Abstract

Birds of prey have developed talons, a hooked beak, and a tongue and oral cavity replete with pronounced hooks and papillae for prehending large boluses of food. The size of the proventriculus in relation to the ventriculus and weak musculature of the ventriculus, combined with an extremely acidic luminal pH, are consistent with a gastric digestive physiology maximized for protein digestion. The pyloric sphincter retains indigestible matter in the stomach, which is later compressed into a pellet and egested. The ventriculus, pylorus, pancreas, and an elongated duodenum coordinate to maximize neutralization of acidic peptic juices and increase the efficiency of digestion and absorption. Raptors are susceptible to a variety of infectious and noninfectious diseases that affect the digestive tract. Diagnostic testing and treatment recommendations for raptor intestinal disease conditions are discussed in this article. Copyright 2010 Elsevier Inc. All rights reserved.

Key words: bacteria; gastrointestinal; fungal; raptor; parasites; viral

Raptors serve an important role in the ecosystems in which they are found. These animals are built to function in the top of the food web, and as such have developed a gastrointestinal tract suitable for a carnivorous diet (Fig 1).

Anatomy and Physiology

Beak and oral cavity. The raptor beak is hooked and the tomia are very sharp over the rostral one half to two thirds of its length. The pliability of the beak commissures allows for insertion of fingers into the beak for an oral examination or when administering medications. In most birds of prey, the opening and closing strength of the beak is not significant because the leg and neck muscles are primarily used for capturing and ripping the flesh of their prey. The maxilla is hooked into the flesh of the prey item and then pulled vertically to shred off boluses for swallowing. The palatine ridges, choanal borders, caudal borders of the tongue, and the glottal mound have caudal-projecting epithelial projections that direct food into the esophagus (Fig 2). The mobility of a raptor’s tongue is greater than that found in parrot species, in that the entire glottis can be projected beyond the oral commissure. Extreme tongue mobility in raptor species may help prevent suffocation when swallowing large food boluses. The epithelial projections (hooks) located on the caudal border of the tongue can be utilized together with the lower beak to secure an endotracheal tube and prevent retraction of the tongue and the incidence of spontaneous extubation. The rostral portion of the tongue is generally thickened and keratinaceous. Because of the anatomic and physiologic composition of the raptor tongue, application of a pulse oximetry transducer at this location is not recommended; however, the choanal entrance is still a viable choice for a reflectance probe.

From the Avian Specialty Veterinary Services of Alaska, Bremerton, WA USA.

Address correspondence to: Scott Ford, DVM, Dip. ABVP (Avian), Avian Specialty Veterinary Services of Alaska, 13861 Hillcrest Dr. NW, Poulsbo, WA 98370. E-mail: vet@alaskabirddoc.com.

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Ingluvies

All birds of prey possess an ingluvies (crop) except Strigiformes (e.g., owls).1,2 When distended, the crop rests against the cranial edge of the clavicle. The raptor crop is fusiform-shaped and has a poorly developed lower sphincter into the thoracic esophagus. Because a raptor may not be successful during each hunting attempt and the size of prey may be variable, the crop allows the bird to consume large volumes of food as quickly as possible. The speed of consumption is particularly important with smaller birds of prey, which can be killed by other raptors and piracy of hard-won prey is also avoided. The crop also allows slower, more complete digestion at a resting location of the bird’s choosing.3

Proventriculus and Ventriculus

The raptor proventriculus is consistently more developed than the ventriculus when compared with Psittaciformes or other granivorous birds (Fig 3). The alternating thick and thin muscle areas noted in the ventriculus of other birds are absent in raptors because most animal protein requires relatively little mechanical digestion but rather requires extensive autoenzymatic digestion.2 Two types of glands exist in the wall of the proventriculus: (1) tubular glands for the secretion of mucus and (2) gastric glands that secrete hydrochloric acid and pepsinogen.3 Pepsinogen is activated to pepsin by hydrochloric acid or previously activated pepsin and has the ability to break down a wide range of tertiary structures of protein, exposing peptide bonds for additional digestion. The stomach pH of Falconiformes averages approximately 1.6 to 1.7 and in Strigiformes 2.4.2,3 The pellets of Strigiformes often contain incompletely digested remains (e.g., bones) and are the most likely the result of a higher stomach pH.2 In contrast to most florivorous birds, the ventriculus is lacking muscular development, has a relatively large lumen, and is more pliable. In addition, the proventricular-ventricular isthmus is not present so that the 2 organs form 1 large pear-shaped cavity in many birds of prey. This lack of an isthmus and shape of the raptor stomach allow space for larger pieces of prey and...
decrease the time required for preparing food for swallowing.

