Principles of
Zoological Animal Medicine
Mitchell Bush, D.V.M.
Richard J. Montali, D.V.M.

INTRODUCTION

The recent growth of zoos, wildlife parks, and preserves has led to a need for newer information concerning the diseases and therapy of the variety of exotic animals that are held in captivity. With the recent trends for conservation, zoos are becoming less the consumers and more the perpetuators of many animal species that are in danger of extinction in our diminishing natural environment.

Exotic animal medicine, therefore, is becoming a very essential specialty. Interest has surged on the part of veterinarians and veterinary students, but there is currently very little well-documented information regarding the veterinary aspects of the classes of animals (amphibians and reptiles, birds, and mammals) normally existing in a zoological collection.

The purpose of this article is to introduce the principles and philosophy of the practice of medicine as they occur in a zoological park (National Zoological Park) that has a full-time veterinary clinical and pathology staff devoted to the health care of approximately 3000 exotic animals. The material presented will be limited to conditions common to most zoo settings; however, unique to any zoo, there are always medical problems that are based on the types of species exhibited, the climate, and other factors. This will necessitate reporting some of our individual experiences.

One useful comparison is that of relating specific disease problems of exotic animals to those of domestic species. Essentially, we are dealing with familiar diseases in unfamiliar species. For example, many problems in birds are relatable to the highly studied conditions of domestic poultry. Likewise, all exotic cats are basically similar to domestic cats, while canids are relatable to the domestic dog. Carnivores such as badgers (*mustelids*), civets (*viverrids*), raccoons (*procyonids*), and others within these families have some medical problems of both dogs and cats. The majority of exotic hoofstock are either *bovidae* or *cervidae* which can be compared to the domestic cow and common deer, respectively. Zebra, tapir, rhinoceros, and to some extent, the elephant, can all be compared to the horse.

Although early detection of disease symptoms is an important principle in any medical program, it is often difficult to determine if a zoo animal is ill and should receive veterinary attention. Many animals in the wild appear to have the facility of masking serious illnesses, even until the very end. This may be a type of defense mechanism so that predators are not easily alerted. Likewise, many of these animals in captivity do not frequently exhibit overt clinical signs until near death. A good rapport is therefore necessary between the veterinary staff and the keeper staff. Keepers are more apt to detect subtle abnormalities because they have more contact with the individual species' eating and elimination patterns, and know well its general attitude and body carriage. Furthermore, in the presence of the veterinary staff, many animals will become excitable because of past experiences associated with treatment, and therefore important clinical signs may be obscured.

PREVENTIVE MEDICINE

Preventive medicine is an important aspect in formulating an overall health program for a zoo collection. When possible, all animals within the existent collection should undergo periodic physical examinations and routine hematologic studies and fecal tests for parasite eggs. Parasite surveillance is an extremely important procedure and when performed on a regular basis, can keep parasitic diseases that are often responsible for high mortalities in many zoological collections under control.

Also important in a program of preventive medicine is a strong quarantine program with a separate layover facility for all new acquisitions. All incoming animals should undergo rigid physical examinations with complete hematological studies, at least two fecal tests for parasites and, in selected cases, tuberculin testing and vaccinations.

General rules of thumb pertaining to quarantining the various classes of animals are as follows:

1. **Reptiles and Amphibians.** Reptiles should be examined for parasites, including ticks and mites, as well as for oral and cutaneous inflammatory lesions which are good sources of gram-negative septicemia. These septic conditions can be lethal, particularly under conditions of stress. When first introduced into a collection, reptiles often do not adapt well and do not feed. They must be observed closely and at times be force fed until they begin to eat on their own.

2. **Birds.** Birds should be quarantined for two to six weeks, depending upon the species and origin. Hematocrits, total protein, and total white cell counts are good indicators of their general condition. Low blood glucose ($<$ 200 mg %, Dextro-Stix, Ames) and low total protein values ($<$ 3g %) are indicative of poor nutrition. Birds with these lowered values should receive immediate attention by tube feeding, isolating, and warming them with a heat lamp. Elevated white blood cell counts[54] may indicate an infectious process, but in our experience it has not always been possible to make a specific diagnosis by culturing for causative microorganisms. Many of these birds, however, do respond well to broad-spectrum antibiotics. During physical examination, those found unduly thin with prominent keels should be suspected of having avian tuberculosis or aspergillosis—conditions not uncommon in captive exotic birds. Examinations for endo- and ectoparasites are also important.

3. **Mammals.** Quarantine procedures for mammals vary with the type of animal and the facilities available. Important quarantine procedures for hoofstock include tuberculin testing[57], hematologic studies, and fecal examinations for parasite eggs.

Primates should be quarantined for three to six weeks depending upon the type and source of the animal. Primates received from the wild demand longer quarantine periods and more careful checks for such diseases as tuberculosis, salmonellosis, and shigellosis, some of which are acquired from handlers in the countries of origin. Chimpanzees may harbor human viral hepatitis and some Old World monkeys can carry Herpes-B and Marburg viruses. Proper precautions should therefore be instituted to protect the health of zoo personnel having contact with these primates as well as vice versa.

Exotic carnivores, in addition to receiving physical examinations and blood and parasite tests, should be vaccinated for either canine or feline distemper or both. We recommend *killed* vaccine in all cases[10,30]. Exotic felids should be vaccinated for feline panleukopenia. All exotic canids should be vaccinated for canine distemper and members of the families Procyonidae such as raccoons, Mustelidae such as badgers, Viverridae such as civets, and Aleuropodidae such as lesser pandas should be vaccinated for both feline and canine distemper. Our vaccination procedures are as follows: each animal is vaccinated yearly with the respective killed virus vaccine; all newly arrived animals are vaccinated during quarantine and then given yearly boosters; the newborn animals are vaccinated at six to seven weeks, and then every two to three weeks until 14 weeks of age. There have been no outbreaks of either canine or feline distemper at the National Zoo with this program.

Animals such as bears and other carnivores, marsupials, marine mammals, etc. should receive physical examinations and blood and

parasite tests before they are released into the exhibit areas.

The quarantine of exotic animals for foreign and reportable diseases is done under the auspices of the United States Department of Agriculture (USDA) and therefore such diseases as psittacosis, exotic Newcastle disease, (also known as viscerotrophic-velogenic Newcastle disease, or VVND), hoof and mouth disease, rinderpest, etc., are checked prior to an animal's entrance into this country and therefore are not likely to be a threat. It also should be noted and emphasized that currently there are few or no regulations relating to the control of tuberculosis in exotic animals[43]. At present, the American Association of Zoo Veterinarians (AAZV) is formulating recommendations to have all primates and hoofstock receive tuberculin tests prior to shipment.

PHYSICAL EXAMINATION

When it becomes apparent that an animal is ill, a decision must be made as to whether it should be restrained and examined or held under further observation. The decision of what to do is like a double-edged sword in that the animal may die from further stress of restraining it; however, if left unattended, it may continue to deteriorate from what may have been a relatively easily treatable problem if examined. This type of judgement comes only with many years of practice and with the knowledge of various idiosyncrasies associated with certain species.

When there is a decision to catch and restrain an animal for further evaluation, it should be done with the intent of obtaining maximum information with a minimal amount of trauma and stress to the exotic animal. Furthermore, the safety of zoo personnel handling the animals must be considered. If chemical restraint is used, a complete physical examination should ensue. There are limitations, however. For example, body temperature is often difficult to evaluate because the animal's temperature may rise 2-3°C (4.4-6.6°F) in association with the stress of capture. Hypothermia is usually more significant, indicating a severely debilitated animal which needs an external heat source. In a manually restrained animal, abdominal palpation, cardiopulmonary auscultation, and other routine diagnostic procedures may be limited because of tenseness and struggling on the part of the animal.

A good practice also is to obtain a blood sample, even if the animal is examined for routine purposes. Complete hematological studies and serum chemistries should be done to fill the void of information on baseline values for exotic species. A data bank of this information should be at hand. Although there are increasing numbers of reports in the literature of normal baseline data, there may be some variation from one collection to another.

RESTRAINT AND ANESTHESIA

The question frequently arises as to whether to restrain an animal manually or to use chemical immobilization. In primates, we have found through blood gas studies[6] that it is better to chemically immobilize the excitable animals, even for routine procedures. Otherwise, during physical restraint severe metabolic and respiratory acidosis can occur, with blood pH reaching as low as 6.8[6]. It is therefore recommended that all diagnostic procedures in primates be performed with some type of dissociative anesthetic prior to the manipulative procedure. This is particularly true in animals with concurrent disease problems.

Once the decision has been made to chemically restrain an exotic animal, there are several basic types of drugs that can be used for this purpose.

Paralytic Drugs. Drugs inducing muscle paralysis, such as succinylcholine and nicotine sulfate, were the only types available to immobilize exotic animals 10-15 years ago and are mentioned mainly for historical purposes. Their use was plagued with relatively high mortality of 10% or greater, with the majority of the deaths due to asphyxia associated with overdosing. These drugs are not anesthetics and there is no analgesic effect at all—only paralysis. They are currently recommended for use only on occasion, for example, in management of deer herds such as white-tailed or red deer. The drug does provide a rapid knockdown and a person with experience can develop good proficiency.

Morphine Derivatives. A class of synthetic morphine drugs has evolved for the restraint and anesthesia of large exotic hoofstock. These drugs include etorphine (M-99, D-M Pharmaceuticals) and fentanyl citrate (Fentanyl, Janssen Pharmaceutical). They are most commonly used on exotic equidae, cervidae, and bovidae, as well as on elephants, hippopotamuses, and rhinoceros. These drugs are very potent and are capable of a rapid knockdown and a quick reversal with the antagonist, diprenorphine (M5050, D-M Pharmaceuticals). Disadvantages are extreme respiratory depression and special licensing requirements to obtain the drugs. They also are expensive, and can be a hazard to personnel using them if adequate precautions are not taken. Dosimetry is extremely varied and not always related to body weight. For example, in our experience a full-grown rhinoceros may require only a total dose of 1 1/2 mg, whereas a white-tailed deer weighing several hundred pounds may require 7 mg. There is a tremendous amount of species variation to these drugs, and familiarity with these differences is essential for their safe use[33]. These drugs are more commonly used in combination with other drugs, such as tranquilizers.

