Differential Diagnoses by Clinical Signs—Crocodilians

Javier G. Nevarez

STRESS AND IMMUNOSUPPRESSION

Stress and immunosuppression are often underlying factors for many diseases affecting captive crocodilians. Stress has been defined as "a physiological answer to a perceived threat that includes, but is not restricted to, increased adrenal secretion."[1] Stress is also thought of as any event that challenges homeostasis, and likely the response to that challenge involves more than an adrenal response. The autonomic nervous system, the hypothalamic adrenal axis, neuropeptides, neurotransmitters, and neuroimmunologic mediators all have a role in the response of the immune system to stress.[2] Studies in crocodilians have evaluated the stress associated with restraint, long-term corticosterone implants, cold shock, and stocking densities.[3–6] Lance et al. provide an overview of the physiology and endocrinology of stress in crocodilians.[7] Catecholamines, glucocorticoids, glucose, and lactate have been implicated in the stress response of crocodilians. In addition, an argument is made for immunosuppression on the basis of changes observed in the white blood cells.[3–5,7] Factors that influence stress in crocodilian and reptile species are reviewed by Rooney and Guillette.[1] Enough evidence exists to suggest that stress plays an important role in the physiology of crocodilians, and it may indeed predispose them to illness. Overcrowding, handling, excessive noise, diet changes, water and air quality, temperature irregularities, and more should all be considered as predisposing or confounding factors of disease.

SYSTEMIC SIGNS

Anorexia, Lethargy, and Acute Mortalities

The first signs of illness in captive crocodilians are usually nonspecific in nature. These include anorexia, lethargy, and death. In commercial operations, the workers may notice excess food remaining from the day before, which is sometimes followed by a perceived change in the behavior of the animals. One should not underestimate these observations because most workers are well tuned to the daily routine and behavior of the animals. A visit to the ranch or farm should be performed during feeding time to avoid additional stress to the animals. This also allows observation of the feeding and water change practices. At this time, a thorough history and a collection of animals for diagnostics and necropsy can be obtained. In addition to routine samples, tissues should be frozen for possible bacterial, fungal, or viral cultures.

The list of differentials for nonspecific clinical signs must be narrowed after diagnostic results are obtained. However, one must remember that husbandry and disease go hand in hand. Most systemic diseases of captive crocodilians are thought to be secondary in nature. Bacterial and fungal diseases are commonly encountered. The majority of data available on bacterial disease in crocodilians are from studies involving the American alligator (*Alligator mississippiensis*). Table 136.1 presents

a large number of isolates from diseased and nondiseased animals, including a survey of fecal coliforms.[8] Huchzermeyer et al. also have extensive data on enteric bacterial and fungal isolates from African Dwarf Crocodiles (Osteolaemus tetraspis).[9] Important bacterial infections reported in crocodilians include *Mycoplasma alligatoris*,[10] *Mycoplasma crocodyli*,[11] and *Chlamydia* spp.

Fungal organisms are often opportunistic invaders of the integument and respiratory system, but primary fungal infections are also reported. Viral diseases are likely underdiagnosed in reptile medicine because of the challenges in diagnostic techniques and scarce information. Poxvirus, West Nile virus (WNV), and herpesvirus have been reported as pathogens in crocodilians. An adenovirus-like infection in captive Nile Crocodiles (*Crocodylus niloticus*) has also been reported.[12] Coronavirus, influenza C virus, and paramyxovirus have been identified with transmission electron microscope in the feces of crocodilians, but their significance remains to be determined. Finally, evidence exists of seroconversion to paramyxovirus and eastern equine encephalitis virus in crocodilians.[13]

Toxicities are not common in captive crocodilians. Lead toxicity is reported from alligators fed lead-shot nutria. Clinical signs include weakness, lethargy, anorexia, and death. Feeding of nutria is no longer common practice.

For privately owned and zoo animals, there are additional differentials to be considered. A faulty or broken heater can lead to sudden death from electrocution. This can also present a risk for humans, therefore, it is important to isolate, protect, and inspect all equipment within the enclosure. A thermostat malfunction can lead to altered water temperatures that can affect the animal's behavior. In colder climates, animals may become lethargic if the heater is not working properly. Conversely, if a thermostat fails, allowing the heater to remain on, the animals' behavior may change because of increased temperatures. At first they may appear agitated but eventually anorexia and lethargy will occur if overheated. Overheating may also occur as a result of inappropriately sized or placed heat lamps.