**Casting.** Formation of gastric castings is not unique to birds of prey. It is also a daily activity in corvids and some other Passeriformes and gulls, depending on dietary intake. However, casting activity has been primarily investigated in birds of prey. Most species of birds do not possess enzymes that can break down the protein keratin, a common constituent of nails, hair, feathers, and scales. After digestion of a meal, the contractions of the ventriculus increase in amplitude for about 12 minutes before pellet egestion, compacting the pellet and directing it to the cranial aspect of the proventriculus where it is then egested by esophageal retroperistalsis in about 8 to 10 seconds. The meal-to-pellet interval is variable. Egestion of the pellet usually occurs in the morning unless food is ingested late the previous day. Presentation of a new meal to a hungry great horned owl will delay pellet egestion to allow the bird to consume another meal, thereby producing a larger pellet later. Field observations have suggested that owls may sometimes need to clear a pellet before ingesting the next meal. The pyloric region of the ventriculus is located on the right side of this organ at a 90° angle to the longitudinal axis of the proventriculus. A combination of interdigitating folds and a sphincter control the exit of material from the stomach, favoring the passage of small, liquid, or soft items.

**Intestine, Liver, Pancreas**

The raptor duodenum is relatively long and includes secondary loops in some species (e.g., sea eagles; see Figs 4-6). Pancreatic and bile ducts empty into the ascending loop of the duodenum (Fig 6). Birds of prey possess a relatively large gallbladder. Bile salts are important for lipid digestion in birds and are recycled in the liver from the enterohepatic circulation. A unique trait of the avian digestive tract is the periodic active reflux of digesta from the ileum and duodenum back into the stomach. Duke suggests this retroperistaltic action is an adaptation to
prolong digestion and increase digestive efficiency without increasing digestive tract size or feeding frequency. In raptors, the reflux of digesta from the intestine back into the stomach occurs approximately every 30 to 60 minutes. Bicarbonate from the pancreas neutralizes the pH of digesta, which facilitates the function of bile salts and enzymes in the intestine. The pancreas also produces trypsin, chymotrypsin, and carboxypeptidase for reduction of protein to free amino acids and oligopeptides of 2 to 6 amino acids in length. The free amino acids and oligopeptides are further hydrolyzed by enzymes emanating from the brush border of intestinal villi. Klasing points out that Falconiformes that pursue aerial prey have intestines that are 20% to 40% shorter than those that hunt by soaring or pouncing. There may be a loss in digestive efficiency in birds with a shorter intestinal tract, which may be compensated by a lighter body weight, increased aerial agility, and greater hunting success.

Ceca, Rectum, and Cloaca

The ceca are a pair of blind sacs emerging from the ileocolic junction. In diurnal birds of prey, the ceca are vestigial. In Strigiformes, the ceca are well developed and appear to function primarily in water resorption and nitrogen homeostasis. The ceca are periodically emptied and produce a sticky, homogeneous, particularly malodorous dropping at the rate of 2 to 3 per day in the domestic fowl. The large intestine of raptors is short and linear, except in the kestrel, which is noted for possessing an unusually long large intestine (believed to augment water conservation). The mucosa of the large intestine has villi, though not to the degree of the small intestine, and is an important site for water resorption from both feces and urinary products. Urine enters the large intestine by active retroperistalsis from the uredeum of the cloaca. Because of the high nitrogen content of a protein-rich diet, birds of prey produce large amounts of nitrogenous waste. The extra liquid necessary to excrete and transport the large amounts of nitrogenous waste is recovered efficiently by the ceca (in Strigiformes), cloaca, and large intestine. The process can be so efficient that many birds of prey do not require water beyond that provided in their prey, although water should still be provided to these birds in captivity.

