e Animais Peçonhentos. Guarapari-ES, Secretaria de Estado da Saúde do Espirito Santo—Coordenadoria de Epidemiologia—Coordenação do Risco Ambiental/ Zoonoses, p. 85.
42. Nogueira, M.M.; Lopes, C.A.M.; Modolo, J.R.; and Sadatsune, T. 1999. Avaliação da presença de *Enterobacteriaceae,* Aeromonas, Campylobacter e Cryptosporidium em fezes de capivaras, *Hydrochoerus hydrochaeris hydrochaeris* (L. 1766) e determinação do perfil de susceptibilidade bacterinanas a diferentes drogas [Detection of the presence of Enterobacteriaceae, Aeromonas, Campylobacter and Cryptosporidium in the feces of capybaras *Hydrochoerus hydrochaeris* an determination of bacterial susceptibility to different drugs]. In Anais 3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens. São Pedro-SP, ABRAVAS, p. 34.
43. Passos, E.C. 1998. Evaluation of Inactivated Suckling Mouse Brain Rabies Vaccine for Immunization of Capuchin Monkeys (*Cebus apella,* Linnaeus, 1758) Held Confined. Doctoral thesis, University of São Paulo School of Public Health, p. 77.
44. Passos, J.J.; Campello, I.B.; Figueiredo, M.J.; and Marmori, Z.; and Bastos, C.S.P. 1974. Raiva em animais silvestres [Rabies in wild animals]. In Resumos 14° Congresso Brasileiro de Medicina Veterinária. São Paulo-SP, Sociedade Paulista de Medicina Veterinária and Sociedade Brasileira de Medicina Veterinária, p. 103–104.
45. Pertz, C.; Dubielzig, R.R.; and Lindsay, D.S. 1997. Fatal *Toxoplasma gondii* infection in golden lion tamarins (*Leontopithecus rosalia rosalia*). Journal of Zoo Wildlife Medicine 28(4):491–493.
46. Piccinini, R.S.; and Freitas, C.E.A. 1985. Experiences with rabies control in Brazil. In E. Kuwert, C. Mérieux, H. Koprowski, and K. Bögel, eds., Rabies in the Tropics. Berlin, Springer-Verlag, pp. 737–741.
47. Poltkay, S. 1992. Disease of the callitrichidae: A review. Journal of Medical Primatology 21:189–236.
48. Raso, T.F.; and Berchieri, A., Jr. 1999. Detecção de infecção por *Clamydia psittaci* em papagaios do gênero *Amazona* mantidos em cativeiro [Infection by *Chlamydia psittaci* in parrots of the genus *Amazona* kept in captivity]. In Anais 3° Congresso e 8° Encontro da Associação Brasileira de Veterinários de Animais Selvagens. São Pedro-SP, ABRAVAS, p. 6.
49. Richardson, J.H.; and Humphrey, G.L. 1971. Rabies in imported nonhuman primates. Laboratory Animal Science 21:1082–1083.
50. Juan-Salles, C.; Prats, N.; Marco, A.J.; Ramos-Vara, J.A.; Borras, D.; and Fernandez J. 1998. Fatal acute toxoplasmosis in three golden lion tamarins (*Leontopithecus rosalia*). Journal of Zoo Wildlife Medicine 29(1):55–60.
51. Santa Rosa, C.A.; Sulzer, C.R.; Giorgi, W.; Da Silva, A.S.; Yanaguita, R.M.; and Lobão, A.O. 1975. Leptospirosis in wildlife in Brazil, isolation of a new serotype in the pyrogenes group. American Journal of Veterinary Research 36(9)1363–1365.
52. Santa Rosa, C.A.; Sulzer, C.R.; Yanaguita, R.M.; and Da Silva, A.S. 1980. Leptospirosis in wildlife in Brazil: Isolation of serovar *canicola, pyrogenes* and *grippotyphosa.* International Journal of Zoonoses 7:40–43.
53. Schiefer, B.; and Loew, F.M. 1978. Amebiasis and salmonellosis in a woolly monkey (*Lagothrix*). Veterinary Pathology 15(3):428–431.
54. Stetter, M.D.; Mikota, S.K.; Gutter, A.F.; Monterroso, E.R.; Dalovisio, J.R.; Degraw, C.; and Farley, T. 1995.

Epizootic of *Mycobacterium bovis* in a zoologic park. Journal of the American Veterinary Medical Association 207(12).

55. Wolfe, N.D.; Escalante, A.A.; Karesh, W.B.; Kilbourn, A.; Spielman, A.; and Lal, A.A. 1998. Perspectives in wild primate populations in emerging infectious disease research: The missing link? Emerging Infectious Diseases 4(2):149–158.
56. World Health Organization. 1992. WHO Expert Committee on Rabies, 8th report. Technical Report Series 824. Geneva, World Health Organization.

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Laboratory Support in Wild Animal Medicine

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Laboratory analysis of wild animal samples provides vital information for the diagnosis, prevention, and control of diseases. It is also important to have reference values for the species in a collection.

SAMPLING AND SAMPLE CONSERVATION

Samples for Hematology

Large variations may occur on tests performed in different laboratories due to the different techniques used, time of sampling, animal management, age, physical effort, nutritional status, physiologic state and mental status, season of year, anesthetic used in the restraint, and environmental conditions; therefore, a regional standardization of the sampling methods and laboratory techniques becomes important. There are seasonal variations, particularly in ectothermic animals and those that hibernate, in the percentage of defense cells, packed cell volume, and various other parameters. The sampling method will be determined by the size of the animal and by the required amount of blood volume for a particular test.[15] Blood samples may be obtained from various sites, but blood components may vary according to the site of collection (vascular, capillary, or cardiac).