Foreign bodies should be a differential for anorexic crocodilians, especially those in outdoor exhibits where trash, coins, and other objects may be tossed in by the public. It is also important to pick up and secure all tools such as buckets and hoses, which may also be ingested by animals.

Physiological anorexia can be observed as part of the normal reproductive cycle, in particular during the later stages of egg development. However, dystocia (egg binding) can lead to a more prolonged anorexia that may be the first indication that the animal is ill. Unless the actual mating date is known or the reproductive cycle is monitored by ultrasonography, it can be very difficult to ascertain what constitutes true dystocia. In animals housed outdoors in their natural climate, dystocia should be considered if oviposition has not occurred for 30 days past the expected date. For those housed indoors or outside their

TABLE 136.1 Bacteria Isolated From *Alligator Mississippiensis* With and Without Signs of Disease

Isolate	Tissue	Clinical Signs/ Lesions	Isolate	Tissue	Clinical Signs/ Lesions
Aeromonas hydrophila	Blood[34]	Yes	*Klebsiella oxytoca*	Skin[55]	Yes
	Lungs, heart, liver, kidneys, intestines, oral cavity[49]	Yes, No		Oral cavity[53]	No
			Klebsiella sp.	Lungs[25]	Yes
	Lungs, blood[50]	Yes	*Klebsiella pneumonia*	Cloaca[8]	No
	Eye[51, 52]	Yes	*Micrococcus kristinae*	Blood[34]	Yes
	Oral cavity, water[53]	No	*Moraxella* sp.	Oral cavity, water[53]	No
	Cloaca[8]	No	*Morganella morganii*	Blood[55]	Yes
Aeromonas sp.	Lungs[10]	Yes		Oral cavity[53]	No
Acinetobacter calcoaceticus	Oral cavity[53]	No		Lung[10]	Yes
Aerobacter radiobacter	Oral cavity[53]	No		Cloaca[8]	No
Bacteroides asaccharolyticus	Oral cavity[53]	No	*Mycoplasma alligatoris*	Multiple tissues[34]	Yes
Bacteroides bivius	Oral cavity, water[53]	No	*Pantoea* spp.	Cloaca[8]	No
Bacteroides loescheii/denticola	Oral cavity, water[53]	No	*Pasteurella haemolytica*	Oral cavity[53]	No
Bacteroides oralis	Oral cavity[53]	No	*Pasteurella multocida*	Lungs[56]	Yes
Bacteroides sordellii	Oral cavity[53]	No	*Pasteurella pneumotropica*	Cloaca[8]	No
Bacteroides thetaiotamicron	Oral cavity[53]	No	*Pasteurella* sp.	Oral cavity, water[53]	No
Bacteroides vulgatus	Oral cavity[53]	No	*Peptococcus magnus*	Oral cavity[53]	No
Bacteroides sp.	Water[53]	No	*Peptococcus prevotii*	Oral cavity[53]	No
Citrobacter freundii	Blood[55]	Yes	*Pleisomonas shigelloides*	Cloaca[8]	No
	Oral cavity[53]	No	*Proteus mirabilis*	Blood[34]	Yes
	cloaca[8]	No	*Proteus vulgaris*	Oral cavity[53]	No
Citrobacter braakii	Cloaca[8]	No		Oviduct[54]	Yes
Clostridium bifermentans	Oral cavity, water[53]	No		Blood[34]	Yes
	Lungs[10]	Yes		Lung[10]	Yes
Clostridium clostridioforme	Oral cavity[53]	No	*Proteus* sp.	Blood[55]	Yes
Clostridium innoculum	Water[53]	No	*Providencia alcalifaciens*	Cloaca[8]	No
Clostridium limosum	Oral cavity[53]	No	*Providencia rettgeri*	Cloaca[8]	No
Clostridium sordellii	Oral cavity, water[53]	No	*Pseudomonas cepacia*	Oral cavity[53]	No
Clostridium sporogenes	Blood[10]	Yes	*Pseudomonas diminuta*	Water[53]	No
Clostridium tetani	Oral cavity[53]	No	*Pseudomonas fluorescens*	Water[53]	No
Clostridium sp.	Blood[10]	Yes	*Pseudomonas pickettii*	Oral cavity[53]	No
Corynebacterium sp.	Tail abscess[50]	Yes	*Pseudomonas vesicularis*	Water[53]	No
Diphtheroid sp.	Oral cavity[53]	No	*Pseudomonas* sp.	Lungs, pharynx[50]	Yes
Edwardsiella tarda	Kidney, feces[54]	Yes		Water[53]	No
	Fat body, pericardial fluid[10]	Yes	*Salmonella typhimurium*	Gastrointestinal tract[50]	Yes
	Cloaca[8]	No	*Salmonella braenderup, anatum Arizona* spp.	Cloaca[50]	No
Edwardsiella aerogenes	Cloaca[8]	No	*Salmonella* sp. (subgroup III)	Lung[57]	Yes
Enterobacter aggiomerans	Blood[55]	Yes	*Serratia marcescens*	Skin[55]	Yes
Enterobacter cloacae	Oral cavity, water[53]	No		Cloaca[8]	No
	Cloaca[8]	No	*Serratia odorifera*	Oral cavity[53]	No
Enterobacillus sp.	Lungs[50]	Yes	*Staphylococcus aureus*	Lungs[56]	Yes
Escherichia coli	Cloaca[8]	No	*Staphylococcus cohnii*	Blood[34]	Yes
Fusobacterium nucleatum	Oral cavity[53]	No	*Streptococcus* sp., hemolytic	Lungs[10]	Yes
Fusobacterium varium	Oral cavity[53]	No	*Vibrio parahaemolyticus*	Blood[34]	Yes
Hafnia alvei	Cloaca[8]	No	*Vibrio cholerae, putative*	Cloaca[8]	No
			Vibrio fluvialis	Cloaca[8]	No