Dissociative Anesthetics. This is a relatively new class of anesthetics for use in human and small animal medicine. The parent drug, phencyclidine hydrochloride (Sernylan, Bio-Ceutic Laboratories), can be followed by ketamine hydrochloride (Ketaset, Bristol). At low dosages, these drugs produce a cataleptoid type of anesthesia with some muscle rigidity, but swallowing, blinking, and gag reflexes are present. They are also capable of producing surgical anesthesia. This class of drugs can be used in primates, small animals, marsupials, birds, and some of the small hoofstock.

A newer and very promising dissociative anesthetic that contains a tranquilizer, tiletamine-zolazepan (Telazol or CI-744, Parke-Davis), has been the subject of extensive clinical trials and, when available, should offer a reliable and safe anesthetic for use in many exotic species. It can be made in high concentrations, so low volumes can be used in projectile darts for immobilization.

Miscellaneous Immobilizing Drugs. Xylazine (Rompun, Haver-Lockhart) has marked sedative and immobilizing effects in some exotic species[53] and can either be used alone or in combination with the morphine derivatives or dissociative anesthetics. If used by itself, it will produce good anesthesia in certain species, especially the bovids. On the other hand, it must be used with caution in some aggressive animals, as they might appear to be adequately immobilized but, if stimulated, can sometimes mount a defensive attack.

Azaparone is another drug used in combination with morphine derivatives for immobilization of large hoofstock. This drug has been used extensively in Africa. The use of the drug alone has been reported to be very beneficial in the sedation of elephants[33].

Gaseous Anesthetics. For prolonged procedures or where supplementation of injectable anesthetics is required, we recommend halothane (Fluothane, Ayerst), with or without nitrous oxide. Mixtures of those gases can also be used for immobilization and restraint of birds, small primates, and reptiles. The animal is placed in a closed chamber which is filled with the anesthetic mixture. Once anesthetized, the animal can be removed from the chamber and maintained by either a face mask or intubation. For some hoofstock, halothane has proven safe and effective, but in ruminants or animals with a large cecum, nitrous oxide is contraindicated.

With the extreme variation of dosimetry of the various anesthetics, there is no easy method of presenting a chart for anesthetizing zoo animals. Instead, the class of immobilizing and anesthetic drugs which in our experience has proven to be successful are discussed. One important fact in anesthetic regimens is that dosages vary in different collections of animals. A dosage that works in one zoological collection may not work the same in another facility, even under similar conditions. Published dosages should only be used as guidelines. Individual animal dosages may have to be established by trial and error.

The method of injecting an anesthetic into an exotic animal depends upon the individual situation. If possible, the animal is best confined in a net or a squeeze cage and injected. At unreachable distances, pole syringes, blowguns, or guns ("dart" guns) which deliver drug-containing projectiles by CO_2 or powder charge are used[33]. Use of these guns requires care and experience because the projectiles can be lethal to surrounding personnel and animals if improperly used.

The following are guidelines for use of anesthetics in each of the following classes of animals:

1. **Reptiles.** Ketamine or CI-744 can be used in reptiles, but the induction and recovery times are prolonged. It is best to place the reptile in an airtight container such as a plastic trash bag or clear plastic tube (Figure 1) and fill it with 5% halothane and three parts nitrous oxide to one part oxygen. Induction time is from five to 30 minutes, varying with the type of reptile and its state of excitement. Once anesthetized, major surgery, such as removal of impacted eggs, can be performed.

2. **Birds.** Much work can be performed on physically restrained birds, but when needed, ketamine or CI-744 have provided adequate anesthesia; however, they do have a fairly prolonged and sometimes stormy recovery. Halothane, in most cases, has a fast induction and rapid recovery, but in excitable birds, sometimes there is acute respiratory and cardiac arrest. A sub-anesthetic dose of a dissociative anesthetic to calm the bird, followed by halothane and nitrous oxide, usually prevents that problem.

3. **Mammals.** In primates, the drug most widely used is ketamine. In marmosets and prosimians, anesthesia may be induced by placing them in a closed container with halothane and nitrous oxide. For most large hoofstock, the most widely used drug is etorphine, either alone or in combination with acepromazine, xylazine, or azaperone. There is much data on the use of these drugs in large ungulates[33]. For smaller hoofstock (under 15 kg), ketamine, in combination with xylazine, is effective. Xylazine has also been used alone. In bears, etorphine or phencyclidine in combination with promazine hydrochloride (Sparine, Wyeth) have been used successfully[61]. In medium-sized and small carnivores, the dissociative anesthetics are usually preferred. In the large cats, a relatively large volume (up to 20 cc) of ketamine is required for immobilization. This volume creates a problem and precludes the use of a dart gun, since maximum volume for projectile darts is 10 cc. In marsupials, dissociative anesthetics are the most often-used drugs[62]; however, they tend to have short immobilization periods and must be supplemented with either the same drug or with halothane.

BLEEDING PROCEDURES
The following is a list of techniques used to obtain blood samples from various species:

1. **Reptiles.** Snakes and lizards can be bled from the ventral tail vein[13] (Figure 2). Heart punctures are not recommended in these species. In the larger turtles, the jugular and veins of the posterior aspect of the leg can be used[58].

2. **Birds.** In bleeding birds, the medial wing vein is most frequently used, but it is delicate and hematomas can occur. In long-legged birds such as flamingos and cranes, blood samples can be obtained from the vein lying on the medial aspect of the metatarsal bone. In other species, the jugular may be used.

3. **Mammals.** The larger mammals are bled either from the jugular or tail vein, depending on how they are restrained.

MEDICATION
Dosimetry for medications used in zoo animals is often extrapolated from domestic animals and humans. This becomes quite critical for species with markedly different metabolic rates and other physiological differences. For instance, the half-life of the antibiotic gentamicin is 20 minutes in birds, two to four hours in mammals, and ranges from 40-80 hours in reptiles[14,15]. These factors must be considered when administering drugs that are potentially toxic or have low margins of safety. There are no good "rules of thumb" on this subject.

The best way to administer medication is by masking it in a palatable source. Medicating the water can be useful but some species can go for days without water. Injectables should be used with consideration for stress factors.

ORTHOPEDIC PROCEDURES
In general, bone fractures occurring in zoo animals are usually severe, i.e., comminuted, compounded; it is necessary to establish a rapid and stable fixation that requires minimal aftercare. Moreover, additional injuries can occur to the animal when progress checks are made. In mammals, the best method of internal fixation is with bone plates. In large mammals, such as adult lions, several bone plates may be required to provide adequate fixation[7] (Figure 3), but once established, the fracture usually remains stable and bone healing proceeds at a normal rate. Problems with bone plates have been encountered in extremely young, rapidly-growing animals in which mineralization of the cortical bone is not adequate to support the fixation device. Also, young animals, especially hoofstock, tend to self-traumatize areas on which fixation apparatus is placed.

For simpler fractures, coaptation with fiberglass splints (Lightcast II, Merck, Sharp, and Dohme) has many advantages. They are lightweight, extremely strong, waterproof, and can be applied rapidly (Figure 4). They also can be used in combination with some internal fixation devices[8].

Historically, bone fractures in birds have been a problem to treat. Even if repaired, secondary problems such as contracture, joint stiffness, and muscle shortening can render the wing or leg functionless. Our method of choice for avian fracture repair is either the half-pin or the full-pin splint[5]. These methods allow a rapid, solid fixation

Figure 1. Snake in plastic tube attached to anesthesia machine.

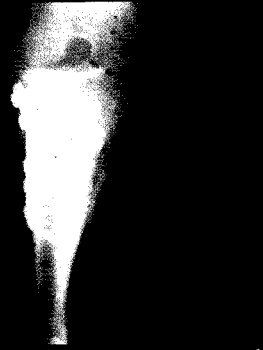Figure 2. Blood sampling technique, ventral tail vein of snake.

Figure 3. Radiograph of fractured tibia with bone plate in adult lion.

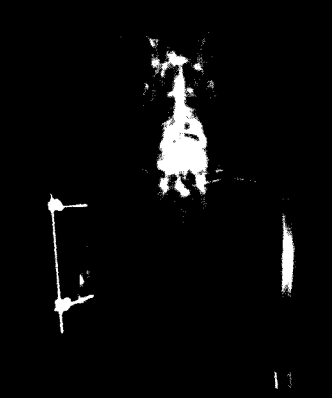Figure 4. Golden marmoset with fiberglass cast.

Figure 5. Fracture repair of the tibio-tarsus bone with half-pin splints in a red-tail hawk.

Figure 6. Coaptation of fractured tarso-metatarsus with fiberglass cast in an American merganser.

Figure 7. Laparoscopic view of ovary in a guinea fowl.

Figure 8. Laparoscopic view of testis in a greater sandhill crane.
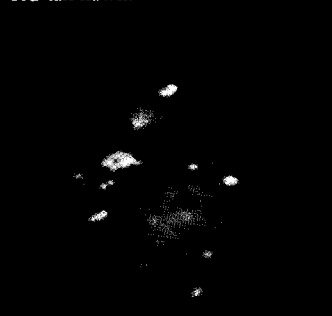
Figure 9. Laparoscopic view of tubercles in the liver of a guinea fowl.
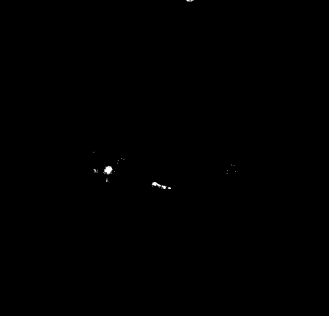
Figure 10. Laparoscopic view of a liver biopsy site in a cheetah.
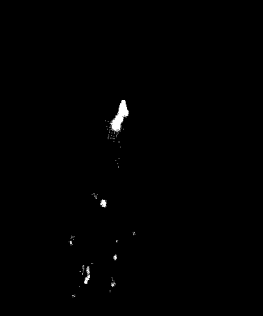
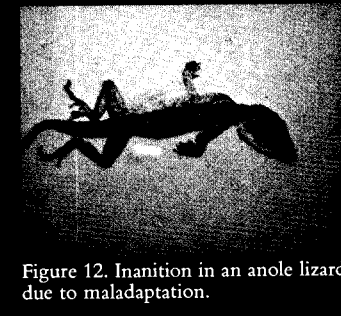Figure 11. Laparoscopic view of the ovary of a cheetah.

