

CHAPTER

19

**GASTRO-
ENTEROLOGY**

■
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Diseases of the alimentary tract occur frequently in birds. Nonspecific clinical signs of gastrointestinal diseases may include anorexia, dysphagia, regurgitation, vomiting, constipation, diarrhea and tenesmus. With polyuria, the feces are normal and are surrounded by a large volume of clear fluid, while with diarrhea the feces are abnormal (see Color 8). The composition and quality of food and ingestion of bedding material, poisonous plants or chemicals may influence gastrointestinal signs. Weight loss and generalized weakness are characteristic of chronic diseases.

Fecal evaluation, hematology, blood chemistry, radiology and esophago-ingluvio-(gastro)scopy or laparoscopy are considered indispensable diagnostic tools in avian gastroenterology. Diseases that may affect the gastrointestinal system are listed in Table 19.1.

Cytologic examination of a fresh ingluvial aspirate is best for detecting flagellates (*Trichomonas* spp.). Examination of freshly voided feces is essential to detect *Histomonas meleagridis*, *Hexamita* spp., *Giardia intestinalis*, *Cochlosoma* sp. and *Chilomastix gallinarum*. Direct microscopic examination of feces may reveal helminthic ova and protozoal oocysts. Flotation and sedimentation techniques are best for detecting the low number of eggs or oocysts that occur in an early parasitic infection (see Chapter 36). Parasites infecting the liver, kidney, uterus and pancreas can deposit ova or oocysts that can be detected in the feces. Parasite ova originating from the respiratory tract may be coughed up, swallowed and found in the excrement.^{28,78}

TABLE 19.1 Differential Diagnosis of Clinical Signs Associated with the Gastrointestinal Tract**Regurgitation or Vomiting in Adults**

- Iatrogenic - apomorphine, levamisole, trimethoprim/sulfadiazine (macaws), ketoconazole, doxycycline suspension (particularly macaws and Amazons)
- Fear and excitement (vultures, pelicans, penguins)
- Courtship behavior (male psittacines)
- Crop milk feeding in pigeons
- Physiological cast formation - (raptors)
- Goiter (particularly budgerigars)
- Callus formation after coracoid fracture
- Neuropathic gastric dilatation (NGD)
- Food allergies
- Motion sickness
- Viral diseases - looping ill virus, Pacheco's disease virus, pigeon herpesvirus, avian polyomavirus, avian viral serositis (togavirus), poxvirus (diphtheritic form)
- Bacterial diseases - megabacterial infection, most Enterobacteriaceae, Pasteurella, Serratia
- Mycotic diseases - candidiasis, aspergillosis
- Helminths (oropharynx/ingluvies/esophagus) - capillariasis, ser-ratospicilliasis
- Protozoal disease - trichomoniasis of upper digestive tract, Plasmodium (penguins, gyrfalcon)
- Poisoning - alcohol, arsenic, copper, lead, organochlorine (lindane), organophosphate, carbamate, organomercurial, rotenone, phosphorus, polytetrafluoroethylene (Teflon), sodium chloride, thallium, zinc
- Plants - Yew (*Taxus baccata*), *Philodendron* spp., *Rhododendron* spp. (azalea), Solanaceae (green berries and roots)
- Obstructed alimentary tract - stricture, foreign body, neoplasia, intussusception, volvulus, hernia, stenosis, parasites, impaction, paralytic ileus
- Organopathy - renal disease, hepatopathy, pancreatitis, peritonitis, egg binding, electrolyte disturbances

Regurgitation in Neonatal Psittacines (Sour Crop)

- Overgrowth of bacteria or yeast (improper food storage)
- Overheated formula
- Underheated formula
- Crop burns
- Foreign body ingestion (eg, substrates)
- Improper formula consistency
- Over-stretching the crop
- Aerophagia
- Fear and excitement
- Infectious agents
 - Avian polyomavirus
 - Avian viral serositis
 - *Candida* spp.
 - Gas-producing bacteria

Diarrhea^{28,78}

- Use of antibiotics
- Dietary changes
- Bowel obstruction
- Toxins
- Obstruction
- Foreign bodies
- Organopathy - hepatopathy, renal disease, pancreatitis
- Viral diseases - Newcastle disease virus, paramyxovirus type 3, influenza, adenovirus, astrovirus, calicivirus, coronavirus, enterovirus, Pacheco's disease virus, pigeon herpesvirus, duck virus enteritis, herpesvirus (Ciconiidae), herpesvirus (guiformes), Marek's disease virus, orthoreovirus, parvovirus, reovirus, rotavirus, togavirus-like agent, retrovirus (leukosis/sarcoma group)
- Bacterial diseases - borreliosis, spirochaetosis, most Enterobacteriaceae, *Campylobacter* spp., *Streptococcus* spp., *Erysipelothrix rhusiopathiae*, *Listeria monocytogenes*, megabacteria, *Clostridium* spp., *Mycobacterium avium*, *Yersinia pseudotuberculosis*, *Aeromonas hydrophila*, *Pasteurella multocida*, *Pasteurella anatipestifer* (new duck disease)
- Chlamydia
- Mycoplasma
- *Candida albicans*
- Protozoa - *Histomonas meleagridis*, *Hexamita* spp., *Giardia* spp., *Cochlosoma* sp., *Chilomastix gallinarum*, coccidiosis
- Helminths - nematodes, trematodes, cestodes