In mammals, the collection site must be chosen by considering the convenience of collecting the sample and the safety of the veterinarian and the staff that are helping in the sampling procedure. In small mammals, such as small primates, rodents, and carnivores, the femoral or saphenous veins are commonly used.[13,24,29,31,37,39]

In birds, the brachial (ulnar), tarsal, or jugular veins are commonly used. The specimen must be collected gently with a needle of small caliber, and, followed by application of soft compression to the site until the risk of hemorrhage ceases.[1,8,13,17,19,20,38]

In reptiles, samples may be obtained from various sites according to the different groups. In large snakes, the palatine-pterigoyd veins may be used, making sure the oral cavity is healthy. Snakes have a ventrocaudal tail vein that is used for sampling. The insertion site is immediately caudal to the hemipenes in males or the cloaca in females.[20,23,32,33] In crocodilians and lizards, blood samples may be obtained from the tail vein and by cardiocentesis. To collect a sample from the tail vein, the animal is positioned in dorsal recumbency and the needle is inserted through the skin and tissue to the caudal vertebra. In cardiocentesis, the animal is also positioned in dorsal recumbency, and the heart is located on the midline, about 11 scales from the shoulders. Blood samples may also be obtained in these animals from the supravertebral vein, located caudally to the occipital bone and immediately dorsal to the spinal cord. Care must be taken not to pass the needle through the vein, because if the needle penetrates too deeply, the spinal column may be punctured.[25–27] If this happens, the sample may be diluted with cerebrospinal fluid. Arterial blood may be obtained from the tail artery, which passes dorsal to the vein. These samples are collected in order to evaluate the blood gases of these animals.

In chelonians samples may be obtained from juvenile individuals by cardiac puncture (introducing the needle between the plastron folds). From adults, besides the cardiac puncture (that requires the drilling of the plastron), jugular venipuncture may also be used.

In fish, the ventral caudal vein may be used;[6,8] however, in larger fish, the penetration of the needle must be from the lateral part of the fish. The erythrocytes of fish are quite sensitive to hemolysis, so a hemogram must be performed quickly.

Ethylenediaminetetraacetic acid (EDTA) or heparin anticoagulants are used for hematological examinations of reptiles. Watch for incompatibilities in animal species with regard to some anticoagulants. Some animals, even from the same family, may have different reactions to the same anticoagulant. Northeastern Curassow (*Mitu mitu mitu*) blood will hemolyze if heparin is used. Sodium citrate causes loss of water from the erythrocytes with consequent dilution of the plasma but does not appear to be as damaging to the cell membrane of reptilian erythrocytes as is EDTA.[1,15,19,23,37]

The general sampling techniques and laboratory procedures for wild animals are the same as for domestic animals. Refer to standard domestic animal clinical pathology texts.

HEMATOLOGY

It may be necessary to perform a manual count in a Neubauer chamber (improved) instead of using electronic counters, when counting red blood cells, white blood cells, and platelets, in order to obtain better standardization of the size of these cells. It may be possible to calibrate an electronic counter to deal with one specific animal, but if numerous species are handled, it is necessary to count the cells manually.

In birds, reptiles, amphibians, and fish, the red blood cells are nucleated so manual counting is necessary. The red blood cell and white blood cell counts are obtained in one unique dilution with physiological saline or Gowers diluent plus Giemsa stain in a proportion of one or two drops of the stain to 10 mL of diluent, as described by Almosny et al.[1,2,3,4] This solution is not stable, so it must be prepared at the time of the examination. For the dilution of blood, a Thomas pipette may be used for the red blood cell count. Counting is performed at the magnification of ×400, in order to differentiate the cells.

The stain delineates the membranes and stains the nuclei, which facilitates the differentiation between erythrocytes, leukocytes, and thrombocytes. After dilution, the material must be left resting for a moment in order to induce thrombocyte aggregation, which facilitates their differentiation from the lymphocytes.

The disadvantage of the direct method is the difficulty of differentiating small lymphocytes from thrombocytes. The disadvantage of indirect methods is linked to the fact that certain species, or even individuals, have a low percentage of heterophils and eosinophils, increasing the chance of error.[19]

The thrombocyte count is invariably done in a hemocytometer, paying attention so that they are not confused with small lymphocytes if all cellular types are present at the same time in the hemocytometer.[1,2]

Other hematologic tests are conducted as for domestic animals.

Cellular Morphology

In wild mammals, the morphology of red blood cells in the peripheral blood is similar to that of domestic animals, being roundish, biconcave, and devoid of a nucleus. In camelids, for example, llamas (*Lama glama*) and alpacas (*Lama pacos*), the erythrocytes tend to be elliptical and rather dense in the central area. In some animals, such as the cervids, sickle cell erythrocytes may be observed frequently, which in humans would mean sickle cell anemia. Howell-Jolly bodies are occasionally observed, and reticulocytes may be demonstrated by using new methylene blue and other specific stains. Fusiform, pyriform, or spherical erythrocytes are among morphological anomalies that may be seen, as macrocytosis or microcytosis.[16,19,31,35,36,37] South American primates, especially marmosets (*Callithrix* sp.)and the golden lion tamarin (*Leontopithecus rosalia*), usually exhibit a considerable number of Heinz bodies.

Intra- and extraerythrocytic hemoprotozoa and the presence of microfilariae must be evaluated.[19] In reptiles, amphibians, fish, and birds, all blood cells retain the nucleus during their lifetime. The erythrocyte in these species is an oval cell that contains a cytoplasm rich in hemoglobin in its interior, presenting a homogeneous coloration that ranges from yellowish to reddish, as seen with Giemsa or similar stain. It also has a nucleus that frequently presents an irregular shape and can be partially lobulated. Enucleated erythrocytes called erythroplastids may occur occasionally, but unless they are present in large numbers, their importance is considered minimal. Nuclei free from cytoplasm or cells with little cytoplasm, called hematogonias, may be found in the plasma between adjacent cells, but are of doubtful importance. The red cells of turtles and alligators have an uncommonly lengthy half-life. A relationship seems to exist between the half-life of the erythrocytes and the metabolism of the animal. Accordingly, in animals with a high metabolism, the erythrocytes have a shorter half-life.