Figure 12. Inanition in an anole lizard due to maladaptation.

Figure 13. Turtle with Vitamin A deficiency eye lesions.

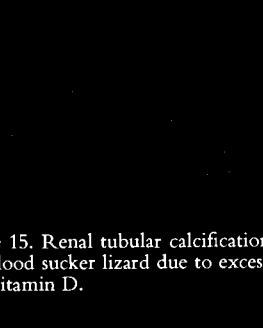Figure 14. Squamous metaplasia of ophthalmic gland (bottom) due to Vitamin A deficiency.

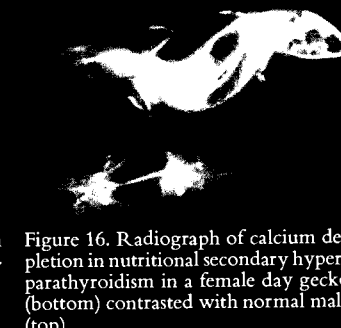Figure 15. Renal tubular calcification in a blood sucker lizard due to excessive Vitamin D.

Figure 16. Radiograph of calcium depletion in nutritional secondary hyperparathyroidism in a female day gecko (bottom) contrasted with normal male (top).

Figure 17. Radiograph of gastrolith in stomach of a leopard lizard over-supplemented with mineral mixture.

Figure 18. Gastrolith forms cast of stomach.

Figure 19. Visceral gout in the pericardium of a South American vine snake.
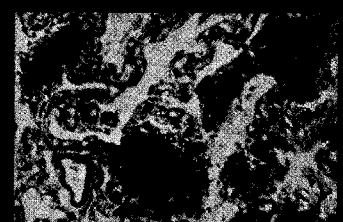

Figure 20. Gentamicin induced gout—urate crystals and tophi in the lungs of a bullsnake.

Figure 21. Slipped-tendon in a rhea.

Figure 22. Rhea with splay-legged condition.

Figure 23. Calcium deficiency in a young sun bittern. Radiograph shows pathologic folding fractures of lower limb.

Figure 24. Pale streaked heart of brown pelican with Vitamin E-selenium deficiency.

Figure 25. Microscopic appearance of Vitamin E-selenium deficiency cardiomyopathy in a brown pelican.

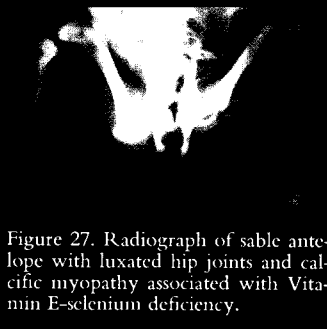Figure 26. Visceral gout, pericardium of North American wood duck.

Figure 27. Radiograph of sable antelope with luxated hip joints and calcific myopathy associated with Vitamin E-selenium deficiency.

Figure 28. Red and white streaked appearance of myocardium in a young camel due to Vitamin E-selenium deficiency.

Figure 29. Overgrown hooves in a sable antelope.

Figure 30. Demineralized and deformed skeleton of a young lion with nutritional secondary hyperparathyroidism.

Figure 31. Radiograph of ruminant-like compartmentalized stomach in a colobus monkey.
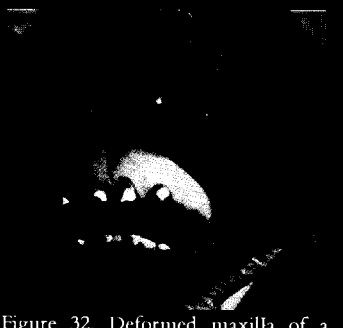

Figure 32. Deformed maxilla of a woolly monkey with nutritional secondary hyperparathyroidism.

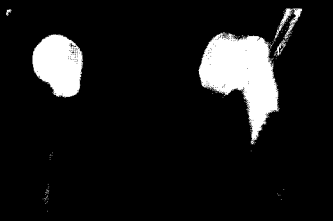Figure 33. Cephalohematoma in a squirrel monkey due to Vitamin C deficiency.

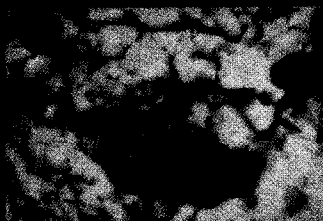Figure 34. Radiograph of monkey in Figure 33 with ossified hematomas.

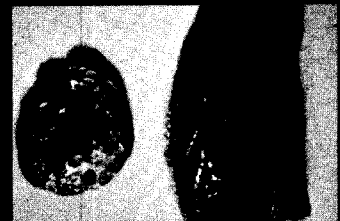Figure 35. Cysts of *E. invadens* show chromatoidal bars.

Figure 36. Thickened and necrotic colon in a snake with amebiasis.
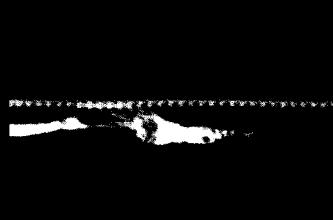
Figure 37. Trophozoite of *E. invadens*.

Figure 38. Barium study of snake with cryptosporidia associated hypertrophic gastritis.

Figure 39. Hyperemic and hypertrophied gastric mucosa associated with cryptosporidiosis in a corn snake.
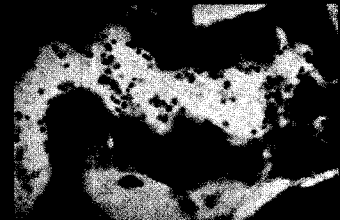
Figure 40. Giemsa stained section of stomach shows numerous cryptosporidia organisms.

Figure 44. Exoerythrocytic schizont of *P. elongatum* in a penguin.

Figure 41. Pentastomes in the lung of an indigo snake.

Figure 42. Mites (*O. natracis*) on the lower jaw of a boa constrictor.

Figure 43. Red blood cells from a boa constrictor with hemagregarine gametocytes.

Figure 45. Pneumonia associated with malaria in a penguin.

Figure 46. Caseous core in the cecum of a Swinhoe's pheasant with histomoniasis.
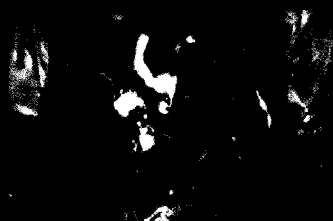
Figure 47. Liver lesions in a wild turkey with histomoniasis.

Figure 48. Microscopic appearance of histomonads in the liver of a wild turkey (PAS stain).

Figure 49. Ascarids obstructing the small intestine of an East African crowned crane.

Figure 50. Tumor-like nodules in the cecum of a golden pheasant with *Heterakis isolanche*.

(Figure 5) of the fracture without interference with the blood supply or the fracture site, particularly the endosteal callus which, in birds, is an extremely important uniting segment[11,50]. If intramedullary pins are used, their displacement of the medullary cavity disrupts the internal callus formation and can lead to prolonged healing or non-union fractures. The fiberglass cast can be used for fractures of the lower legs in birds (Figure 6). It is lightweight and birds can fly about the exhibit; wading birds can enter the water with it.

LAPAROSCOPY IN EXOTIC ANIMALS

We have found the laparoscope to be extremely useful in zoo practice. It can be used to sex birds that do not show sexual dimorphism as occurs in many exotic species[9] (Figures 7, 8). As a diagnostic tool, we have used it to screen for avian tuberculosis and aspergillosis (Figure 9). Since most exotic mammals have to be anesthetized for physical examination, laparoscopy can be performed at that time since the major danger is the initial anesthesia. We have successfully biopsied the liver and kidney of exotic cats (Figure 10).

The laparoscope is also useful in evaluating the ovaries and uterus to estimate reproductive capabilities in difficult breeders (Figure 11). Ovarian activity can be correlated with behavior, vaginal cytology, and serum hormone levels.

The use of laparoscopy in exotic animals is still in its infancy; it should enhance the overall health care of the animal population in a zoological environment.

NUTRITIONAL AND METABOLIC DISEASES

Of all the known entities that affect zoo animals, perhaps least is known about nutritional and metabolic conditions. Exact nutritional requirements are non-existent for most exotic species. Most dietary needs are extrapolated from their domestic counterparts. A great deal of empiricism is used when formulating diets. In most cases, diets that appear to adequately nourish the animal can be formulated. Currently, there are a number of diets, prepared specifically for exotic species, that can be obtained commercially. These can replace some of the archaic methods of feeding zoo animals. They can also replace whole-carcass and raw-meat feeding which, although necessary in some situations (for example, reptiles) and useful occasionally as a supplement or appetite stimulant, can promote the introduction of infectious diseases and parasites. Horse meat tained with anesthetic (used for euthanasia) can be deleterious to large cats, which are exquisitely sensitive to barbiturates[16].

Nutritional and metabolic problems vary considerably among the classes of animals. In the following discussion, some common problems are outlined for each, along with appropriate therapy.

1. **Reptiles and Amphibians.** Failure "to feed," particularly in new acquisitions and neonates, leads to states of inanition and dehydration (Figure 12) and finally to death. Usually there are no identifiable organic disturbances and the causes are more likely associated with stress and maladaptation[20]. Snakes in this condition may be fed the following formula: 1 raw egg, 1 jar of strained baby beef, 1/2 teaspoon of a vitamin supplement, 1 gram of chlortetracycline (soluble powder), 500 mg of vitamin C, and 50,000 units of vitamin A. The mixture is prepared in a blender and administered by stomach tube at 5 cc/kg body weight every 10 days. Snakes can also be force fed with newborn mice (pinkies).

Environmental temperature is extremely important. If the reptiles are too cold, food may putrify in the digestive tracts. Temperature

recommendations are 24-29°C (75-85°F) for most snakes and turtles, although some lizards such as the iguanids need a range from 29-37°C (85-100°F)[40].

Although not well studied, a number of vitamin and mineral deficiencies in reptiles have been reported[65]. Vitamin A deficiency is most commonly observed in chelonians, in which the ophthalmic glands undergo squamous metaplasia, swell, and displace eyelid and conjunctiva (Figures 13, 14). Squamous metaplasia may also occur within visceral organs[23]. Daily supplementation with several drops of cod liver oil or a vitamin mixture (Pervinal, Mitchum-Thayer, Inc.) is usually effective in treating this condition.