natural climate, behavior together with plasma biochemistry, hematology, and diagnostic imaging can be used to aid in the diagnosis of dystocia.

Secondary nutritional hyperparathyroidism can also cause animals to be lethargic and anorexic. This is more common in juvenile animals. Older animals may succumb to secondary renal hyperparathyroidism.

Poor Growth and Runting

Another phenomenon seen in captive crocodilian operations is runting, the lack of growth and failure to thrive of some animals within a group. In some operations, animals are separated by size and some buildings contain only the runts. A clear size difference is observed in same-age animals between the runts and the otherwise healthy ones. These animals

are not as hardy in the captive environment and are potentially more susceptible to disease. Dominance by other animals, environment, and even incubation factors[14] contribute to the presence of runts.

DERMATOLOGICAL SIGNS

Integumentary disease is often secondary to poor water quality, poor enclosure design, stress, and immunosuppression or a combination of these factors. Lacerations, abscesses, and draining tracts can all be observed in captive and wild crocodilians. Consequently, these open wounds can serve as a nidus for bacteria and fungi. An additional factor in commercial operations or small enclosures is the accumulation of a fatty slime layer on the surface of the water when meat supplements, in particular chicken, are fed in addition to a dry commercial diet. This slime attaches to the skin of the animals and the enclosure, creating an environment for bacterial and fungal growth. The same is true for accumulation of biofilm on the surface of the enclosure. A soap or detergent can be used as a surfactant to help reduce the fatty layer on the water surface. Any product used must be nontoxic, or the animals must be removed from the enclosure while in use. Fungal dermatitis is also common because many fungi thrive in the water column and environment of captive alligators. In most instances, both bacteria and fungi are present in skin lesions. Culture and sensitivity testing can be frustrating in these cases because of the mixed flora found in the lesions. The first step in addressing integumentary abnormalities is to analyze water quality and make improvements as needed. In addition to improvements in water quality, antimicrobial or antifungal therapy may be necessary. Medicating a large group of crocodilians is challenging because it must usually be done orally by mixing the medication with feed. In zoos or private collections, individual crocodilians can be medicated by injecting the prey item or via injection. A pole syringe can be used in large or aggressive animals. There is a lack of pharmacokinetic data for oral antimicrobials in crocodilians, and it has been shown that oral tetracycline is not effectively absorbed in American alligators.[15] These factors pose a challenge for effective treatment and the potential for promoting antimicrobial resistance. Therefore a combination of good hygiene, water treatments with disinfectants, and improved husbandry is recommended.