Additional Digestion Considerations

The digestive and metabolic strategies of birds of prey are optimized for a diet high in protein and low in carbohydrates. Relatively few studies have investigated protein accretion, gluconeogenesis, and fatty acid synthesis in faunivorous birds relative to omnivorous (e.g., poultry) or florivorous (e.g., parrots) birds. Raptors generally have lower plasma insulin levels relative to chickens, although it was determined that insulin is more responsive to glucose level in kestrels than in chickens. However, it appears that insulin is not the only significant factor involved in the regulation of glucose in kestrels. Barn owls were found to take longer to absorb orally delivered glucose and to clear blood glucose than chickens. Delayed insulin clearance is believed to result from a decreased ability to downregulate gluconeogenesis from lactate or threonine, possibly because these substrates, particularly threonine, are consistently abundant in the natural diet (whereas they vary from insufficient to excessive in the diet of omnivores). Delayed insulin clearance could be an important consideration when selecting an appropriate gavage diet for chronic inanition.

Parasitic Diseases

Protozoa

Trichomoniasis. Trichomonas gallinae is the causative organism of trichomoniasis, also known as “frounce” by falconers (or “canker” among pigeon fanciers). Lesions associated with trichomoniasis infections are caseous, diphtheritic, painful, vascular,
and occur in the respiratory and upper digestive systems. Confirmation of a *T. gallinae* diagnosis is made by microscopic visualization of flagellated protozoans on a fresh saline solution mount of lesion material. The organism can still be recovered from warmed saline solution preparations of samples collected from lesions even after several days of carcass refrigeration. The organism is largely adapted to Columbiformes, so that even apparently healthy individuals can be subclinical carriers. Some falconers avoid pigeon meals for their birds altogether, or at least practice freezing and thawing of pigeon carcasses along with removal of the crop, cervical esophagus, and head because of the possibility of exposing their birds to this parasite. Because trichomonads can be present in other tissues of a diseased bird, simply removing the upper gastrointestinal tract alone will not completely eliminate risk of bird exposure. Direct transmission of *T. gallinae* between raptors has been reported. Treatment with metronidazole at 30 to 50 mg/kg every 24 hours for 5 to 7 days or carnidazole (Spartrix; Wildlife Pharmaceuticals, Inc., Fort Collins, CO USA) at 30 mg/kg every 12 hours for 3 days is usually effective to treat birds infected with *T. gallinae*. Birds that have severe oral, esophageal, or ingluvial lesions may require parenteral fluid support, gavage feeding, or placement of a pharyngostomy tube for feeding. Pain and the potential for secondary bacterial infection should also be addressed by treating the patient with butorphanol (1-2 mg/kg orally [PO]/intramuscularly [IM] every 8-12 hours as needed).

Coccidiosis. *Eimeria* spp., *Cryptosporidium* spp., *Frenkelia* spp., *Sarcocystis* spp., *Caryospora* spp., and *Toxoplasma gondii* have all been represented in raptors. Most coccidian infections are nonpathogenic in raptors, but significant clinical disease may occur in young birds, in large or mixed collections, and in animals with compromised immunity. Clinical signs of coccidiosis in birds of prey include lethargy, depression, anorexia, salivary gland inflammation, regurgitation, enteritis, cloacitis, conjunctivitis, sneezing, diarrhea, and death. The most common pathogenic genus of coccidia diagnosed in raptor species appears to be *Caryospora* spp., particularly in merlins. Oocysts are sporulated in *Sarcocystis* spp. and nonsporulated in *Caryospora* spp. when identified on microscopic fecal examination. Tissue cysts of *Sarcocystis* spp. are found in some birds of prey and may not be associated with clinical disease. In the case of cryptosporidia, a definitive diagnosis can be achieved through cytological examination of mucosal swabs or feces, using direct microscopy and staining techniques. Published treatments for avian coccidiosis include sulfadimethoxine (Albon; Pfizer Animal Health, Exton, PA USA) at 25 to 55 mg/kg PO every 24 hours for 3 to 7 days, pyrimethamin (Daraprim; Catalytica Pharmaceuticals, Inc., Greenville, NC USA) at 0.5 mg/kg PO every 12 hours for 14 to 28 days, toltrazuril (Baycox; Bayer, Leverkusen, Germany) at 7 mg/kg PO every 24 hours for 2 to 3 days, paramomycin (for *Cryptosporidium* spp.) at 100 mg/kg PO every 12 hours for 7 days, or amprolium at 15 to 22 mg/kg PO every 24 hours for 4 to 6 days (supplementation of thiamine may be recommended). Forbes has investigated the develop-
ment of a vaccine to decrease the transmission of Caryospora spp., which can be particularly important in facilities housing mixed species.16