Although rachitic-like conditions probably associated with vitamin D deficiency occur, more common is the over-use of vitamin D, which results in metastatic calcification and renal failure (Figure 15). Calcium deficiencies are not uncommon in smaller reptiles whose diet may be limited to meat, fish, and insects. This leads to abnormal calcium-phosphorus ratios and fibrous osteodystrophy of long bones associated with nutritional secondary hyperparathyroidism (Figure 16). Lime water used as a drinking source or calcium carbonate added to the diets are useful preventives. Calcium requirements of breeding females are higher during egg-laying periods. Over-supplementation, however, should be guarded against, as gastroliths believed to be associated with excessive intake of calcium sources have been observed in lizards (Figures 17, 18).

Other nutritional problems encountered in reptile collections include vitamin E deficiency-steatitis of croccodilians, goiter with myxedema in chelonians, which is responsive to iodized salt, and vitamin C deficiency, thought to be a predisposing factor to stomatitis (mouth rot) in snakes and lizards.

Gouty conditions (Figure 19) are common in reptiles and have been associated with high protein intake and states of dehydration[1]. Gout can also be induced by renal failure, as most terrestrial reptiles metabolize their waste nitrogen to uric acid, which is subsequently secreted by the proximal tubules of the kidney. Recently, it has been shown that gentamicin, an extremely effective antibiotic in reptiles but also potentially nephrotoxic, can produce gout (Figure 20) if given at mammalian dose schedules[52]. Because of the extremely long half-life of this antibiotic in reptiles, the recommended dosage which produces effective plasma levels at minimum risk is 2.5 mg/kg every 72 hours[14,15].

2. **Birds.** Dietary habits and requirements of captive exotic birds are as variable as the vast numbers of different species that occur in the avian family. Some birds may have highly specific requirements (e.g., hummingbirds need nectar), and many birds are strictly insectivorous, granivorous, herbivorous, carnivorous, or omnivorous. One must therefore be familiar with the peculiarities of each order or species. A great deal of information on nutritionally-related diseases has been extrapolated to exotic gallinaceous birds and, to some extent, to anseriformes from the large amount of knowledge accumulated on domestic poultry and ducks.

Some of the classical vitamin and mineral deficiencies such as curley-toe paralysis and perosis (slipped-tendon) (Figure 21) are not uncommon in gallinaceous chicks and ducklings. Some of these conditions respond to riboflavin in the former case, and manganese, choline, biotin, in the latter case. There are, however, a number of other neonatal abnormalities including "splay-legs" (Figure 22), torticollis, and some forms of perosis which do not respond to conventional supplementation and can have a genetic basis.

A great deal has been learned about feeding exotic birds by trial and error. Some birds such as ratites (ostrich-like) must even be trained
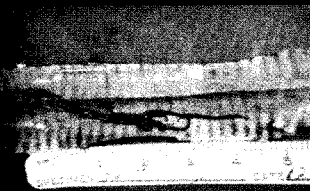
Figure 51. Trachea of a young rhea with syngamiasis.

Figure 52. Characteristic operculated eggs of *S. trachea.*

Figure 53. Scaly leg disease (cnemidocoptic mange) in a psitticine.

Figure 54. Radiograph of pulmonary densities due to angiostrongylosis in a fennec fox.

Figure 56. Microscopic appearance of adult angiostrongyles in the pulmonary artery of a fennec fox.

Figure 55. Verminous pneumonia due to *Angiostrongylus sp.* in a fennec fox.
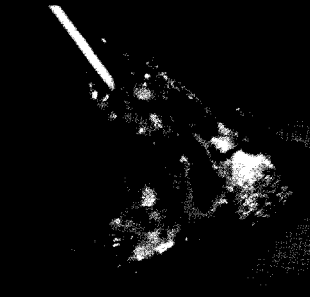
Figure 57. Necrotic stomatitis (mouth rot) in a boa constrictor.
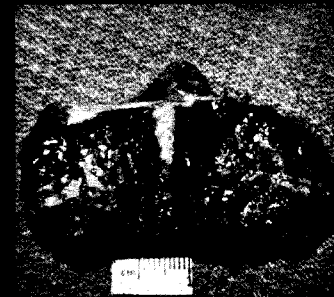
Figure 58. Aeromonas pneumonia in a Malaysian water monitor.

Figure 59. Pseudomonas colitis in a reticulated python.

Figure 60B. Herpetic-like inclusion bodies in the oral mucosa of lizard in Figure 60A.

Figure 61. Avian tuberculosis, liver of a golden pheasant.
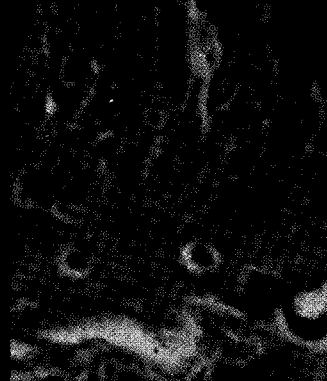
Figure 60A. Necrotic stomatitis in a Malaysian water monitor.

Figure 63. Radiograph of wing joint of a spur-winged plover with avian tuberculosis.

Figure 62. Avian tuberculosis, spleen, of a kildeer.

Figure 64. Abdominal air sacs of a bufflehead duck with aspergillosis.

Figure 65. Esophageal pseudomembrane due to DVE in a North American black duck.

17

to eat by using turkey poults which are allowed to graze with them. When ratites have too rapid growth from diets excessively high in protein and energy (for example, laying mash) it may lead to leg weaknesses and "downer birds." Prepared diets are available to prevent this condition. Birds strictly on sunflower seeds (as occurs with psitti-cines), insectivorous birds, and carnivorous birds on pure red-meat diets are prone to nutritional secondary hyperparathyroidism with resultant osteodystrophic lesions of the skeleton. Clinically, these birds may show tetanic seizures with radiographic evidence of cortical bone thinning and occasionally pathologic fractures[66]. Similar bone changes have also been observed in neonatal long-legged birds (Figure 23) that have responded to calcium supplements. A number of other deficiencies are suspected in exotic birds, although little documented evidence exists. Vitamin E-selenium deficiency, which produces well-known, naturally-occurring syndromes in poultry, has been suspected as the cause of such conditions as yolk-sac abnormalities, poor hatch-ability, and myopathies in exotic birds. Based on these observations, all of our diets where feasible are supplemented with vitamin E-selenium at a level of .2 ppm Se and 132 I.U. vitamin E/kg of feed. A most striking skeletal and cardiac myopathy, suspected of being vitamin E-selenium deficiency-related, occurred in a flock of brown pelicans (*Pelicanus occidentalis*) (Figure 24) whose diet consisted of frozen smelt. Birds were found dead or died even on being approached or when handled, due to extensive cardiac lesions (Figure 25).

Frozen fish may also contain thiaminase, necessitating thiamin replacement in birds fed solely these frozen diets[67].

Another common metabolic condition of zoo birds is gout. Articular gout manifests clinically as lameness with the formation of urate deposits (tophi) which are often visible as whitish thickenings around the joints. Visceral gout is generally a postmortem finding with the white-chalky tophi in serous membrane and within visceral organs (Figure 26). The specific causes and mechanisms of avian gout, again, are not well known, but have been associated with excessive protein intake, vitamin A deficiency, renal disorders, toxemias, and debility. Therapy is aimed at rectifying these conditions. Drugs used in human gout are generally not effective and not recommended for the avian forms.

3. **Mammals.** At one time, a number of conditions in our exotic hoofstock were attributed to vitamin E-selenium deficiencies. These included skeletal (Figure 27) and cardiac myopathies, obscure lame-nesses, muscle weaknesses, and failure to suckle in neonates. Sudden death associated with vitamin E-selenium deficiency (Figure 28) has been reported in young camels[25]. Because of this, all commercially prepared diets for mammals are supplemented with .2 ppm selenium and 66 I.U. vitamin E/kg of feed. These substances are incorporated by the feed manufacturer and because of the potential toxicity of selenium, should not be added to the animal's diet by hand. All hay is obtained from selenium-rich areas. Since the initiation of this supple-mentation program, conditions formerly attributed to vitamin E-selenium deficiency no longer occur.

Another common problem in exotic hoofstock is over-supplemen-tation with protein. Many of the exotic species are browsers, unaccus-tomed to rich concentrate feeds. This is thought to lead to overgrown hooves (Figure 29), obesity, and intestinal disorders.

Exotic cats, particularly the young, on all-meat diets are subject to secondary hyperparathyroidism with demineralization of skeleton and pathological fractures (Figure 30). Balanced, prepared diets are avail-able (for instance, Zu-preem, Nebraska Feeds Co.), or meat can be supplemented with bone meal.

In primates, there may be a large diversity of dietary needs. Some primates are carnivorous with protein requirements as high as 26%, while others (for example, Colobus) are herbivorous with large rumi-nant-like stomachs (Figure 31). Nutritional problems readily occur in these types of monkeys, as little is known about their dietary require-ments. "Cage paralysis," "simian bone disease," and "woolly monkey disease" are well-known entities of New World primates fed solely fruit diets. They may develop pathological fractures and thickening of the facial bones (Figure 32). These may be accompanied by low-grade respiratory infections and continuous mild gastroenteritis. Death can occur due to respiratory failure associated with collapse of the rib cage. Causes for these entities are attributed to low protein, high phos-phorous, low calcium, and inadequate vitamin D. New World monkeys require vitamin D-3 if they are housed indoors, unexposed to an ultraviolet source. Change to prepared diets can result in some improvement, but complete resolution of the deformed·bones will not occur. On the other hand, care must be taken when strictly using some of the commercially-prepared diets, as vitamin-C deficiencies can occur if the feed preparations are stored too long. This can lead to scurvy, or in squirrel monkeys, subdural hematomas[2]. The hematomas may ossify and create hard, unsightly swellings of the skull (Figures 33, 34). Correction of the diet and the surgical drainage of the hema-tomas is usually all that is required.

Often overlooked in exotic animals is malnourishment associated with management problems. This can occur, for example, in a herd or flock situation in which subordinate animals are kept away from feeders by dominant animals. It is often necessary to provide extra feeders that are well spread out to insure that all animals have access to the feed. Although most common in reptiles, some newly acquired mam-mals may not eat because of problems adjusting to their new environ-ment. This requires that the animals be placed in a secluded area and given individual attention.