The lifetime of reptilian erythrocytes varies according to the temperature in which the individual is kept and may be as long as 3 years. Reticulocytes may be demonstrated as Howell-Jolly bodies, morphological anomalies, macrocytosis, or microcytosis. Inclusion bodies are found in some viral infections in reptiles. Rubricytes may be encountered when evaluating the blood of young animals, animals in ecdysis, or responding to anemia.[23,25,26,27]

Binucleated erythrocytes, erythrocytes with abnormal nuclear shape, or even mitotic figures, may be found in reptiles. These findings are more frequent in animals with serious inflammatory processes or animals recovering from hibernation.[23]

The number of the red cells is inversely proportional to their size. Animals with small erythrocytes have them in larger numbers, and the inverse may also be observed. Thus, the percentage of erythrocytes in relation to the total blood volume (packed cell volume) will not have large variation between the species.

Leukocytes

Leukocytic evaluation in wild mammals is similar to that of domestic mammals. However, in lagomorphs and several species of South American rodents, there are heterophils: cells that have color characteristics similar to eosinophils but with lower numbers of granules in the place of neutrophils.[28]

There may be seasonal variations in the differential count, a tendency rather pronounced in animals that hibernate and in ectothermic ones. Even in tropical regions these variations may be noted.

Birds and reptiles also have heterophils. In these animals, however, the feature of the granules is quite different from the granules of the eosinophils. The number of neutrophils is higher than that of lymphocytes in most carnivores, and the reverse is true in herbivores. The number of heterophils is greater in carnivorous and piscivorous birds, and the lymphocyte count is higher in omnivorous birds. Pronounced differences between the mononuclear cells (lymphocytes and monocytes) of birds and domestic mammals have not been observed.[1,19,21,23] The basophils of birds and reptiles have many granules, resembling those of ruminants.[1,23]

Amphibians and fish have neutrophils;, however, the morphological variation of this cell in these animals is so wide that an intensive study to differentiate them is needed.[8]

The basophil is a round cell with a roundish nucleus of dark coloration (under Romanowsky-like stain). The morphological variation between mammals, birds, reptiles, amphibians, and fish is primarily limited to the number of cytoplasmic granules.[1,19,23]

The neutrophils of mammals are polymorphonuclear. The granulocytes have pinkish granules with an affinity to neutral stains, except in lagomorphs and several species of South American rodents, which have heterophils containing intracytoplasmic eosinophilic granules, but the neutrophils do not.[1,18,28]

Fish and amphibians have type I and type II neutrophils. The morphology of these cells differs from conventional neutrophils in that they have a round nucleus and large granules similar to the azurophils described in reptiles.[6,8]

Mammalian eosinophils contain eosinophilic granules and, in lagomorphs and several species of South American rodents, must be differentiated from heterophils. With a cytochemical stain using the Sudan Black B method, it may be observed that the heterophil granules stain black and the eosinophil granules stain metallic olive green.[23,28,32,33]

In birds and reptiles, the eosinophil granules are large and generally round; however, this pattern is not maintained in some species of birds, because in some owls elongated eosinophilic granules are observed. With the Sudan Black B method, bird eosinophil granules stain metallic olive green, whereas those of heterophils are pinkish.[28]

In general, heterophils are large cells with eccentric nuclei that have an oval to rounded shape, with intracytoplasmic eosinophilic elongated granules. The nuclei may be lobulated in some species. Biochemically, the heterophils are deficient in proteases, which makes the birds' exudate caseous and not fluid as it is in mammals.[23,28,32]

Birds and reptiles also have heterophils. The heterophils of birds are peroxidases negative and do not acquire special coloration with Sudan black B. The heterophils are cytochemically characterized by acid phosphatase in the elongated granule membrane (and in immature ones by arylsulfatase B) in the periphery of the granules; by unspecific esterase in the plasmatic membrane and in small cytoplasmic granules; by peroxidases neutral in the granules; by presence of basic proteins in the granules; by presence of glucoproteins in the cell membranes; and by presence of glycogen distributed in the cytoplasm.[12,28]

The azurophil is described in reptiles as a mononuclear cell with a more azurophilic cytoplasm. This cell is also referred to as a neutrophil.[15] In snakes, the number of azurophils is greater than the number of monocytes.[23]

Monocytes are generally the largest cells in the blood, with a bluish-gray cytoplasm (under Romanowsky-like stain) with "fine" granules and frequent vacuoles. It has, generally, a single nucleus that may vary in shape from ovoid to bean shaped.[23]

The number of monocytes is generally low and may vary from 0 to 10% of the counted leukocytes. Monocytes seem to vary little among mammals and other orders.

With similar morphology in most mammals, lymphocytes may appear small, medium, or large in size. These cells are round with basophilic and azurophilic cytoplasmic granules. The nucleus is also rounded and may have one or, occasionally, two nucleoli in young cells. The number of lymphocytes may vary according to the season of the year, mainly in ectothermic animals and in those that hibernate.

The blood of mammals has platelets that are fragments of megacaryocytes and function in hemostasis. Thrombocytes are found in birds, reptiles, and amphibians. They are fusiform cells with a basophilic central nucleus, transparent to pale blue in color, and occasionally with azurophilic cytoplasmic granules. Contrary to mammalian platelets that originate from megacaryocytes, thrombocytes derive from erythroid cells. These erythroid cells are considered to be erythrocytes that did not reach maturity. In a few cases, they may synthesize hemoglobin.[23] The thrombocyte participates in blood coagulation by phagocytizing. It may even become an erythrocyte, due to its pluripotentiality, when necessary.[15] The function of phagocytizing does not interfere with the ability to aggregate in blood coagulation.[23]

Interpretation of the Hemogram

The same parameters used in domestic animals are used in interpreting wild animal cells and biochemical values. See the individual species values dispersed throughout the book.