A number of parasites are known to affect the skin of crocodilians. *Paratrichosoma* spp. are capillaroid parasites that cause a zigzag lesion on the skin. These lesions appear to be purely cosmetic. This parasite is known to affect various crocodile species in the wild and is believed to have a stage of its lifecycle dependent on soil; therefore, it is not observed in captive scenarios where animals are kept on concrete.[13] Regardless of the etiology, good hygiene is essential in the prevention and treatment of integumentary disease in crocodilians. The etiology itself is at times not as important as what conditions predisposed them to the disease. A herpesvirus was identified via TEM from saltwater crocodiles *(Crocodylus porosus)* with concurrent pox virus and bacterial infection of the skin.[46]

Dermatophilosis (Brown Spot Disease)

Dermatophilosis causes brown-red to ulcerative lesions most commonly located between scales on the ventral skin (Fig. 136.1). Most of the cultures appear to resemble *Dermatophilus congolensis*.[16,17] These filamentous bacteria do not respond well to antibiotic therapy; therefore, intensive hygiene practices are necessary to prevent and control outbreaks.

Pox Virus

Parapoxvirus or poxlike viruses have been identified in five different crocodilian species: Spectacled caiman (*Caiman crocodilus fuscus*),[18,19] Brazilian caiman (*Caiman crocodilus yacare*),[20] Nile crocodile,[21,22] saltwater
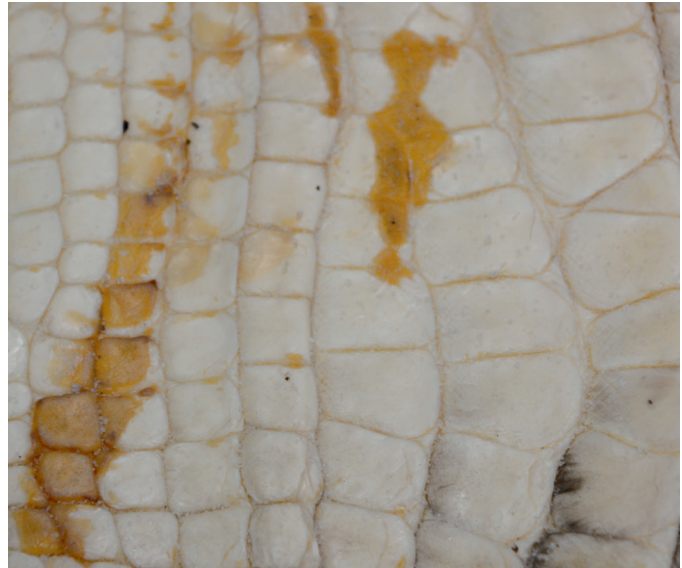

FIG 136.1 Brown lesions typical of dermatophilosis on the ventral skin of an American alligator (*A. mississippiensis*). Lesions usually arise at the junction of the scales and then spread outward. (Courtesy of Javier G. Nevarez.)

crocodile (*Crocodylus porosus*),[23] and freshwater crocodile (*Crocodylus johnstoni*).[23] In caimans, poxvirus is characterized by 1- to 3-mm-diameter, gray to white, coalescing to macular skin lesions. These can be found on the head, palpebrae, maxilla, mandible, limbs, palate, tongue, and gingiva.[18–20] Other signs observed include palpebral and generalized edema. Resolution of clinical signs was observed 6 weeks after improvement of husbandry in one case[19] and after 5 months in another case, with no changes in husbandry.[20]

Lesions in crocodiles are described as 2- to 8-mm-diameter, yellow to brown, wartlike, sometimes firm, and unraised to raised nodules with occasional shallow ulcers. These lesions can be found on the head, palpebrae, nostrils, sides of the mouth, oral cavity, limbs, ventral neck, and coelom and at the root of the tail.[21,22] Resolution of lesions was reported to occur as early as 3 to 4 weeks.[21] Light microscopy reveals epithelial hyperplasia, acanthosis, hyperkeratosis, and necrosis, and at times Borrel and Bollinger's bodies are also visible.[18–22] Secondary bacterial and fungal infections may also be present. Horner reported the use of an autogenous vaccine to treat poxvirus in Nile crocodiles.[21] No specific treatment recommendations exist. Maintaining appropriate husbandry is essential in the prevention and resolution of poxvirus in crocodilian farms.