PARASITISMS

Diseases due to parasites can be a detriment to zoological collections. Rigid parasite surveillance, as previously outlined, and regular treat-ment with anthelmintics is essential. Many animals arrive heavily para-sitized. Depending on the parasite's life cycle, these new arrivals may introduce and perpetuate the condition among their cagemates. On the other hand, many parasite life cycles requiring intermediate hosts that are not accessible in a zoo setting are broken, thereby eliminating the parasitism. Stress and nutrition often play a major role in how the zoo animals adapt to their parasitisms. The following parasites are most troublesome in zoo animals. Treatments, where known, are given.

1. **Reptiles.** Some important parasitic diseases of snakes and other reptiles are as follows: amebiasis (*Entamoeba invadens*) can produce explosive epizootics or smolder as sporadic cases in reptile collections. Turtles, usually resistant to this disease, may carry and shed the tetra-nucleate cysts (Figure 35). When ingested by snakes, the cysts develop into the infective trophozoites which produce severe necrotic gastro-enterocolitis (Figure 36). The amebas may spread to the liver and occa-sionally the kidney and produce necrotic lesions. Snakes sick with amebiasis may show anorexia and general malaise. There may be swell-ing in the posterior third of the body created by a thickened colon. Blood or mucus may be found in the feces, with smears showing amebic trophozoites (Figure 37). Treatment consists of diloxanide furoate (Furamide, currently unavailable in U.S.), 0.5 gram per kilo-gram orally, plus a diiodohydroxyquin (Diodoquin, G.D. Searle) enema. Metronidazole (Flagyl, Searle), a standard amebacide for hu-
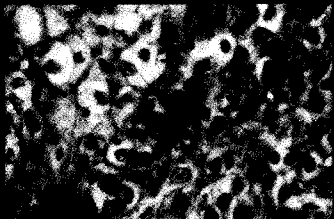
Figure 66. Herpetic intranuclear inclusion bodies, DVE, in a wood duck.
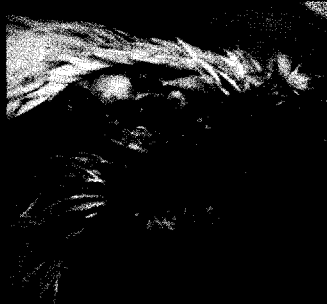
Figure 67. Pox in a Rothschild's mynah bird.

Figure 68. Mycotic abomasitis in a giraffe calf. Silver stain of phycomycetes in mucosa and submucosal blood vessel.

Figure 69. Osteolytic lesion due to phycomycosis in the radius of a baby reindeer.

Figure 70. Gross appearance of lesion in Figure 69.
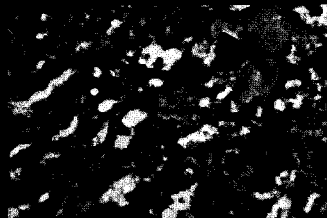
Figure 71. Microscopic appearance of phycomycetes from bone lesion in Figure 20.
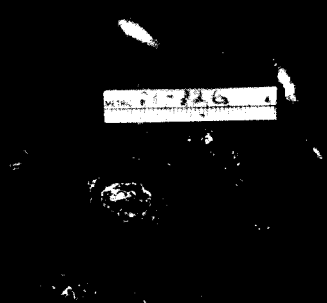
Figure 72. Necrotic enteritis and peritonitis due to yersiniosis in a blesbok.
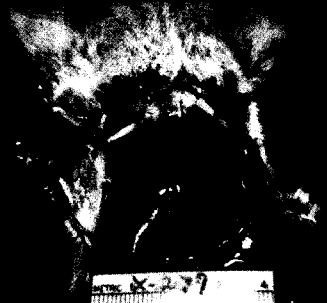
Figure 73. Wild rat captured at zoo with yersinia lesions in the liver.

Figure 74. "Lumpy jaw" in a red kangaroo.
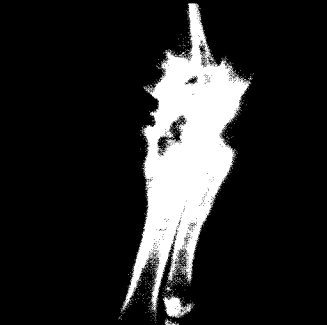
Figure 75. Radiograph of hock joint with tuberculous arthritis due to *M. avium* in a rat kangaroo.
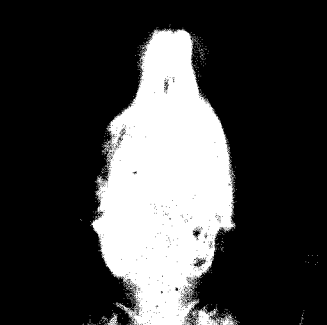
Figure 76. Inflamed tympanic bullus of brush tail rat with pseudomonas otitis media.

Figure 77. Gross appearance with obliteration of one of the middle ear chambers (top right) from Figure 76.

Figure 78B. Disseminated Herpes viral infection in a monkey. Focal necrosis in the liver.

Figure 78A. Disseminated Herpes viral infection in a monkey. Ulcers in the tongue.
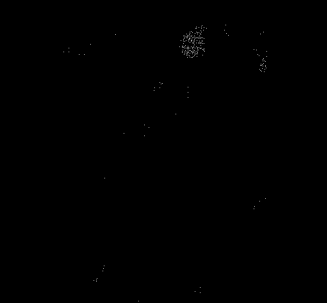
Figure 79. Intranuclear inclusion bodies due to *Herpes T.* in the tongue of a tamarin.

19

mans, has been shown to be effective at 275 mg/kg given by stomach tube in one dose[22]. Prophylactic steps include never mixing turtles and other reptiles with snakes and strict quarantine procedures for new arrivals.

In snakes, coccidiosis due to *Eimera sp.* usually occurs sporadically and can be found incidentally or in stressed individuals at necropsy. Rabbit coccidia may appear in the feces of snakes fed whole rabbits but do not represent true infections. The rabbit coccidia can be distinguished microscopically from the reptilian forms. Recently, cryptosporidiosis associated with hypertrophic gastritis (Figures 38, 39) has been described in snakes[18] which develop mid-abdominal swelling and have postprandial vomiting. The disease is progressive (weeks to months) and usually fatal. Diagnosis can be made by direct fecal smear with Jenner-Giemsa stains which reveal oval-to-round bodies 2-4 μm in diameter (Figure 40).

Important helminths of snakes include a variety of cestodes treatable with niclosamide (Yomesan, Chemagro) at 125-250 mg/kg, and several common nematodes including: Ophidascaris (round-worm, intermediate stages in amphibians), Physoloptera (stomach worms), and Kalicephalus (hookworm-like), found throughout the digestive tract[47], and oxyurids of turtles and lizards[38]. All of these nematodes respond to some degree to 200-400 mg/kg of thiabendazole. The latter drug may be toxic in king snakes and geckos. Dichlorvos (Task, Shell) at 12-20 mg/kg daily for two days given in "feed-mice" or by stomach tube also is an effective anthelmintic but should be used cautiously.

Several types of pentastomes whose intermediate stages occur in small rodents, carnivores, and primates can be found in the lungs of snakes and frequently cause obstruction of the air passages (Figure 41). There is no practical therapy; the condition is frequently found at autopsy.

Ectoparasites including skin mites (*Ophionyssus natricis*) (Figure 42) can be treated with a desiccant powder (Dri-die-67) or with insecticide strips (Shell) which are placed near the cage for six to eight hours. This is repeated in ten days. The snake mites may produce anemia, and a hemorrhagic septicemia due to *Aeromonas hydrophila* has been associated with them. The mites are also a vector for hemogregarines, red-blood cell parasites (Figure 43), and therefore should be eliminated.

2. **Birds.** In zoo birds, protozoal parasites of the red blood cells are common[46] but seldom give rise to serious problems. An exception is malaria, one species (*Plasmodium elongatum*) of which has been reported in African penguins (*Spheniscus demersus*). It produces a rapidly fatal disease in these birds which apparently are poorly adapted hosts. This form of malaria is characterized by extensive exoerythrocytic schizogony (Figure 44), resulting in inflammation of reticuloendothelial organs, pneumonia (Figure 45), and hepatitis, but little anemia or icterus[28]. Treatment consists of triple sulfa given orally at 3.5 grains/kg daily for ten days. Another important protozoal disease of exotic gallinaceous birds is blackhead (*Histomonas sp.*), which in our collection has occurred in Swinhoe's pheasants (*Lophura swinhoei*) and Eastern wild turkeys (*Meleagris g. sylvespris*)[45]. Lesions are typical of those occurring in domestic turkeys and consist of caseous cecal cores (Figure 46) and yellowish necrotic hepatic (Figure 47) foci. Microscopically, liver lesions may contain numerous histomonads (Figure 48). The carrier nematodes, *Heterakis gallinarum*, are frequently in the cecae. Treatment with dimetridazole (Emtryl,Rohm) placed in the feed has been effective. Ideally, removal of the birds from the ultimate source of the infection, the earthworm, which contains the histomonad-infected heterakid larvae, is the best but not always the most practical way to control the diesase.

Coccidia are commonly found in many birds and may be a clinical problem in some collections. Diagnosis and treatment would be similar to that of domestic poultry.

There is an endless list of helminths[21] which have been recovered from exotic birds but few occur epornitically. Ascarid infestations may be a problem in exotic pigeons (Columbiformes) as well as in psitticines and cranes. Massive parasitic loads can occur resulting in debility and sometimes intestinal obstruction and death (Figure 49). Treatment for ascarids consists of piperazine adipate by stomach tube at a dose rate of 440-660 mg/kg of body weight.

Heterakis gallinarum and its complications have been mentioned. Another heterakid, *H. isolonche*, can cause a chronic wasting disease in pheasants and produces nodular typhlitis with tumor-like lesions in the cecal wall (Figure 50) of golden pheasants (*Chrysolophus pictus*)[32,36].

Syngamiasis due to gape-worms (*Syngamus trachea*) is not uncommon in commercial and game birds and has been reported in many avian orders. The worms attach to the tracheal mucosa and can produce almost complete obstruction. We have observed syngamiasis in young common rheas (*Rhea americana*) which showed classical gaping (Figure 51). Eggs, which are doubly operculated, were found in the feces (Figure 52). Treatment with thiabendazole at 300 mg/kg weekly for three weeks proved effective.