Evaluating Blood Smears for Hemoparasites

Blood smears should be prepared from peripheral blood. If the sample is from a needle prick, it is important to use the first drop because it is considered to correspond to peripheral blood, whereas succeeding drops are from the circulating blood.

Anaplasma sp. are generally located in the periphery of the erythrocytes of domestic and wild ruminants and are related to hemolytic anemias.[23]

Babesia sp., an intraerythrocytic parasite described in domestic and wild animals, is related to severe hemolytic anemias. *Babesia* may be found in lizards, snakes, and chelonians: *Sauroplasma* is found only in lizards, and *Serpentoplasma* is found only in snakes.[7]

Aegyptianella, Sauroplasma, and *Serpentoplasma* are identified as very small intraerythrocytic inclusions with roundish to pyriform morphology.

In reptiles, *Hepatozoon* sp. parasitize erythrocytes, but in domestic and wild carnivores this genus parasitizes neutrophils. Transmission of *Hepatozoon* sp. between snake species and between snakes and lizards is through the ingestion of an infected intermediate host.

Haemobartonella sp. are commonly observed in the erythrocytes of carnivores but may also be seen in the plasma. In chronic cases, the presence of the parasites in the circulating blood is periodic. Daily blood samples taken over at least a 4-day period, and at varied times of the day (but mainly at nightfall), are recommended. The examination should be done by stained blood smears, preferably stained by Giemsa or another similar stain used for hematology.[23]

Ehrlichia sp. is a rickettsia that is also found in stained blood smears. It parasitizes the leukocytes and platelets of domestic and wild mammals. It produces a normocytic normochromic anemia associated with cyclic thrombocytopenia in an acute phase and pancytopenia in a chronic phase. There are cases in which ehrlichiosis occurs with babesiosis causing serious disease.

Plasmodium sp. is observed in blood smears stained by Giemsa or similar stains. *Plasmodium,* such as *Haemoproteus,* may be intraerythrocytic, but *Plasmodium* may even be intraleukocytic or be found inside thrombocytes, as well. Another way of differentiation between both genera is that only *Plasmodium* presents schizogony in the peripheral blood. *Plasmodium* and *Haemoproteus* are found in birds, mammals, chelonians, and in many lizards.[7]

Leishmania sp. parasitizes mammals and lizards, but is rarely found in the peripheral blood of other reptiles, being identified mostly by culture. In the peripheral blood, *Leishmania* may be found in the cytoplasm of thrombocytes, lymphocytes, and monocytes.[7,23]

Filariasis is diagnosed by finding microfilariae in the blood. Infestations by filariae may cause clinical disease or death of the animal; however, in most cases, neither clinical signs, nor hematological, nor biochemical alterations will be noticed.

Trypanosoma sp. are distributed throughout the world in all orders of reptiles, fish, birds, and mammals. Their presentation in the circulating blood is invariably of the extracellular shape.[7,11]

The gametocytes of *Saurocytozoon* sp. are found inside the leukocytes of lizards and have a rounded shape, unlike the hemogregarines.[7,23]

Schellackia sp. is identified by intraerythrocytic or intraleukocytic (lymphocytes) oval to round gametocytes in lizards.[23]

The interpretation of biochemical values found in blood, urine, exudates, transudates, or cerebrospinal fluid of wild animals is done in a manner similar to that done for domestic animals. Likewise, bacteriologic, cytologic, parasitologic, mycologic, and other laboratory tests are similar to those used for domestic animals.

REFERENCES

1. Almosny, N.R.P.; Silva, K.P.; Melo, D.L.S.; Vasconcelos, T.C.; and Monteiro, A.O. 1998. Hematologia de aves: Valores normais em hemograma de mutum de alagoas (*Mitu mitu mitu*). [Avian hematology: Normal hemogram values for northeastern currassow (*Mitu mitu mitu*)]. Revista Brasileira de Ciência Veterinária (UFF) 5(3).