West Nile Virus

West Nile virus (WNV) has been reported to cause lymphohistiocytic proliferative syndrome of alligators (LPSA).[24,25] LPSA skin lesions are a chronic manifestation of WNV infection and occur in animals that have survived a WNV outbreak.[25] This production problem affects the quality of the hide and consequently decreases profit for alligator ranchers. Gross lesions can be seen as multifocal, 1 mm to 2 mm, gray to red foci on the ventral mandibular, abdominal, and sometimes tail scales (Fig. 136.2). They can be found on any section of a scale and do not appear concave or convex with respect to the scale's surface. Routine microscopic examination reveals dermal nodular lymphoid proliferation with perivascular cuffing. Similar lesions can be found in other tissues besides the skin. Identification of the lesions is complicated by the fact they are sometimes not seen until the hides are removed from the animal. Therefore antemortem identification alone is not very accurate.
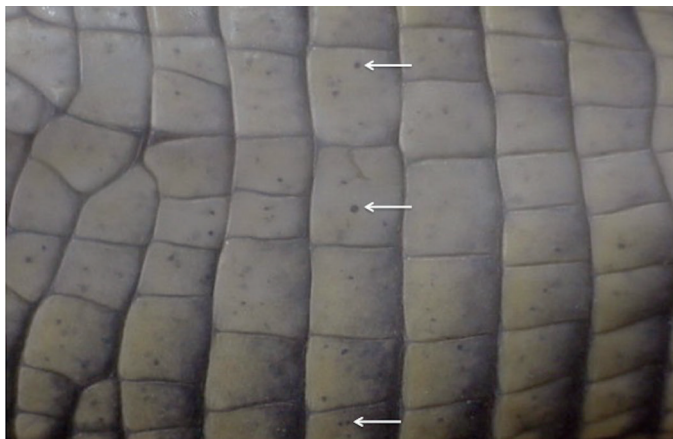
FIG 136.2 Gross lesions (arrows) of lymphohistiocytic proliferative syndrome of alligators on the ventral skin of an American alligator (*A. mississippiensis*). Lesions appear in animals that survived WNV infection. There are over 50 lesions in this image. (Courtesy of Javier G. Nevarez.)

FIG 136.3 Young alligator (*Alligator mississippiensis*) with the head tilt typical of those affected with West Nile virus. Notice that it still maintains an aggressive stand with the mouth open.

NEUROLOGICAL SIGNS

Neurological deficits are not commonly seen in crocodilians and deserve special attention if encountered. Clinical signs may include swimming in circles or on one side of the body, abnormal flotation, lethargy, ataxia, head tilt, and muscle tremors. Anorexia often accompanies the neurological signs. Thiamine deficiencies should be considered in animals fed a frozen fish diet. Signs of thiamine deficiency typically include severe lethargy that leads to coma.

Hypoglycemia has also been reported as a cause of neurological signs in alligators.[26] These animals have muscle tremors, loss of the righting reflex, and mydriasis. Stress seems to be the main contributing factor. The author has also observed neurological signs associated with low oxygen levels inside alligator enclosures. Neurological signs may also be observed after ingestion of metal foreign bodies, including coins, containing lead or zinc.

West Nile Virus

West Nile virus causes neurological signs in captive-reared American alligators raised indoors.[27,28] Presence of antibodies, but no clinical signs, is reported from free-ranging and captive crocodilians housed outdoors.[29,30] WNV in crocodilians is transmitted by mosquitoes and horizontally via fecal shedding. This represents an opportunity for zoonosis in captive operations. Strict building quarantine and hygiene strategies should be implemented to prevent spread to other animals in the facility. WNV is a reportable disease, and one should contact the state veterinarian when diagnosis is confirmed.

WNV can affect an animal of any age, but hatchlings often experience severe per-acute mortalities of up to 50% to 60%.[28] WNV has two disease stages in alligators, acute and chronic. Clinical signs during the acute stage include swimming in circles, head tilt (Fig. 136.3), muscle tremors, weakness, lethargy, anorexia, and death. Animals that survive the acute infection go on to develop LPSA skin lesions, the chronic stage of WNV. There is no treatment. Definitive diagnosis is accomplished with reverse transcriptase polymerase chain reaction (RT-PCR) of liver and brain. However, it must be noted that RT-PCR can detect viral RNA from a commercially available WNV vaccine for alligators (BIVI, St. Joseph, MO). Therefore vaccine status must be known before diagnosis. Maternal transfer of antibodies has also been observed in hatchlings.[31] Veterinarians should take additional precautions during necropsies of suspect animals. Aggressive mosquito control and vaccination have

proven extremely effective at controlling WNV infection in commercial operations.