Ectoparasites including many species of mites and lice can be safely treated with pyrethrin-based sprays. A scaly disease of the face and legs is common in some passerines and psitticines and is caused by Cnemidocoptic mange mites (Figure 53). Treatment is most effective when the condition is diagnosed early and consists either of the application of A&D ointment to the affected area three times weekly, which smothers the mites, or the use of an acaricide, Dettol (R. T. French Co.) A 10% solution of this drug is applied to affected areas with a cotton swab daily for one to two weeks[34].

3. **Mammals.** Parasitic diseases can cause great losses, particularly in exotic hoofstock. This is mainly because of the lack of dilution factors as in the wild or the inability to provide pasture rotation as in domestic herd settings. Most exotic animals are kept at high population densities and therefore parasitisms should be treated on a herd basis. Furthermore, with the advent of the larger habitat-oriented exhibits, parasite eggs can accumulate in these areas more readily.

Parasites most dealt with are those of the digestive tract common to ruminants (with similar incidences and propensities for them). For example, exotic sheep (Mouflon) must be dewormed regularly for *Hemonchus sp.* and many other types of nematodes. Zebra, onagers, and other exotic equidae must be continually surveyed and dewormed for strongyles and ascarids. All of these parasites are controlled well with anthelmintics used in domestic livestock including thiabendazole and mebendazole.

Coccidia are commonly found in many of the hoofstock species and are usually of no consequence except in young animals. The coccidiostat, amprolium, may be pre-mixed at the level of 330 mg/kg of feed. Coccidiosis can be a major problem in marsupials and should be treated either with sulfamethazine or amprolium, particularly if young marsupials are being raised[27].

Parasites of exotic carnivores are very similar to those encountered in a pet animal practice with the exception that in bears and large cats, ascarids may be a lifelong problem. Therefore, periodic treatment with piperazine adipate at standard dosages should be employed. Newly acquired exotic carnivores may arrive with unusual parasitisms. A fennec fox (*Fennecus zerda*) died of respiratory distress shortly after arriving at our zoo (Figure 54). At necropsy, there was severe ver-

minous pneumonia due to *Angiostrongylus sp.*) (Figures 55, 56), a nematode that harbors in the pulmonary circulation.

Other anthelmintics used successfully in carnivores are thiabendazole, niclosamide, levamisole (Ripercol L), and disophenol (DNP, American Cyanamid Co.—for hookworms).

Most of the major parasitic diseases of primates are well covered in standard texts[24,29]. Two that are encountered frequently in zoo settings are amebiasis (*E. histolytica*) and strongyloidosis (*Strongyloides spp.*). Amebiasis can be symptomless or cause protracted cases of diarrhea and dysentery. Visceral amebic abscesses as occur in man are uncommon. Treatment is with metronidazole at 35-50 mg/kg div BID, and paromomycin sulfate (Humatin, Parke-Davis), 25-35 mg/kg div BID. Several strongyloides species can infect a variety of zoo primates and because of the nature of their life cycle, can produce super-infections resulting in severe debility and death. Effective control consists of thiabendazole at 100 mg/kg and proper hygiene measures. Both amebiasis and strongyloidosis can be zoonotic and personnel handling these primates should be made aware of this possibility.

INFECTIOUS DISEASES

There are a number of ways that infectious diseases can enter a zoological collection. Infections can be brought in by feral animals, wild birds, and vermin that commonly take up residence, and even by stray pets that may pass through the grounds. Newly acquired specimens may introduce organisms, and zoonotic diseases such as human tuberculosis may be acquired from infected handlers and even from visitors. Stress and nutrition play a major role in the spread of infectious diseases, as many infections such as mycoses are merely opportunistic in nature.

1. **Reptiles and Amphibians.** In this class of animals, bacteria, including *Aeromonas hydrophila*, *Pseudomonas sp.*, and *Klebsiella*, can cause a variety of potentially lethal syndromes. "Red-leg" is one in amphibians which may respond to chloramphenicol succinate at 2-4 mg/kg once daily. In snakes, these organisms are often associated with necrotic stomatitis (Figure 57) or pneumonia (Figure 58), which causes gaping of the mouth and a bubbly wheeze, or severe entercolitis (Figure 59). *Salmonella* and *Arizona* can likewise be cultured from these processes[4,37]. In turtles, a septicemic cutaneous ulcerative disease (scud) can be caused by *Citrobacter freundii*[39].

Many of these antibiotic-resistant gram-negative infections respond to gentamicin, but as noted above, dosage levels are critical in reptiles (rec. 2.5 mg/kg every 72 hours), and good hydration must be maintained.

Mycotic diseases are not overly important and reptilian tuberculosis, usually due to *Mycobacterium marinum*, is uncommon in well-managed colonies[17].

Although we have seen a number of diseases of probable viral origin (based on morphologic changes) (Figure 60) which appear to underlie some of the bacterial septic conditions, traditionally, viral diseases have been of little consequence in reptile collections.

2. **Birds.** Of all zoo species, the birds seem most prone to and appear to have the highest frequency of infectious diseases. Many can occur as epornitics which can literally decimate an aviary. Usually there are few clinical signs and birds either die suddenly or are found dead. A specific disease outbreak should be suspected when many of the same species are dying. Complete necropsies and cultures should be performed to distinguish infectious entities which might grossly re-

semble each other. *Salmonellosis*, fowl cholera (*Pasteurella multocida*)[21], and chlamydiosis (*Ornithosis-psittacosis*)[59] must be ruled out in such outbreaks.

Avian tuberculosis (*Mycobacterium avium*) often occurs endemically in zoo aviaries[53] and diagnostic techniques as used in mammals, including tuberculin testing, are ineffective, as are methods of treatment. The disease primarily affects the digestive tract with involvement of liver, intestines, and spleen (Figures 61, 62). It is progressive, primarily affects older birds, and except for bone involvement in a few cases (Figure 63), may go unnoticed until birds become emaciated in the advanced stages. Avian tuberculosis has been a problem in our collection. The incidence has been drastically reduced by removing all birds to a temporary source, rigorously disinfecting the permanent facility (including removal of dirt flooring), and screening all birds in the collection. Birds that were unduly thin, had high white blood cell counts, or had visceral lesions seen by laparoscopy were culled[12]. A recently developed enzyme-labeled antibody test for detecting antibodies in tuberculous birds shows promise in making the screening task an easier one[64].

Birds are exquisitely sensitive to mycotic diseases, with aspergillosis (*A. Fumigatus* and *A. flavus*) and candidiasis being most common. Anseriformes and seabirds appear to be most susceptible to aspergillosis, particularly under conditions of stress or debility. Clinical diagnosis is difficult, but aspergillosis should be suspected in waterfowl which show respiratory distress. Some results are claimed with amphotericin-B, nebulized, with the bird in a "tent." However, most cases of aspergillosis are found at necropsy with extensive pulmonary and air saccular fungal plaques and marked inanition (Figure 64).

Many viral diseases can threaten otherwise stable exotic bird collections. We experienced an epornitic of duck viral enteritis (DVE), which is a herpes virus that can also infect geese and swan. The disease was believed to be brought in by wild migratory waterfowl. Classically, DVE occurs in the spring and may produce extremely high mortality. Lesions typical of DVE including serosal hemorrhages, focal hepatic necrosis, pseudomembranous esophagitis (Figure 65), and necrotic plaques of the intestinal and cloacal mucosa were evident in many of the affected birds. Characteristic herpetic inclusions were found in many of the mucosal lesions (Figure 66). Vaccinations of all susceptible birds with MLV vaccine and extensive sanitation and quarantine procedures were used to control the outbreak[51].

Other herpes viral infections of zoo birds, such as hepatosplenitis of birds of prey[48], occur more sporadically, perhaps due to the more solitary habits of these birds.

Pox may develop in aviaries and infect only one or two species, despite direct contact between infected and noninfected birds. Host adaptability plays an important role for this virus, of which there appear to be a number of strains. In our aviary, Rothschild's mynahs (*Leucopsar rothschildi*) developed pox (Figure 67) which was brought in by starlings[44]. Generally, the disease is milder than fowl pox and can be controlled by isolating the infected birds and treating the lesions palliatively.

Exotic Newcastle disease is currently under a national surveillance program, authorized by the USDA, because of the potential threat to the poultry industry. This disease produces massive hemorrhages in the intestinal tract and reproductive organs and the birds often die before showing other signs[56].

3. **Mammals.** Tetanus has been observed in elephants and zebras and is preventable by use of tetanus toxoid vaccination. Other clostridial diseases such as black-leg, malignant edema, and some of the entero-

toxemias have been reported in geographical locations where there is a high clostridial disease incidence in the domestic species. A number of bacterins are available and can be useful where cases are more than sporadic.

Primary mycotic diseases are uncommon but may occur, particularly in the young and the infirm. Mycotic abomastitis has been seen in giraffe calves (Figure 68). Systemic phycomycosis due to *Absidia corymbifera* has occurred with a high frequency in calves in our herd of reindeer[42]. Early signs include lameness due to joint involvement (Figures 69, 70) with progression of lesions to visceral organs and the brain (Figure 71).

There is some morphologic evidence that these mycotic infections may be associated with an immunodeficiency disorder such as occurs in Arabian foals.

Yersiniosis (*Yersinia pseudotuberculosis*) has been reported endemically in primates, small rodents, and birds in several zoos[55]. An outbreak of this disease occurred in our blesbok (*Damaliscus dorcas*), which developed an acute fulminating disease and died rapidly[3]. The disease was characterized by psueodomembranous enteritis, with abscesses disseminated in many visceral organs, and peritonitis (Figure 72). The yersiniosis was traced to wild rats and pigeons, thereby focusing the control of this disease outbreak on the eradication of these vermin (Figure 73).

Malignant catarrhal fever is a sporadic, fatal viral disease that has occurred in our reindeer and cape buffalo (*Syncerus caffer*). Wildebeest (*Connochaetes taurinus*) are asymptomatic carriers and may shed the virus during parturition. Wildebeest or sheep, also incriminated as carriers, should not be mixed with other hoofstock or even housed in adjacent pens.

Epizootic Hemorrhagic Disease (EHD) is an orbivirus infection of deer (*cervidae*) that produces fever and extensive visceral hemorrhages leading to profound shock and death. Although principally a disease of North American white-tailed deer (*Odocoileus virginianus*), EHD, along with Blue Tongue, a similar viral disease, has been reported in exotic cervid collections[32]. There is no specific treatment and currently no vaccines are available for these diseases.