2. Almosny, N.R.P.; Fedullo, L.P.L.; Vasconcelos, C.H.C.; Rodrigues, R.R.; and Romão, M.A.P. 1992. Avaliação do hemograma de 9 (nove) atobás (*Sula leucogaster*) do litoral de Rio de Janeiro [Hemogram evaluation in 9 (nine) atobás (*Sula leucogaster*) from Rio de Janeiro coast]. In Proceedings of the 22° Congresso Brasileiro de Medicina Veterinária. Curitiba, Paraná, Anclivepa-PR.
3. Almosny, N.R.P.; Nascimento, M.D.; Silva, K.P.; and Melo, D.I.S. 1993. Hemograma de aves: Métodos [Avian hemogram: methods]. Anais do 6° Congresso Internacional de Medicina Veterinária em Língua Portuguesa. Salvador, Bahia, Organização de Médicos Veterinários de Lingua Portuguesa.
4. Almosny, N.R.P.; Silva, K.P.; Melo, D.I.S.; and Nascimento, M.D. 1993. Estudo de valores normais do hemograma dos cracídeos: Mutum [Study of cracidae normal hemogram values: Curassow]. In Anais do 6° Congresso Internacional de Medicina Veterinária em Língua Portuguesa. Salvador, Bahia, Organização de Médicos Veterinários de Lingua Portuguesa.
5. Almosny, N.R.P.; Silva, K.P.; Melo, D.I.S.; and Nascimento, M.D. 1993. Jacuguaçu (*Penelope obscura*): valores médios observados em hemograma [Jacuguaçu (*Penelope obscura*): Mean hemogram values]. In Anais do 6° Congresso Internacional de Medicina Veterinária em Língua Portuguesa. Salvador, Bahia, Organização de Médicos Veterinários de Lingua Portuguesa.
6. Campbel, T.W.; and Murru, F. 1991. An introduction to fish hematology. In D.E. Johnston, ed., The Compendium Collection, Vol. 2. Exotic Animal Medicine in Practice. Philadelphia, W.B. Saunders, pp. 126–132.
7. Campbell, T.W. 1991. Hematology of exotic animals. In The Compendium Collection, Vol. 6. Trenton, New Jersey, Veterinary Learning Systems, pp. 950–956.
8. Campbell, T.W. 1995. Hematology of birds, reptiles and fish. In EV. Hillyer, ed., Exotic Animals—A Veterinary Handbook. Trenton, New Jersey, Veterinary Learning Systems, pp. 196–199.
9. Chiodini, R.J.; and Sundberg, J.P. 1982. Blood chemical values of the common boa constrictor (*Constrictor constrictor*). American Journal of Veterinary Research 43(9):1701–1702.
10. De Biasi, P.; Cardoso, R.P., Jr.; and Santos, S.M. de A. 1989. Presença de *Hepatozoon plimmeri* (Sambon, 1909)—Coccidia, Haemogregarinidae—em exemplar de *Bothrops jararaca* (Wied, 1824)—Serpentes Viperidae, Crotalinae—mantido em cativeiro [Presence of *Hepatozoon plimmeri* (Sambon, 1909)—Coccidia, Haemogregarinidae—in a *Bothrops jararaca* (Wied, 1824)—Serpentes Viperidae, Crotalinae—kept in captivity]. Memorias do Instituto Butanan 51(3):117–121
11. De Biasi, P.; and Pêssoa, S.B. 1972. *Trypanosoma cacavelli* sp. n. parasita da cascavel: *Crotalus durissus terrificus* (Laurenti) [*Trypanosoma cacavelli* sp. n. rattlesnake (*Crotalus durissus terrificus* (Laurenti)) parasite]. Atas da Sociedade de Biologia do Rio de Janeiro 15(2):67.
12. Egami, M.I.; and Sasso, W.S. 1988. Cytochemical observations of blood cells of *Bothrops jararaca* (Reptilia, Squamata). Revista Brasileira de Biologia 48(2):155–159.
13. Fowler, M.E. Ed. 1986. Zoo and Wild Animal Medicine, 2nd Ed. Philadelphia, W.B. Saunders.
14. Fowler, M.E.; and Miller, R.E. Eds. 1999. Zoo and Wild Animal Medicine, Vol. 4. Current Therapy. Philadelphia, W.B. Saunders, p. 747.
15. Frye, F.L. 1986. Hematology of captive reptiles. In M.E. Fowler, ed., Zoo and Wild Animal Medicine, Vol. 2. Philadelphia, W.B. Saunders, pp. 181–184.
16. Garcia-Navarro, C.E.K.; and Pachaly, J.R. 1994. Manual de Hematologia Veterinária. São Paulo, Livraria Varela, p. 169.
17. Gonçalves, I.P.D. 1987(1988. Valores hematológicos de algumas espécie de aves mantidas em cativeiro [Hematologic values for some avian species kept in captivity]. Arquivos da Faculdade de Veterinária da Universidade Federal do Rio Grande do Sul (UFRGS) 15(16:89–94.
18. Hawkey, C.M.; and Samour, H.J. 1988. The value of clinical hematology in exotic birds. In E.R. Jacobson and G.V. Kollias, eds., Exotic Animals. New York, Churchill Livingstone, pp. 109–141.
19. Jain, N.C. 1993. Essentials of Veterinary Hematology. Philadelphia, Lea & Febiger, p. 417.
20. Jenkins, J.L. 1995. Basic avian hematology: Techniques of collection, preparation, and identification. In E.V. Hillyer, eds., Exotic Animals—A Veterinary Handbook. Trenton, New Jersey, Veterinary Learning Systems, pp. 126–135.
21. Kaneko, J. 1997. Clinical Biochemistry of Domestic Animals, 5th Ed. San Diego, Academic Press, p. 932.
22. Mader, D.R. 1996. Reptile Medicine and Surgery. Philadelphia, W.B. Saunders.
23. Monteiro, A.O. 1998. Determinação de Valores Hematológicos e Bioquímicos Normais para Serpentes do Gênero Bothrops (Wagler, 1824). Masters thesis, Universidade Federal Fluminense.
24. Nascimento, M.D.; Pissinatti, A; and Xavier, M.S. 1997. Valores de proteinograma de *Cebus apella xanthosternos* (Wied, 1820) cebidae-primates. In Resumos 7° Congresso Brasileiro de Primatologia e 5° Reunião Latino Americanade Primatologia. João Pesson, Paraiba.
25. Neves, J.M., Jr.; Almosny, N.R.P.; Bressan, A.C.S.; and Bruno, S.F. 1996. Valores hematológicos e bioquímicos em jacarés de papo amarelo (*Caiman latirostris*) de vida livre no Município de Quissamã, R.J. [Hematologic and biochemical values in free-ranging jacarés de papo amarelo (*Caiman latirostris*) in Município de Quissamã, R.J.]. In Proceedings of the 15° PANVET. Campo Grande, Mato Grosso do Sul, Congresso Pan-Americano de Veterinária (PANVET).
26. Neves, J.M., Jr.; Almosny, N.R.P.; Bruno, S.F.; Romão, M.P.; and Bressan, A.C.S. 1996. Avaliação hematólogica do jacaré de papo amarelo (*Caiman latirostris*) [Hematologic evaluation in jacaré de papo amarelo (*Caiman latirostris*)]. In Proceedings of the 48° Reunião Anual da Sociedade Brasileira Para o Progresso da Ciência. São Paulo, Sociedade Brasileira para o Progresso da Ciência.