Nematodes
Capillariasis has been documented in free-living North American raptors.17,18 Many infections are likely subclinical, but overt disease signs include emaciation, depression, diarrhea, dysphagia, oral lesions, and death.8 The oral and esophageal lesions may resemble those associated with trichomoniasis. Gastrointestinal ascarids include Ascaridia spp., Porrocaecum spp., and Contracaecum spp., and clinical signs, if present, may resemble capillariasis. Diagnosis of nematodes in the gastrointestinal tract is accomplished by microscopic analysis of feces, digesta, or mucosal impressions. Because prey may also contain parasite ova, care must be taken to differentiate the source of exposure when a bird has been diagnosed with capillariasis. To determine the exact source of exposure, and because ova may be shed inconsistently, examination of serial diagnostic samples is recommended.19 Ivermectin, fenbendazole, levamisole, mebendazole, and piperazine have been prescribed for the treatment of nematodes. Care must be taken when treating birds with fenbendazole because it has been reported that this antiparasitic agent may cause death in some avian species.8,20

Other Helminths
Trematodes may infest the bile ducts and duodenum of raptors and, in most cases, are not considered pathogenic, even in large numbers, although lethal cases are reported.19,21,22 Clinical signs of trematode infestation can include weight loss, diarrhea, and death. A sedimentation technique is recommended for concentrating trematode ova from feces for microscopic identification.9 Treatment with rafoxanide (Ranide, MSG AGVET; Merck & Co., NJ USA) or praziquantel (Droncit; Haver Lockhart, KS USA) has been described for raptors diagnosed with trematode infestations, although one author notes that praziquantel may only decrease transmission and not eliminate the parasite in nonraptor species.19,23

Cestodes are uncommon and rarely cause disease in raptors.19 Clinical signs, if present, may include mild diarrhea and weakness. Diagnosis of a cestode infestation is usually made by visualization of proglottids in the feces or around the vent. Praziquantel is the treatment of choice for raptors diagnosed with cestodes. Acanthocephalans are rarely reported in raptors and are generally considered nonpathogenic.17,19,21,24 If present, acanthocephalans are usually located in the aborad small intestine, but unfortunately there are no published treatments for raptor species.19