RESPIRATORY SIGNS

This is one of the most common presentations of captive crocodilians, second only to integumentary disease. Respiratory signs can often be confused with neurological signs, and a clear distinction must be established. However, respiratory disease may accompany neurological disease as a consequence of weakness leading to aspiration. Clinical signs associated with respiratory disease in crocodilians may include dyspnea, tachypnea, nasal discharge, excessive basking, abnormal swimming (either in circles or on one side of the body), and anorexia, among others. As the animals become weak, the basihyoid valve does not function properly and they can aspirate food or water. The pharyngeal anatomy of crocodilians makes aspiration unlikely in healthy animals. Therefore, if aspiration is suspected, evaluation for an underlying illness should be pursued. A number of rhinitis and pharyngitis syndromes have also been described in some crocodilian species.[13] Most respiratory infections are either bacterial or fungal in origin. An early report showed a fungal pneumonia associated with *Beauveria bassiana* in two alligators,[32] and *Fusarium moniliforme* was found in another case.[33]

Mycoplasmosis

One well-documented respiratory pathogen is *Mycoplasma alligatoris*. Clinical signs are nonspecific and include lethargy, weakness, anorexia, white ocular discharge, paresis, and edema (facial, periocular, cervical, limbs).[10] Pneumonia, pericarditis, and polyarthritis are often diagnosed on necropsy. Pathogenicity of *M. alligatoris* has been documented for *A. mississippiensis* and for the broad-nosed caiman (*Caiman latirostris*).[34,35] Other crocodilian species closely related to alligators are also potentially susceptible. Helmick et al. reviewed antimicrobial susceptibility for *M. alligatoris*.[36] A second *Mycoplasma* species, *M. crocodyli*, is known to affect Nile crocodiles.[11] Lesions are similar to those observed with *M. alligatoris*. Some studies have examined the use of an autogenous vaccine for *M. crocodyli*, but work is still needed to determine its true efficacy.[37,38] Currently antemortem diagnosis is difficult and requires a combination of serology and tissue biopsy for PCR. Therefore most cases are confirmed via histopathology and PCR postmortem.

Mycobacteriosis

Although not commonly reported from crocodilians, the author has worked on a number of alligator cases with acid-fast organisms consistent with *Mycobacterium* sp. in the lungs. These animals had evidence of pneumonia on necropsy as shown by multiple white foci, 1 to 4 mm in diameter, on the lung parenchyma. Other cases of pulmonary and enteric mycobacterial infections have been suggested.[39] Difficulties of growing *Mycobacterium* sp. make its definitive diagnosis a challenge.

MUSCULOSKELETAL SIGNS

Musculoskeletal disease can be the result of alterations in the incubation temperature or environment (Fig. 136.4) or trauma from fighting (Figs. 136.5), transport, restraint, neoplasia, or secondary to nutritional disease.

FIG 136.4 Congenital abnormality in an American alligator (*Alligator mississippiensis*). This animal was hatched with this deformity and is able to eat and thrive among others in the group.

FIG 136.5 Mandibular fracture in a wild alligator (*A. mississippiensis*). This type of lesion is not uncommon in wild animals, particulary in males during breeding season.

Limb fractures and partial amputations can be seen after altercations. It is possible for animals that have lost limbs after a fight to survive to the point of complete healing (Fig. 136.6). These lesions however may also lead to nerve or muscle damage and consequent paresis or paralysis. As mentioned in the respiratory signs section, *M. alligatoris* and *M. crocodyli* cause polyarthritis, which can be evident antemortem.

Nutritional secondary hyperparathyroidism can occur in young crocodilians not being fed whole prey or pelleted diets. Affected animals may also be runts of the group and have concurrent disease. The innate requirement of ultraviolet B light for the production of vitamin D_3 in crocodilians is not known. They appear to be able to obtain appropriate levels from their diet, but further research in this area is needed.