27. Neves, J.M., Jr.; Almosny, N.R.P.; Cristally, R.; and Fedullo, L.P.L. 1996. Valores bioquímicos em jacarés do pantanal (*Caiman crocodillus*) na Fundação RIO-ZOO R.J. [Biochemical values in jacarés do pantanal (*Caiman crocodillus*) from the RIO-ZOO Foundation, R.J.]. In Proceedings of the 15° PANVET. Campo Grande, Mato Grosso do Sul, Congresso Pan Americano de Veterinária (PANVET).
28. Pereira, N.R.; Fan, L.C.; Nascimeno, M.D.; and Rossi, M.I. 1985. Diferenciação dos leucócitos de galinhas e coelhos pelo método citoquímico de Sudan Black B [Chicken and rabbits leukocytes differentiation by the Sudan Black B cytochemistry method]. In Anais do Congresso Estadual de Medicina Veterinária. Santa Maria, Rio Grande do Sul, CRMV-RS, p. 68.
29. Pissinatti, A; Nascimento, M.D.; and Xavier, M.S. 1997. Alguns valores bioquímicos Séricos de *Cebus apella xanthosternos* (Wied, 1820) cebidae-primates [Some biochemical serum values of de *Cebus apella xanthosternos* (Wied, 1820) cebidae-primates]. In Resumos 7° Congresso Brasileiro de Primatologia e 5° Reunião Latino Americanade Primatologia. João Pesson, Paraiba.
30. Pratt, P.W. 1997. Laboratory Procedures for Veterinary Technicians, 3rd Ed. St. Louis, Missouri, Mosby, p. 660.
31. Rodrigues, R.R.; Vasconcellos, C.H.C.; Almosny, N.R.P.; and Nascimento, M.D. 1996. Determinação do hemograma, bioquímica sérica e pesquisa de hemoparasitos em coatis (*Nasua nasua*) em condições de cativeiro no Estado do Rio de Janeiro [Hematologic and biochemical determination and hemoparasites in coatimundi (*Nasua nasua*) in captivity in Rio de Janeiro]. Revista Brasileira de Ciencia Veterinária 3(3):89–92.
32. Rodrigues, L.M.; Monteiro, A.O.; Almosny, N.R.P.; Melgarejo, A.; and Aguiar, A.S. 1998. Hematological evaluation of snakes of the genera *Bothrops* (Wagler, 1824). In Anais 23rd Congress of the World Small Animal Veterinary Association. Buenos Aires, World Small Animal Veterinary Association (WSAVA).
33. Rodrigues, L.M.; Monteiro, A.O.; Melgarejo, A.R.; Aguiar, A.S.; and Almosny, N.R.P. 1997. Avaliação dos parâmetros hematológicos normais e hemoparasitoses em serpentes do gênero *Bothrops* [Evaluation of normal hematologic parameters and heparasitosis in snakes of the *Bothrops* genus]. Anais do 7° Seminário de Iniciação Científica e 1° Vasconcellos Torres de Ciência e Tecnologia. Niteroi, Rio de Janeiro, EDUFF.
34. Santos, L.C.; Malvëstio, J.S.; Moraes, W.; and Suemitsu, E.S. 1992. Valores de hemograma e dosagens bioquímicas de *Tayassu pecari* (queixada), clinicamente sadios, mantidos em cativeiro no criadouro de animais silvestres da Itaipu Binacional [Hemogram values and biochemical dosages of clinically healthy *Tayassu pecari* (queixada), kept in captivity at Itaipu Binacional]. In Anais 47° Conferência Anual da SPMV, 8° Semana de Medicina Veterinária, e 1° Encontro Nacional da Abravas. São Paulo, Sociedade Paulista de Medicina Veterinária (SPMV).
35. Santos, L.C.; and Moraes, W. 1992. Valores de hemograma e dosagens bioquímicas de *Mazama rufina* (veado bororó), clinicamente sadios, mantidos em cativeiro no criadouro de animais silvestres da Itaipu Binacional [Hemogram values and biochemical dosages of clinically healthy *Mazama rufina* (veado bororo), kept in captivity at Itaipu Binacional]. In Anais 22° Congresso Brasileiro de Medicina Veterinária. Paraná, Anclivepa-PR.
36. Santos, L.C. (In press). Laboratório Ambiental. Cascavel, Edunioeste.
37. Wallach, J.D.; and Boever, W.J. 1983. Diseases of Exotic Animals. Philadelphia, W.B. Saunders, p. 1329.

Appendix
Drug Dosages Used in Avian Medicine

Karin Werther

TABLE A.1. Principle drugs and doses used in avian medicine

Antibiotics			
Drug	**Dose**	**Route**	**Comments**
Amikacin	10–15 mg/kg BID, TID 15–20 mg/kg SID or BID	IM, IV, SC	Broad spectrum; nephrotoxicity increased with dehydrated patients, synergism effect when used with third-generation penicillin
Amoxicillin	150–175 mg/kg TID, SID, BID, QID	PO, IM	Not a very effective drug against common avian pathogens
Ampicillin	100–200 mg/kg TID to QID, BID	PO, IM	Poor gastrointestinal absorption, high gastrointestinal specificity
Cefotaxime	75–100 mg/kg	IV, IM	Penetrates blood-brain barrier. Best results if QID
Cephalosporin	50–100 mg/kg QID	IM, IV	Not absorbed from the gastrointestinal tract, painful injection
Chloramphenicol succinate	30–50 mg/kg every 6 to 8 hours 50–80 mg/kg BID, TID 80–100 mg/kg BID, TID	PO, IM, IV	If administered by IV, the product will be excreted rapidly by the liver. Useful for several bacterial septicemia
Chloramphenicol palmitate			Erratically absorbed from the gastrointestinal tract
Clindamycin	Pigeons 100 mg/kg SID	PO	Monitor renal and hepatic functions during long-term use and for secondary yeast infection. Primarily indicated in cases of osteomyelitis
Enrofloxacin	7.5–15 mg/kg BID, SID: most 5 mg/kg BID: cockatoos, African grey parrots	PO, IM PO, IM	Little evidence of joint problem developing in young species
Lincomycin	75 mg/kg BID: Amazon parrots 100 mg/kg SID: raptors	PO PO	Use carefully, overdose is lethal Useful in dermatitis. May be effective in treatment of chronic respiratory infections caused by *Mycoplasma*
Tylosin	250–400 mg/L drinking water or 400 mg/kg mixed with soft food 10–40 mg/kg TID, QID: most 15 mg/kg TID, QID: cranes	PO IM IM	Useful in *Mycoplasma* upper respiratory infections