Marsupials are relatively susceptible to several types of bacterial infections. Kangaroos develop "lumpy jaw" (Figure 74) which resembles the syndrome of cattle but is usally caused by *Fusobacterium necrophorum*, although other organisms including the actinomycetes and mycobacteria have reportedly been cultured from them[26]. Recently, *Bacteriodes ruminicola* was isolated from a group of captive Bennett's wallabies (*Protemnodon r. frutica*) with "lumpy jaw" in Ireland[41]. The disease appears to be associated with molar eruption and begins as a periodontitis, progressing to osteomyelitis. Crowding, coarse foods, and trauma may be contributory. An affected kangaroo becomes unthrifty and can be seen frequently wiping the sides of its face. These animals should be anesthetized and treated by debriding oral lesions and removing any loose teeth. Oxytetracycline should be given daily intravenously for two to three weeks. In cases from which *B. ruminicola* is isolated, therapy with metronidazole or clindamycin is suggested[41]. In advanced cases, radical surgical drainage of the bony lesions must be instituted.

Tuberculosis, particularly due to *Mycobacterium avium*, is not uncommon in marsupials and appears to have a propensity for bone. We have had a case of disseminated *M. avium* in a tree kangaroo (*Dendrolagus matschiei*) which started in a tail wound and became generalized, with cerebral and ocular involvement occurring within six months[53]. Another case occurred in the hock joint of a rat kangaroo (Figure 75). Diagnosis of these atypical tuberculous infections is sometimes difficult, since tuberculin testing is usually inconclusive; however,

radiography and acid-fast stains and cultures of biopsied lesions can provide the answer. Mycobacteria recovered from marsupials or any animals should be typed[63], since *M. avium* is resistant to all of the currently-used anti-tubercular drugs. Animals infected with these resistant organisms may have to be euthanized since *M. avium* can be progressive and potentially contagious to other marsupials. Marsupials should not be mixed with nor housed adjacent to aviaries, particularly where avian tuberculosis has been or is known to occur.

The infectious diseases occurring in exotic rodent colonies are similar to those of conventional laboratory animals. We have observed otitis media (Figures 76, 77) due to *Pseudomonas aeruginosa* in degu (*Octodon degus*)[35], which was controlled by acidifying the drinking water. Sporadic cases of respiratory infections and local abscesses associated with cage aggression usually respond to isolation and antibiotic therapy. Malocclusion of the incisors may lead to debility and secondary infection and is preventable by periodic dental checks and incisor trimming if necessary.

Susceptibility of exotic carnivores to canine and feline distemper and their vaccination procedures were discussed earlier, in the section on "Preventive Medicine." Both of these diseases can be rapidly fatal and can vary from the classic signs well known in the dog and cat, to rapid acute forms which have minimal signs and are more common in exotic species. Carnivores dying in this manner should be screened for these viruses, necessitating the freezing of appropriate tissues at −50°C.

Carnivores are also prone to septic conditions, often due to bite wounds which are not readily seen under long haircoats.

As discussed, the majority of the infectious diseases of primates should be eliminated when quarantining them. Once in the collection, however, each primate should undergo a yearly tuberculin test. In cases where outbreaks of tuberculosis have occurred, testing should be performed more often. Specific details of tuberculin testing in primates are available elsewhere[49]. Chronic diarrhea is very common and may lead to marked dehydration and death. *Salmonella* and *Shigella* organisms can frequently be cultured. Protozoal infections must be ruled out, but a number of these chronic digestive tract problems are either diet-related or non-specific and are frequently controlled with supportive therapy including fluid replacement and intestinal protectants. *Pseudomonas aeruginosa*, which may be responsive to the cautious use of gentamicin, is a frequent and often lethal opportunist in debilitated New World monkeys.

Herbes viruses, including *H. platyrrhinae* (formerly *Herpesvirus T.*) carried by squirrel monkeys (*Saimiri scirueus*) and *H. hominis* from cold sores in humans, can produce acute typical disseminated herpes virus infections in callitrichids (marmosets and tamarins). Death from these viruses can occur rapidly and oral ulcers may be seen (Figure 78). Lesions typical of disseminated herpes infections include hemorrhage and focal necrosis within many visceral organs (Figure 79). Squirrel monkeys, therefore, should never be mixed with callitrichids nor should Old World and New World species be allowed to come into contact with each other. Personnel with cold sores or other possible viral infections should not have contact with these animals either[19].

CONCLUSIONS

Zoological animal medicine is an ever-encompassing discipline that remains a continual challenge. The basic principles of veterinary medicine usually apply, but there is also a continual urgency to be innovative. There is much fertile ground for research and a great deal to be accomplished, particularly for those interested in comparative medicine.

AUTHOR AUTOBIOGRAPHIES

Mitchell Bush, D.V.M., was born in Santa Ana, California. He received a Bachelor of Science degree in pre-veterinary medicine in 1963 and the degree of Doctor of Veterinary Medicine in 1965, both from the University of California, Davis. He served an internship from 1965 to 1966 at Angell Memorial Animal Hospital, Boston, Massachusetts, where he remained as Clinical Veterinarian until 1967. He held appointments at Johns Hopkins University as Instructor in the Departments of Surgery and Animal Medicine from 1967 to 1969, and as Assistant Professor, Departments of Animal Medicine and Radiology, 1969 to 1972. Other appointments currently maintained include Assistant Professor, Departments of Radiology and Comparative Medicine, Johns Hopkins University, and Visiting Assistant Professor of Radiology, Vanderbilt University, Nashville, Tennessee. Dr. Bush served as Veterinarian at the Baltimore Zoo from 1967 until 1972, and then joined the staff at the National Zoological Park as Veterinarian. In 1975 he became head of the National Zoo's Office of Animal Health, the position he currently holds. His numerous scientific publications deal mainly with exotic animal medicine and surgery. Dr. Bush is a member of the American Veterinary Medical Association, American Animal Hospital Association, American Veterinary Radiology Society, Veterinary Orthopedic Society, and is an academy member of the American Association of Zoo Veterinarians.

Richard J. Montal, D.V.M., was born in New London, Connecticut, where he attended New London High School. He studied pre-veterinary medicine at the University of Connecticut and received the degree of Doctor of Veterinary Medicine from Cornell University in 1964. After spending three years in private veterinary practice, he received a U.S.P.H.S.-NIH post-doctoral fellowship to study comparative pathology at the Johns Hopkins University School of Medicine. He became a diplomate in the American College of Veterinary Pathologists and joined the medical faculty at Hopkins until 1975, at which time he became pathologist and head of the Office of Pathology for the National Zoological Park. Dr. Montali maintains part-time appointments as Assistant Professor of Pathology and Comparative Medicine at the Johns Hopkins University, and Assistant Clinical Professor of Pathology at the George Washington University School of Medicine. Among numerous professional organizations, he is a member of the International Academy of Pathology, the American Veterinary Medical Association, the American Association of Zoo Veterinarians, and the Wildlife Disease Association. He is also a member of Phi Kappa Phi and Phi Zeta, a veterinary honorary society. He has published numerous scientific papers, of which many deal with diseases of captive exotic animals.

REFERENCES

1. Appleby, E. C., and Siller, W. G.: Some Cases of Gout in Reptiles. J. of Path. and Bact., 30: 427-430, 1960. 2. Ardran, G. M., Kemp, F. H., Hovell, G. J. R., and Woods, C. G.: Cephalhematoma in Squirrel Monkeys. J. Am. Vet. Radiology Soc., 18: 70-75, 1977. 3. Baskin, G. B., Montali, R. J., Bush, M., Quan, T. J., and Smith, E.: Yersiniosis in Captive Exotic Mammals. JAVMA, 171: 908-912, 1977. 4. Boever, W. J., and Williams, J.: Arizona Septicemia in Three Boa Constrictors. Vet. Med./Small An. Clin., Vol. 70: 1357-1359, 1975. 5. Bush, M.: External Fixation of Avian Fractures, JAVMA, 171: 943-946, 1977. 6. Bush, M., Custer, R., Smeller, J., and Bush, L. M.: Acid-Base and Physiological Measures of Primates During Physical Restraint and Chemical Immobilization. JAVMA, 171: 866-869, 1977. 7. Bush, M., Hughes, J., Ensley, P. K., and James, A. E., Jr.: Fracture Repair in Exotics Using Internal Fixation. J. Am. An. Hosp. Assoc., 12: 746-753, 1976. 8. Bush, M., and James, A. E., Jr.: A New System of Casting Fractures in Exotic Animals. Vet. Med. and Sm. Ani. Clin., 71: 1288-1295, 1976. 9. Bush, M. Kennedy, S., Wildt, D. E., and Seager, S. W. J.: Sexing of Birds by Laparoscopy. Internat. Zoo. Yearbook in press, 1977. 10. Bush, M., Montali, R. J., Brownstein, D., James, E. A., Jr., and Appel, M. J. G.: Vaccine-Induced Canine Distemper in a Lesser Panda, JAVMA. 169: 959-960. 11. Bush, M., Montali, R. J., Novak, G. R., James, A. E., Jr.: The Healing of Avian Fractures: A Histological Xeroradiagraphic Study. J. Amer. Ani. Hosp. Assoc., 12: 768-773, 1976. 12. Bush, M., Montali, R. J., Smith, E. E., and Peratino, W. S.: Clinical Experience with Tuberculosis in Exotic Birds. In, Mycobacterial Infections of Zoo Animals. Edit. Montali, R. J., Smithsonian Institution Press, Washington, D. C. In press, 1977. 13. Bush, M., and Smeller, J. M.: Blood Collection and Injection Techniques in Snakes. Vet. Med./Small Anim. Clin. 73: 211-214, 1978. 14. Bush, M., Smeller, J. M., Charache, P., and Arthur R., Biologic Half-Life of Gentamicin in Gopher Snakes. Am. J. Vet. Res. 39: 171-173. 1978. 15. Bush, M., Smeller, J. M., Charache, P., and Solomon, H. M.: Preliminary Study of Antibiotics in Snakes. Proc. Amer. Assoc. Zoo Vet. PP 50-54. Edit. Boever, W. J. Published by Hill's Division, Riviana Foods, Topeka, 1976. 16. Bush, M., and Teeple, E.: Barbiturate Toxicity in Lions. J. Zoo An. Med., 6: 25, 1975. 17. Brownstein, D. G.: Reptilian Mycobacteriosis. In, Mycobacterial Infections of Zoo Animals. Edit. Montali, R. J.: Smithsonian Institution Press, Washington, D. C. In press, 1977. 18. Brownstein, D. G., Strandberg, J. D., Montali, R. J., Bush, R. M., and Fortner, J.: Cryptosporidia Associated Hypertrophic Gastritis in Snakes. Vet. Path. 14: 606-617, 1977. 19. Cicmanec, J. L.: Medical Problems Encountered in a Callithricid Colony. In, The Biology and Conservation of the Callithricid. Edit. Kleiman, D., Smithsonian Institution Press, Washington, D. C. In press, 1977. 20. Cowan, D.