Bacterial/Fungal Diseases
Cooper lists Escherichia coli and Proteus spp. as normal bacterial flora within a raptor’s lower intestinal tract.9 In addition to these, Staphylococcus aureus, Bacillus spp., and Corynobacterium spp. are commonly cultured from the pharyngeal area of birds of prey.9 The bacterial organisms Lamberski describes as normal intestinal flora of red-tailed and Cooper’s hawks in California include Staphylococcus/Micrococcus spp., Corynebacterium spp., Pasteurella spp., Streptococcus spp., Salmonella spp., and Bacillus spp.25 Bacteria that originate from or infect the gastrointestinal tract of raptors that may be pathogenic include E. coli, Proteus spp., Pasteurella multocida, Salmonella spp., Klebsiella spp., Pseudomonas aeruginosa, Shigella spp., and Clostridium botulinum.8,26,27 In a survey of falcons in the United Arab Emirates, E. coli, Chlamydia psittaci, Pseudomonas spp., and Clostridium perfringens are the most common bacterial pathogens.28 Clinical signs of disease, if present, of gastrointestinal infection with these organisms listed above are often nonspecific (e.g., slow crop emptying, weight loss, regurgitation, diarrhea, lethargy, death). Initial treatment should be based on the patient’s history, clinical signs, and cytological results of gastrointestinal/flecal swabs. Adjustments to the treatment regimen may be modified depending on organism isolation, antimicrobial sensitivity testing, and response to therapy. Most gastrointestinal infections of raptor species usually involve Gram-negative and anaerobic organisms, therefore the author’s preferences for immediate antibiotic therapy include trimethoprim sulfadimethoxazole (Bactricin; Roche, Nutley, NJ USA) at 48 mg/kg PO every 12 hours, ciprofloxacin (Bayer, Shawnee Mission, KS USA) at 20 mg/kg PO every 12 hours, Clindamycin (Antirobe; Pharmacia and Upjohn, Kalamazoo, MI) at 50 to 100 mg/kg PO every 12 hours, or amoxicillin/clavulanate (Clavamox; Pfizer, New York, NY USA).10 Pasteurella multocida is a Gram-negative aerobe that infects raptors either through consumption of infected waterfowl or from the bites of domestic felids.15,29,30 Infection can originate from crop injuries or abscesses associated with prey ingestion. Clinical signs associated with a P. multocida infection include weakness, animal bite lesions, weight loss, ocularonasal discharge, infraorbital swelling, dysphonia, synovitis, neurological signs, peripheral edema, and whitish plaques of the oral cavity and esophagus.20,31 Infected birds can die suddenly or over a
period of time, depending on the pathogenicity of the organism and the health and species of bird. Histologic lesions of an avian *P. multocida* infection are consistent with septicemia and include disseminated intravascular coagulation, serositis, and microabscessation. Diagnosis can be accomplished by culture of the organism from lesions. Treatment should include fluid support and antimicrobial therapy that may include cefotaxime (Claforan; Hoechst-Roussel/Intervet Inc., Somerville, NJ USA) 100 mg/kg IM every 12 hours or piperacillin/tazobactam (Zosyn; Wyeth, Madison, NJ USA) 200 mg/kg IM every 12 hours.}

A necrotizing gastroenteritis associated with *Clostridium perfringens* infections has been described in raptors, although raptores seem resistant to infection. Diagnosis of *C. perfringens* is based on cytological examination of the fecal material, and, if confirmed, a recommended treatment is trimethoprim sulfadimethoxine at 48 mg/kg every 12 hours for 7 to 10 days.

*Pseudomonas* spp. infections will cause clinical disease in raptor species and has been reported as a contributing infectious organism in falcon stomatitis cases from which *Trichomonas* spp. were isolated.

*Mycobacterium avium* has been the most frequently reported mycobacterial species isolated in raptores, although other species and subspecies of mycobacteria (e.g., *M. avium paratuberculosis*) are emerging as molecular diagnostic techniques improve and their use increases. Mycobacteriosis most commonly affects the gastrointestinal tract and liver, causing formation of granulomatous tubercles and thickening of the intestinal wall. However, other tissues and organs can be infected including meninges, joints, subcutis, skin, spleen, lung, and bone. Because many tissues can be affected, clinical signs are generally nonspecific but may include muscle wasting, swollen and painful joints, subcutaneous nodules, and depression. A complete blood count will usually reveal a significant leukocytosis (20,000–200,000/μL white blood count) with a concurrent heterophilia and monocytosis. A diagnosis of mycobacteriosis can be supported by acid-fast staining of fecal smears (least sensitive method) or polymerase chain reaction testing or culture of feces or lesions. A definitive diagnosis of mycobacteriosis currently requires culture, which involves special laboratory conditions and precautions. Treatment protocols for mycobacteriosis-infected companion birds can be found in the literature, but no controlled treatment studies have been performed in these species. Treatment is not generally recommended for wild birds because the duration of treatment may last at least 12 months or more and there is potential for human infection and recurrence. However, it may be possible to treat mycobacteriosis in permanently captive birds.