continued

TABLE A.1. cont'd

Drug	Dose	Route	Comments
	Antibiotics		
	100 mg/10 mL diluted in sterile saline solution. 1 g diluted in 50 mL DMSO or 100 mg diluted in 10 mL of saline solution.[5]	Nebulization	
Trimethoprim and sulfamethoxazole	16–24 mg/kg BID, TID: Psittaciformes	PO	Regurgitation is a potential side effect. Patients suffering from liver diseases or bone marrow suppression should not be treated with this drug
	8 mg/kg Psittaciformes	IM	
	25 mg/kg SID, BID: toucans, mynahs	PO	For coccidia
	50 mg/kg SID: pigeons	PO	Oral suspension is one of the drugs of choice for treatment of gastrointestinal and respiratory infections
	25 mg/kg BID: pigeons	PO	
	Antifungals		
Amphotericin B	1.5 mg/kg BID, TID	IV	Possible nephrotoxicity, may cause blood dyscrasia
	1 mg/kg BID, TID	Intratracheal	
	1 mg/mL saline solution 15 min BID	Nebulization	
Itraconazole	5–10 mg/kg BID	PO	More effective against aspergillosis and less toxic than other antifungals
	5 mg/kg SID: African gray parrots	PO	
Ketoconazole	20–30 mg/kg BID:	PO (gavage)	Used for candidiasis. May cause hepatocellular necrosis
Flucytosine	20–50 mg/kg BID for 21 days: most birds	PO	Indicated for long-term treatment of aspergillosis. Is toxic to the bone marrow
	20–30 mg/kg QID: Raptors	PO (gavage)	
Fluconazole	2–5 mg/kg SID for 7 days	PO	May not be compatible with another antifungal. In vitro, activity for aspergillosis, candidiasis and criptococcosis. Passes blood-brain barrier
	Antiparasitic Drugs		
Amprolium	200–400 mg/3.8 L drinking water for 5 days	PO	For coccidia
Fenbendazole	5–15 mg/kg for 5 days: anseriformes	PO	For ascarids, repeat after 10 days, for *Capillaria* treatment for 5 days. Don't use during molt
	20–50 mg/kg: most for 3–5 days	PO	
Ivermectin	200 μg/kg: most	IM, PO, topical	Repeat 10–14 days
Metronidazole	10–30 mg/kg BID for 10 days	PO	For *Giardia, Hexamita,* flagellates. Don't use in finches
	50–60 mg/kg BID for 10 days	PO	
	10 mg/kg SID for 2 days	IM	
Praziquantel	10–20 mg/kg	PO	For flukes (3 days SID, IM followed by 11 days PO). Trematodes, cestodes, repeat 10–14 days. Don't use for finches
	9 mg/kg: most	IM	
Sulfamethazine	30 mg/30 mL drinking water, 3 days on, 2 days off, 3 days on	PO	For coccidia

continued

TABLE A.1. cont'd

Drug	Dose	Route	Comments
Vitamins			
Vitamin A	10–25 IU/300 g body weight[6]	IM (weekly)	Epithelial disorders
Vitamin D_3	1 IU/300 g body weight	IM (weekly)	Calcium deficiencies
Vitamin E + selenium	0.05–0.1 mg/kg	IM (every 14 days)	Neuromuscular diseases
Vitamin B complex	1–3 mg/kg thiamine 1–2 g/kg of feed SID: raptors, crows, penguins	IM PO	Neuromuscular disease; debilitating illness of the liver, kidney, and gastrointestinal tract; and anemia
Vitamin C	20–40 mg/kg daily to weekly	IM	
Vitamin K	0.2–2.5 mg/kg SID	IM	For extreme blood loss, anemia, Warfarin toxicity, hemorrhagic disorders.
Other Drugs			
Atropine	0.01–0.02 mg/kg	IM, SC	Not recommended as a preanesthetic in avian species; thickens respiratory secretion that may block endotracheal tube. Used in organophosphate poisoning. Does not dilate pupils in avian species
Ca-EDTA	20–40 mg/kg BID to TID	IM	Chelates lead and zinc, may cause tubular necrosis, and should be discontinued if polyuria/polydipsia occurs
Flunixin meglumine	1–10 mg/kg	IM	Anti-inflammatory, analgesic, antipyretic, may cause diarrhea or vomiting

Source: Modified from references 1–3.
BID, twice a day; DMSO, dimethyl sulfoxide; EDTA, ethylenediaminetetraacetate; IM, intramuscularly; IV, intravenously; PO, by mouth; QID, four times a day; SC, subcutaneously; SID, once a day; TID, three times a day.