F.: Diseases of Captive Reptiles. JAVMA, 153: 848-859, 1968. 21. Davis, J. W., Anderson, R. C., Karstad, L., and Trainer, D. O., Editors: Infectious and Parasitic Diseases of Wild Birds. Iowa State University Press, Ames, IA, 1971. 22. Donaldson, M., Heyneman, D., Dempster, R., and Garcia, L.: Epizootic of Fatal Amebiasis Among Exhibited Snakes: Epidemiologic, Pathologic, and Chemotherapeutic Considerations. An. J. Vet. Res. 36: 807-817, 1975. 23. Elkan, E., and Zwart, P.: The Occular Disease of Young Terrapins Caused by Vitamin A Deficiency. Path. Vet., 4: 201-222, 1967. 24. Fiennes, R., Editor, Pathology of Simian Primates, Part II, Infectious and Parasitic Diseases, S. Karger, New York, 1972. 25. Finlayson, R., Keymer, I. F., Manton, V. J. A.: Calcific Cardiomyopathy in Young Camels (Camelus spp.) J. Comp. Path., 81: 71-77, 1971. 26. Finnie, E. P.: Personal Communication, 1975. 27. Finnie, E. P.: Prophylaxis Against Coccidiosis in Kangaroos. Aust. Vet. J., 50: 276, 1974. 28. Fleischman, R. W., Squire, R. A., Sladen, W. J. L., and Melby, E. C., Jr.: Malaria (Plasmodium elongatum) in Captive African Penquins (Spheniscus demersus). JAVMA, 153: 928-935, 1968. 29. Flynn, R. J.: Parasites of Laboratory Animals. The Iowa State University Press, Ames, IA, 1973. 30. Gray, C.: Immunization of the Exotic Felidae for Panleukopenia. J. of Zoo An. Med., 3: 14-15, 1972. 31. Griner, L. A., Migaki, G., Penner, L. R., McKee, A. E., Jr.: Heterakidosis and Nodular Granulomas caused by Heterakis isolonche in the Ceca of Gallinaceous Birds. Vet. Path. 14: 582-590, 1977. 32. Griner, L. A.: Some Diseases of Zoo Animals. In, Advances in Veterinary Science and Comparative Medicine. Edit. Brandley, C. A., and Cornelius, C. E. Vol. 18, pp 251-270, Academic Press, New York, 1974. 33. Harthoorn, A. M.: The Chemical Capture of Animals. Bailliere Tindall. (MacMillan Publishing Co., New York), 1976. 34. Hasholt, J.: In, Diseases of Caged and Aviary Birds. Edit. Petrach, M. L., Lee and Feeger, Philadelphia, PA, 492-497, 1969. 35. Heller, R. M., Bush, M., Strandberg, J., James, A. E., Jr., and Smith, E.: Inflammatory Disease of the Temporal Bone of the Brush Tail Rat (Octodontomys gliroides): A Radiologic Clinical-Pathologic Correlation. J. Am. Vet. Radiol. Soc., 17: 161-164, 1976. 36. Hemboldt, C. F., and Wyand, D. S.: Parasitic Neoplasia in the Golden Pheasant. J. Wildl. Dis., 8: 3-6, 1972. 37. Jackson, C. G., and Jackson, M. M.: The Frequency of Salmonella and Arizona Microorganisms in Zoo Turtles. J. Wildl. Dis., 7: 130-132, 1971. 38. Kane, K. K., Corwin, R. M., and Boever, W. J.: Impaction due to Oxyuridae Infection in a Fiji Island Iguana. VM/SAC, 71: 183-184, 1976. 39. Kaplan, H. M.: Septicemic Cutaneous Ulcerative Disease of Turtles. Proc. Anim. Care Panel, 7: 273-277, 1957. 40. Kaplan, H. M.: Reptiles in Laboratory Animal Science. In, Handbook of Laboratory Animal Science, Edit. Melby, E. C., Jr., and Altman, N. H. CRC Press, Cleveland, 301-303, 1974.

41. Keane, C., Taylor, M. R. H., Wilson, P. Smith, L., Cunningham, B., Devine, P., English, L. F.: *Bacteriodes ruminicola* as a Possible Cause of "Lumpy Jaw" in Bennett's Wallabies. Vet. Microbiol., 2: 179-183, 1977. **42.** Kennedy, S., Montali, R. J., Smith, E. E., and Bush, M.: An Outbreak of Systemic Mycosis with Absidia Corymbifera in a Reindeer Herd. Proc. Amer. Assoc. Zoo Vet., pp 40-43. Edit. Boever, W. J. Published by Hill's Division, Riviana Foods, Topeka, 1976. **43.** Konyha, L. D.: Regulations Needed to Control Spread of Tuberculosis in Non-Domestic Animals. In, Mycobacterial Infections of Zoo Animals. Edit. Montali, R. J., Smithsonian Institution Press, Washington, D. C. In press, 1977. **44.** Landolt, M., and Kocan, R. M.: Transmission of Avian Pox from Starlings to Rothchild's Mynahs. J. Wildl. Dis., 12: 353-356, 1976. **45.** Lund, E. E., Chute, A. M., Wilkins, G. C.: The Wild Turkey as a Host for *Heterakis gallinarum* and *Histomonas meleagridis.* J. Wildl. Dis., 11: 376-381, 1975. **46.** Manwell, R. D., and Rossi, G. S.: Blood Protozoa of Imported Birds. J. Protozool., 22: 124-127, 1975. **47.** Marcus, L. E.: Parasitic Diseases of Captive Reptiles. In, Current Veterinary Therapy, 5: 632-638, Edit. Kirk, R. W., 1974. **48.** Mare, C. J.: Herpes Virus of Birds of Prey. J. of Zoo An. Med., 6: 6-11, 1975. **49.** McLaughlin, R. M., and Marrs, G. E.: Tuberculin Testing in Non-Human Primates, OT vs. PPD. In, Mycobacterial Infections of Zoo Animals. Edit. Montali, R. J., Smithsonian Institution Press, Washington, D. C. In press, 1977. **50.** Montali, R. J., Bush, M.: Avian Fracture Repair: Radiographic and Histopathologic Correlation. Proc. Amer. Assoc. Zoo Vet.: 150-153, 1975. **51.** Montali, R. J., Bush, M., and Greenwell, G. A.: An Epornitic of Duck Viral Enteritis in a Zoological Park, JAVMA, 169: 954-958, 1976. **52.** Montali, R. J., Bush, M., and Smeller, J.: The Pathology of Nephrotoxicity of Gentamicin in Snakes: A Model for Reptilian Gout. Vet. Path. Submitted 1977. **53.** Montali, R. J., Bush, M., Thoen, C. O., and Smith, E.: Tuberculosis in Captive Exotic Birds, JAVMA, 169: 920-927, 1976. **54.** Natt, M. P., and Herrick, C. A.: A New Blood Diluent for Counting Erythrocytes and Leukocytes of the Chicken. Poultry Science, 31: 735-738, 1952. **55.** Obwolo, M. J.: Yersiniosis in the Bristol Zoo. ACTA Zool. Pathol. Antverpte (Editum Consilio, Vanderbergh, W.), 64: 81-90, 1976. **56.** Pierson, G. T., and Pfow, C. J.: Newcastle Disease Surveillance in the United States. JAVMA, 169: 801-803, 1975. **57.** Rapley, W., Kelton, O. W., Scollard, N. D., Mehren, K. G., and Trillo, L. A. T.: Diagnosis and Control of Tuberculosis During the Establishment of a Modern Zoo. In, Mycobacterial Infections in Zoo Animals. Edit. Montali, R. J., Smithsonian Institution Press. Washington, D. C. In press, 1977. **58.** Richter, A. G., Olsen, J., Fletcher, K., Benirschke, K., and Bogart, M.: Techniques for Collecting Blood from Galapagos Tortoises and Bay Turtles. VM/SAC, 72: 1376, 1977. **59.** Schacter, J., Sugg, N., and Sung, M.: Psitticosis: The Reservoir Persists. J. of Infect. Dis. 137: 44-49, 1978. **60.** Schmidt, J. J.: A Preliminary Report on the Use of Rompun in Captive Asian Elephants. J. of Zoo An. Med., 6: 13-21, 1975. **61.** Seal, U. S., and Erickson, A. W.: Immobilization of Carnivora and Other Mammals with Phencyclidine and Promazine. Fed. Proc. 28: 1410-1419, 1969. **62.** Smeller, J. M., Bush, M., and Custer, R. W.: The Immobilization of Marsupials. J. of Zoo An. Med., 8: 16-19, 1977. **63.** Thoen, C. O.: Isolation and Identification of Mycobacteria from Tissues of Exotic Animals. In, Mycobacterial Diseases of Zoo Animals. Edit. Montali, R. J., Smithsonian Institution Press, Washington, D. C. In press, 1977. **64.** Thoen, C. O., Bush, M., and Montali, R. J.: An Enzyme-Labeled Antibody Test for Detecting Antibodies in Tuberculous Chickens and Exotic Birds. Avian Diseases, 22: (Jan-March), 1978. **65.** Wallach, J. D.: Environmental and Nutritional Diseases of Captive Reptiles. JAVMA, 159: 1632-1643, 1971. **66.** Wallach, J. D., and Flieg, G. M.: Nutritional Secondary Hyperparathyroidism in Captive Birds. JAVMA, 155: 1046-1051, 1969. **67.** Ward, F. P.: Thiamine Deficiency in a Peregrine Falcon. JAVMA, 159: 599-601, 1971.