*Candida albicans* infections occur in raptors, particularly falcons. Samour cites that immunosuppression (e.g., stress, malnutrition [hypovitaminosis A], medical treatment) is usually involved in raptors diagnosed with candidiasis. *Candida albicans* infections usually involve the oral cavity, crop, or esophagus, with affected birds exhibiting dysphagia, crop stasis, regurgitation, and depression. Palpation of the crop may reveal thickening and roughening of the ingluvial wall. Lesions consist of white or gray plaques and a “turkish towel” appearance to the mucosa. Stained cytological preparations from swabs of lesions or fecal material may reveal abundant budding yeast. Published treatments include the use of nystatin, ketoconaazole, itraconazole, fluconazole, or miconazole.

**Viral Diseases**

**Herpesvirus**

Geographically, avian herpesviruses is a global disease. Herpesvirus has been identified in pigeons and doves on every continent except Antarctica, and in owls, falcons, and eagles in North America and Europe. Herpesviruses tend to be adapted to a specific host species and become widespread within the free-living populations of this species. Infections in healthy, natural hosts are generally mild, subclinical, and become latent. More severe or lethal disease from host-adapted herpesviruses occurs most often with young birds or in older birds that are immuno-compromised with concomitant illness or injuries. Herpesviruses are spread mainly by activities that require direct contact (e.g., consumption, mating, feeding of young or mates, fecal-oral routes). There are no published reports of herpesviruses being vertically transmitted. Consumption of pigeons latently infected with Columbid herpesvirus (CHV) is most likely the primary route for herpesviral infection and disease in falcons. In these nonadapted hosts, the virus causes greater morbidity and mortality (up to 100% flock mortality). One authority suspects that falcons traditionally from regions that are free of common pigeons may be more susceptible to disease from CHV (Dewey Savell, falcon breeder, Oakley, CA, personal communication, September 2007).

Gross lesions associated with a herpesvirus infection include hepatomegaly and splenomegaly, usually with multifocal pale necrotic foci throughout the
parenchyma. Pseudomembranous lesions may also be found in the oral cavity and any point within the gastrointestinal tract, the bronchioles, thymus, thyroid, kidneys, and gonads.\textsuperscript{44} Histologically, herpesvirus infections produce intranuclear inclusion bodies in affected cells.\textsuperscript{48} Birds are typically lethargic, weak, experience slow crop emptying, and exhibit biliverdinuria.\textsuperscript{45} The disease may progress rapidly in extremely susceptible birds, and they will usually die within 1 to 3 days of infection.\textsuperscript{39,42-44}

Treatment for avian herpesvirus is rarely rewarding and focuses on aggressive supportive care (e.g., parenteral fluids, supplemental heat, removal of crop contents in the event of crop stasis). Antiviral drugs such as acyclovir have appeared to anecdotally benefit infected birds but will not clear the infection (W. Ferrier, oral communication, January 2007). Prevention of herpesvirus infections centers mainly on careful inspection of pigeons used for raptor food or total avoidance of this type of bird as a dietary choice. Closed aviary concepts applied to both the feeder pigeon flock and to the raptors themselves are also important and should be a cornerstone to the husbandry practices of that facility. Other avian herpesviruses, such as isolates associated with Pacheco’s disease, have been addressed by vaccination. A CHV vaccine has been successfully tested in challenge trials with gyrfalcon hybrids but is still not widely available.\textsuperscript{49} Herpesviruses generally do not remain active for long outside of the host, therefore they are spread mainly by activities that require direct contact (e.g., mating, feeding of young or mates, fresh fecal contamination and consumption).\textsuperscript{39,42} Because herpesviruses have not been shown to have vertical transmission, herpesvirus-free collections of birds can be created if eggs are removed and incubated either artificially or under a surrogate hen.

**Pox**

Poxviruses have been identified in a number of raptor species around the world.\textsuperscript{39,50} Poxvirus lesions primarily occur on the face, feet, legs, conjunctiva, oral cavity, or vent. Birds may be unable to feed if lesions mechanically obstruct the oral cavity or obstruct vision. Generally, birds that clear the infection are considered immune, but recurrence has occurred in recovered birds undergoing stressful events, suggesting that some birds may remain latently infected.\textsuperscript{39} Control of biting insects (e.g., mosquitoes), quarantine of infected birds, and good facility hygiene are important preventative measures. A commercial vaccine to obtain immunity against poxvirus infections has been used in falcons.\textsuperscript{31}