TABLE A.2. Medication interactions

Drug No. 1	Drug No. 2	Effects of Drugs No. 1 + 2
Chloramphenicol	Anesthesia (barbiturates)	Prolongs anesthesia
Aminoglycosides	Narcotics	Intensifies muscular relaxation
Muscular relaxants	Neomycin, kanamycin or streptomycin	Synergism with the motor end plate
Tetracycline	Bi- or trivalent cations (Ca^{++})	Forms no absorbable complexes in the intestine
Sulfonamide	Alkaline substances ($NaCO_2$)	Increases excretion of sulfonamides
Sulfonamide	May separate other drugs or poisons that are bound to plasma protein	Intensifies the effects of the drugs or poisons
Sulfadimidine	Pantothenic acid	Increases excretion of sulfonamides
Cardiac glycosides	Diuretic	Increases cardiac sensitivity to the glycosides
Furazolidone	Amprolium or nitrobenzamides	Neurotoxic
Ionophore	Tiamulin	Toxic for chicken and turkey
Lasalocid	Chloramphenicol	Causes paresis and ataxia in chickens
Quinacrine	Primaquine	Toxic synergism
	Bactericides and Bacteriostatics	
Primary Bactericides		
Polimixin B	Tetracycline	Reduced antibacterial efficiency
Colistin	Chloramphenicol or novobiocin	Reduced antibacterial efficiency
Neomycin	Oleandomicin or viomicin	Reduced antibacterial efficiency
Aminoglycosides	Spiramycin or sulfonamide or clindamycin	Reduced antibacterial efficiency
Secondary Bactericides		
Bacitracin	Tetracycline	May reduce efficiency
Penicillin	Chloramphenicol or novobiocin	May reduce efficiency
Cephalosporin	Oleandomycin	May reduce efficiency
Aminoglycosides (streptomycin and kanamycin)	Spiramycin or sulfonamide or clindamycin	May reduce efficiency
Gentamicin	Carbenicillin or penicillin or cefalotoxin or tetracycline	Reduced antibacterial effect
Amphotericin B	Flucytosin	Potentiates amphotericin B. Use only 25% of the usual doses

Source: Modified from reference 4, p.155.

TABLE A.3. CONTRAINDICATION, COLLATERAL EFFECTS

Bird Species	Drug	Effect
Most birds	Corticoid	Hyperglycemia, hyperuricemia, ulcers, lymphopenia, death
Most birds	American doxycycline	Necrosis at IM injection site
Most birds	Lincomycin intravenous	Death
All birds	Procaine in every composition	Paralysis and death
All birds	Xylocaine as injectable diluent	Paralysis and death
Debilitated or stressed birds	Anesthesia with halothane	Bradycardia and vomiting
Ostriches	Thiabendazole	Don't support
Cormorants	Mebendazole	Don't use in cormorants
Penguin	Drugs for malaria treatment	Death
	Fenilmercuridinaftilmetadi sulfonate directly in the air sacs for aspergillosis	Death
	Mebendazole	Death
	Niclosamide (Yomesan) PO	Do not use
	Nihydrazone	Do not use
	Dihydrostreptomycin IM	Do not use
	Benzimidazole (anthelmintics), 5-Nitroimidazole (antiprotozoan), nitrofuran, reserpine, Nicarbazin, Pyremethamine	Interferes with fertility and egg hatching
	Prednisolone after 1–2 injections of 5 mg/kg	Intestinal ulcer
	Mebendazole and fenbendazole	Do not use
Pigeons during the molt	Fenbendazole	Do not use
Psittaciformes	Mebendazole	Do not use because it may cause death by intestinal occlusion
	Streptomycin IM	Do not use
	Haloxon	Do not use
Loriinae (lories)	Tetramisole	Do not use
	Gentamicin IM, SC, IV	Sensible
	Nitrofurazone in drinking water or as substitute for nectar	Seizures and death
Cacatuinae (cockatoos)	Levamisole SC or IM	Depressions, paralysis
	Ticarcillin associate with tobramycin IM	Hepatotoxic
Amazon parrots	Cephaloridine SC, periorbital	Causes blindness
Budgerigars	Perdixemulsion for mites treatment	Paralysis, death
	Dimetridazole during reproduction	Do not use
	Ivermectin IM	Do not use
	Levamisole SC and IM	Depressions ataxia, vomiting, paralysis, mydriasis
	Diazepam	Death after 17–24 hours
Cockatiel (4–11 days old)	Dimetridazole solution 0.1% in feed	After 24 hours debility, after 48 hours death, tremors
Cockatiel (24–31 days old)	Dimetridazole solution 0.5%	Ataxia after 24 hours
Raptors	Streptomycin, insecticide, organochlorine	High sensitivity
	Haloxon, repeat use of metomidate	High sensitivity
Waterfowl	Haloxon	High sensitivity
Waterfowl (young)	Holofuginon (coccidiostatic)	Progressive weight loss and death
Geese, ducks	Furazolidone	Do not use
Geese	300 mg/kg levamisole	Death
Canaries	Fenbendazole in drinking water	Ataxia, depression and mydriasis
Mynah	Levamisole SC	Do not use

Source: Modified from reference 6.
IM intramuscularly; IV, intravenously; PO, by mouth; SC, subcutaneously.

REFERENCES

1. Rupley, A.E. 1999. Manual de Clínica Aviária. São Paulo, Editora Roca, pp. 535–552.
2. Ritchie, B.W.; and Harrisson, G.J. 1994. Formulary. In B.W. Ritchie, G.J. Harrisson, and L.R. Harrison, eds., Avian Medicine: Principles and Applications. Lake Worth, Florida, Wingers Publishing, pp. 457–478.
3. Tully, T.N., Jr. 1997. Formulary. In R.B. Altmann, S.L. Clubb, G.M. Dorrestein, and K. Quesenberry, eds., Avian Medicine and Surgery. Philadelphia, W.B. Saunders, pp. 671–688.
4. Gylstorff, I.; and Grimm, F. 1987. Vogelkrankheiten. Stuttgart, Verlag Eugen Ulmer, p. 155.
5. Tully Jr., T.N.; and Harisson, G.J. 1994. Pneumonology. In B.W. Ritchie, G.J. Harisson, and L.R. Harisson, eds., Avian Medicine: Principles and Applications. Lake Worth, Florida, Wingers Publishing, p. 574.
6. Ritchie, B.W. 1995. Avian Viruses: Function and Control. Lake Worth, Florida, Wingers Publishing, p. 298.

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