Gout also occurs in crocodilians (Figs. 136.7A and B). Clinical signs can be nonspecific in nature, but limb paresis/paralysis and joint enlargement may be observed. A case of gout with concurrent suspected hypovitaminosis A in crocodile hatchlings has been reported.[40]

Deficiencies of vitamins A, B, C, or E can also lead to a variety of musculoskeletal disorders. These are not commonly seen when commercial diets are fed in addition to meat products.

GASTROINTESTINAL SIGNS

Anorexia is likely the most common clinical sign associated with gastrointestinal disease in captive crocodilians. Foreign body ingestion, gastric ulcers, enteritis (Fig. 136.8), and trauma to the oral cavity can be observed in crocodilians. Ingestion of foreign bodies is more common in wild crocodilians. However, it must also be a differential in captive specimens. Malfunction of water pumps, water filters, or construction can lead to the presence of foreign bodies in the enclosures. In outdoor exhibits, the public is often responsible for the presence of foreign objects that are thrown into their enclosures. These can then be ingested by the animals and cause severe problems in the case of nails and other sharp objects (Fig. 136.9).

Infectious enteritis may be difficult to assess grossly because crocodilians have very thick intestinal walls normally. However, crocodilians appear to have a characteristic response to insult of the gastrointestinal tract. Accumulations of fibrous material or necrotizing lesions are commonly seen on necropsy (Fig. 136.10A and B). This reaction is aggressive and can even lead to obstructions caused by the fibrous material. Obstruction can also occur with fecal impactions and torsions. Gastric ulcerations and abnormalities of the gastric mucosa are also routinely observed on necropsy and may be associated with stress and diet. Finally, the intestinal tract contains a large amount of Peyer's
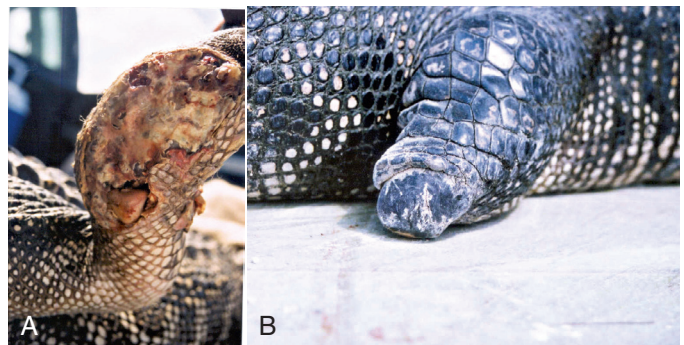
FIG 136.6 (A) Rear limb open fracture with tissue necrosis in a wild alligator (*A. mississippiensis*). Wild animals can recover from these types of lesions, but they can lead to severe septicemia in captive ones. (B) Wild alligator with a healed lesion that resulted in amputation of the distal rear limb.

FIG 136.7 (A and B) Renal and visceral gout in an American alligator (*A. mississippiensis*). Oversupplementation with a vitamin and mineral mix was suspected in this case. (Courtesy of Javier G. Nevarez.)
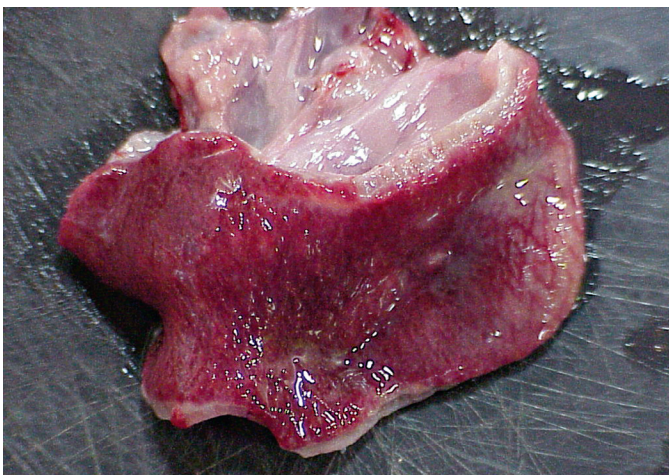
FIG 136.8 Enteritis in a captive-reared alligator (*A. mississippiensis*). Notice the red mucosa.