**Adenovirus**

Falcon adenovirus appears to be host-adapted and widespread among peregrine falcons but can also cause disease in nestling aplomado, teita, and teita × peregrine falcon hybrids.\textsuperscript{52,53} One case report describes adenovirus in a Harris hawk, Bengal eagle owl, and Verreaux’s eagle owl.\textsuperscript{54}

**Botulism**

Toxins produced by \textit{Clostridium botulinum}, which may be present in carrion or affected waterfowl prey, are particularly potent.\textsuperscript{8} Clinical signs associated with botulism toxicosis include flaccid paralysis of neck, limb, pharyngeal, and respiratory muscles. Diagnosis is based largely on history and clinical signs but can be confirmed by mouse inoculation neutralization assay or with toxin analysis of frozen kidney or liver from the affected bird. Treatment of botulism toxicosis includes supportive care (e.g., parenteral fluids, nutritional support, supplemental heat) and administration of \textit{C. botulinum} type A or C antitoxin (0.05-1.0 mL/d).\textsuperscript{8}

**Lead Intoxication**

Lead is ingested as fragments from fishing tackle, bullets, or other projectiles that are embedded or contained in the gastrointestinal tract of carrion or live prey.\textsuperscript{55,56} Lead intoxication is commonly diagnosed in raptor species and is a worldwide concern. Neurological effects (e.g., depression, paresis, intention tremors, hyperesthesia, seizures), anorexia, biliverdinuria, and anemia are common clinical signs observed in lead-intoxicated birds.\textsuperscript{55-57} There appears to be some species variability regarding the disease severity associated with lead intoxication.\textsuperscript{56} Affected birds may also experience gastric hemorrhage and enteritis with resultant melena or hematochezia. Radiographs and blood lead level will confirm the diagnosis of lead toxicosis. Recommended treatment includes the use of chelators such as CaNa\textsubscript{2} EDTA (Calcium Disodium Versenate; 3M Pharmaceuticals, Northridge, CA USA) 35 mg/kg IM every 12 hours.\textsuperscript{10} After stabilizing the patient, removal of any lead foreign body within the gastrointestinal system via gastric lavage, endoscopic retrieval, or proventriculotomy is indicated.\textsuperscript{55,58}

**Sour Crop**

Ingluvial stasis can result from obstruction or disease of the gastrointestinal tract (e.g., trichomoniasis,
candidiasis), from overfeeding, or from systemic conditions such as severe dehydration or inanition followed by feeding. In addition to crop stasis, the bird may regurgitate, and the regurgitant may have a fetid odor because of fermentation of crop contents, whereby the animal becomes depressed and produces small mutes consisting of bile and urates. A concurrent bacterial infection can ultimately cause crop edema and endotoxemic shock. Treatment for ingluvial stasis includes removal of food, either orally or by ingluviotomy, followed by oral antibiotics, such as trimethoprim sulfadimethoxazole at 48 mg/kg PO every 12 hours or clindamycin at 50 to 100 mg/kg PO every 12 hours until clinical signs resolve. Some authors also lavage or gavage diluted antiseptics (e.g., chlorhexidine) into the upper gastrointestinal tract after removal of fermented crop contents. Radiographic images and other diagnostic tests are recommended to identify and address the underlying cause of ingluvial stasis. Food should slowly be reintroduced in conjunction with fluid therapy.

**Trauma and Foreign Bodies**

Because of the anatomic and physiologic pyloric function in birds of prey, ingested foreign bodies should be confined to the upper alimentary tract and stomach. Fishhooks and other fishing tackle can be a serious disease concern in bald eagles, ospreys, and other piscivorous birds of prey. In some cases, the hook does not actually perforate the gastrointestinal tract, and consequently the birds are able to regurgitate this foreign body or they can be carefully retracted and removed under anesthesia. Occasionally, the removal of hooks can be accomplished by pressing them through the mucosa and surface epithelium to expose the barbs (if present), at which time the tip of the hook is cut off, and the remainder, with any attached line, is orally retracted. Often, surgical removal via ingluviotomy or proventriculotomy is necessary. Hooks in the crop or cranial thoracic esophagus are usually accessed via an ingluviotomy incision, whereas hooks lower in the gastrointestinal tract require a left lateral approach into the proventriculus. Survey radiographic images of the entire upper gastrointestinal tract should be taken after removal of the known foreign body(s) to ensure all objects (e.g., tackle, hooks, sinkers) have been extracted. Birds affected by heavy metal intoxication may not be able to cast or otherwise regurgitate all of the ingested lead fragments from the ventriculus. Because of the absence of a constricted proventricular-ventricular isthmus, compact dense particles, such as lead fragments or fishing sinkers, may be removed by gastric lavage via an ingluviotomy incision. If a gastric lavage is unsuccessful, endoscopic retrieval via an ingluviotomy or proventriculotomy incision is indicated.