FIG 136.9 Farmed alligator (*A. mississippiensis*) with a nail foreign body that was ingested and perforated its stomach. (Courtesy of Javier G. Nevarez.)
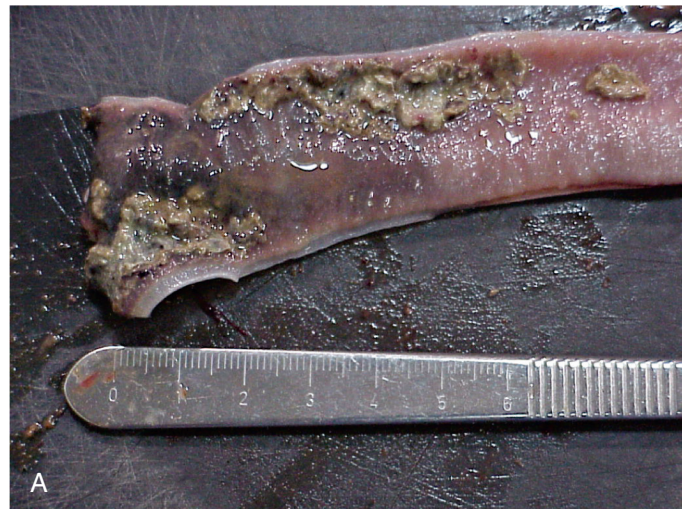
FIG 136.10 (A and B) Fibrotic membrane on the intestinal mucosa of a captive-reared alligator (*A. mississippiensis*). This was an incidental finding for this animal.

patches and likely represents a site of aggressive inflammatory response to infectious agents. A novel herpesvirus was reported from an American alligator with lymphoid follicular inflammation of the cloaca.[45]

OCULAR SIGNS

Chlamydia

Chlamydiosis has been reported from *C. niloticus* (South Africa and Zimbabwe*), C. porosus* (Papua New Guinea), and *C. siamensis* (Thailand).[41-43] The author has also diagnosed Chlamydiosis in *A. mississippiensis* (United States). Although it has been proposed as a *Chlamydophila psittaci*-like strain in *C. niloticus*,[44] the *Chlamydia* species affecting crocodilians is likely a novel specie(s). Infection usually leads to hepatitis and/or conjunctivitis (Fig. 136.11), but lung and spleen can also be infected. Morbidity and mortality can exceed 50%, resulting in significant economic losses for farmed animals.[42] Treatment with oxytetracycline is anecdotally reported to be effective in crocodiles. This creates significant concerns for possible antimicrobial resistance, zoonosis, and the safety of the meat from these animals.

MISCELLANEOUS

A novel herpesvirus was reported from an American alligator with lymphoid follicular inflammation of the cloaca.[45] A herpesvirus was identified via TEM from saltwater crocodiles (*Crocodylus porosus*) with concurrent pox virus and bacterial infection of the skin.[46] In captive *C. porosus* and freshwater crocodiles (*C. johnstoni*) from Australia, three novel herpesviruses have been identfied.[47] *C. porosus* exhibited conjunctivitis-pharyngitis, systemic lymphoid proliferation and encephalitis, or lymphonodular skin lesions.[47,48] *C. johnstoni* had
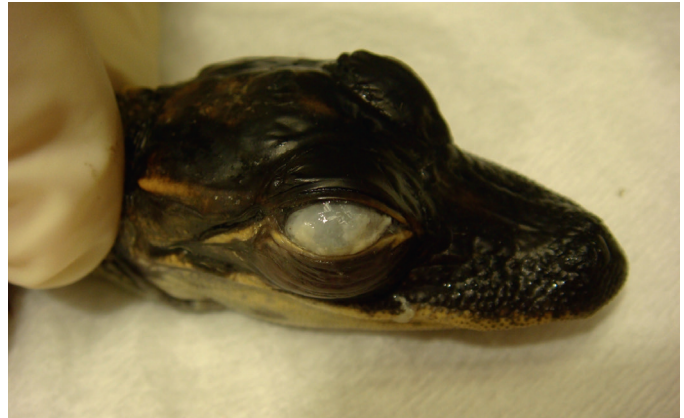
FIG 136.11 Keratitis and conjunctivitis because of chlamydiosis in a hatchling American alligator (*A. mississippiensis*). (Courtesy of Javier G. Nevarez.)

systemic lymphoid proliferation.[47] Chlamydiaceae was also identified from conjunctival and pharyngeal tissues of some of the *C. porosus* cases.[48] Of interest is that these syndromes occur concurrently with other infectious agents and have been associated with significant morbidity and mortality of infected animals.

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See www.expertconsult.com for a complete list of references.

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