Severe trauma or disease (e.g., trichomoniasis) of the crop wall may result in devitalization of this tissue and the overlying surface epithelium, resulting in the formation of a fistula or granuloma. Such defects must be surgically debrided and closed in 2 layers (crop wall and skin). Ingluvial obstruction due to a concentration of bones from prey (food) is a relatively common gastrointestinal emergency presentation among falconry birds. Frequently, the obstruction is the neck of a quail or other avian prey item. Preventative measures include offering necks only if they have been cut into several pieces, particularly when feeding birds that demonstrate a tendency to quickly gulp their food. Reduction of a bird’s tendency to quickly eat its food can be managed by increasing the bird’s flying weight slightly or by increasing the bird’s sense of security when feeding (i.e., feeding them indoors or out of sight from other perceived competitors). When ingluvial obstructions occur, the bird may make frequent, unsuccessful motions to “put over” their crop (a motion involving alternately extending the neck and shrugging the shoulders to force food from the crop into the thoracic esophagus). Resolution of ingluvial obstruction is accomplished by performing an ingluviotomy. The dependent ventral midline region of the crop is a relatively avascular area and easily accessible point of entry, and consequently is the recommended site for surgical entry. To perform an ingluviotomy, longitudinal skin and matching crop incisions are made, whereby the surgeon enters the crop lumen. After removal of the ingesta, a blunt instrument or endoscope should be used to inspect the cervical and thoracic esophagus for remaining objects that may be perforating the wall or blocking the lumen. The crop and skin are closed in 2 layers with monofilament suture in a simple interrupted pattern.

Lower gastrointestinal traumatic or obstructive conditions are rare in birds of prey. However, urolithiasis does occur in chronically or severely dehydrated individuals. Disabled free-living birds of prey may suffer from uroliths during severe water- and food-deprivation because of the efficiency of water resorption in the rectum and cloaca. The presence of uroliths will cause visible and audible straining during attempts to defecate. The urate concretions are easily detected as a gritty sensation when gently probing the cloaca with a thermometer.
enema or mechanical reduction and retraction with thumb forceps will usually result in an uneventful removal of the uroliths (Fig 7).

**Nutritional Concerns**

Provision of a whole-prey, wholesome diet is important to the long-term health of raptors. When feeding whole prey is not possible, the use of a vitamin supplement intended for birds of prey (e.g., Vitahawk; DB Scientific, Oakley, CA USA) is recommended. Whole prey provides all essential amino acids in their correct proportions for sustaining life, while hair and feathers should assist in casting.

Reintroduction to spontaneous alimentation after gastrointestinal stasis, chronic inanition, or other severe disease should be accomplished carefully with particular attention to hydration and type of metabolizable energy provided. Fluid therapy is a primary concern because a raptor’s digestive processes involve abundant secretion. Digestion is also energy intensive, so a critical care diet’s energy sources should be easily absorbed. As mentioned earlier, raptors are particularly well-adapted to metabolizing amino acids to meet their energy needs. This process can also aid in the metabolism and elimination of lactic acid, which generally accompanies inanition and chronic debilitation. Therefore, use of carbohydrate-rich gavage diets may not be as helpful as diets rich in fat and protein, particularly when these constituents are reduced to short chains or amino acids that are easily absorbed by the intestinal mucosa.

**References**

28. Muller MG, Mannil AT, George AR: Most common bacterial infections in falcons in the United Arab...