Hematochezia

- Cloacal papillomas
- Egg laying
- Ulcers
- Hepatitis
- Infectious enteritis - bacterial, viral, parasitic
- Aflatoxicosis
- Coagulopathies
- Heavy metal intoxication
- Foreign bodies
- Cloacal neoplasias

Passing Undigested Food

- Gastric foreign body
- Gastrointestinal dysfunction
- Neuropathic gastric dilatation
- Enteritis - bacterial, viral, parasitic
- Pancreatitis
- Use of antibiotics
- Food allergies
- Hepatitis

Tenesmus

- Egg-laying problems (binding)
- Abdominal mass
- Goose venereal disease
- Cloacal pathology
- Prolapse
- Papilloma
- Stricture
- Cloacolith
- Cloacitis
- Intestinal obstruction (eg, constipation)
- Uterine prolapse
- Rectal prolapse
- Enteritis - diarrhea
- Bacteria
- Parasites
- Fungi
- Viruses
- Toxins
- Decreased bacteria (eg, indiscriminate antibiotic use)

Bacteriologic cultures of the gastrointestinal tract must be interpreted with respect to the normal flora.⁷⁸ Gram-positive microorganisms including lactobacilli, staphylococci, streptococci and *Bacillus* spp. are common in the oropharynx of healthy psittacine birds. *Mycoplasma* spp. and *Aspergillus* spp. are sometimes encountered.^{168,176} Enterobacteriaceae are normally not found in the feces of Psittaciformes and Passeriformes, where gram-positive organisms, especially *Corynebacterium* sp. and *Bacillus* sp., predominate.¹⁷ The isolation of a large number of Enterobacteriaceae in pure culture from Psittaciformes or Passeriformes is suggestive of a primary or secondary infection. *E. coli* and other Enterobacteriaceae are normal inhabitants of the gastrointestinal tract in Galliformes, Columbiformes, Falconiformes, Strigiformes and Corvidae.¹⁴⁰

Routine bacteriologic examination of the feces may fail to reveal some important microbes that can cause diarrhea, including mycobacteria, campylobacter and chlamydia. A technique for identifying mycobacteria is described in Table 19.2. Detection of campylobacter can be augmented by the use of Hemacolor; the bacteria appear S-shaped or in gull-wing form. Chlamydia is best detected using an antigen capture system.^{21,102}

TABLE 19.2 Detection of Acid-fast Bacteria in Feces⁷⁸

- Combine 4 grams of feces and 12 ml of 15% sputofluol (Merck)
- Gently mix for 30 minutes
- Centrifuge for 5 minutes 10,000 rpm
- Make smear of sediment
- Stain with Ziehl-Neelsen

The Beak

Anatomy and Physiology

The avian beak is a continuously growing, dynamic structure composed of bone, vascular layers, keratin, dermis, joints and a germinative layer. In psittacine birds, the upper and lower jaws are connected to the skull via a kinetic joint. The keratinized sheath covering the upper and lower beaks is called rhamphotheca and can be divided into the rhinotheca (maxillary keratin) and the gnathotheca (mandibular keratin).

The median dorsal border of the rhinotheca is called the culmen, and the median ventral border of the gnathotheca is called the gonys. The cutting edges of the rhamphotheca are called the tomia. The rhinotheca is perforated by the paired nostrils. Aviculturists classify caged birds into hardbills (eg, most psittacine birds) and softbills (eg, mynahs, starlings).

In ducks and parrots, the tip of the bill contains well developed mechanoreceptor nerve endings. The beak is used for prehension, for the physical preparation of food, and in some species such as parrots, for locomotion.¹³⁰

The rate of keratin replacement is strongly dependent on the use of the beak. In large parrots, the complete rhinotheca is replaced in about six months, while in toucans the rhinotheca grows approximately 0.5 cm over a two-year period. The rate of growth of the gnathotheca is about two to three times faster than that of the rhinotheca.³⁸ Shedding and replacement of the rhinotheca has been described in capercaillie (annually)⁴ and Suriname finches.

Beak Diseases

A variety of congenital and acquired defects, including scissor beak and mandibular prognathism, can interfere with normal beak function. In gallinaceous birds, a deformed upper mandible has been associated with embryonic deficiencies of folic acid, biotin or pantothenic acid. Crusty, scab-like lesions in the corners of the mouth are considered a definite sign of biotin or pantothenic acid deficiency in these birds.⁵ Examples of acquired lesions that can lead to malformations or necrosis of the beak include punctures, lacerations, splits and avulsions. Traumatic fractures, especially of the mandible, occur frequently in psittacine birds that get caught in hooks suspended from the ceiling of their enclosures or as a result of fighting.

Any bacterial, mycotic, viral or parasitic pathogen that damages the germinative layers of the beak can cause developmental abnormalities.¹⁰⁸ Examples include *Candida albicans*, psittacine beak and feather disease virus, *Knemidokoptes* spp. in Psittaciformes or *Oxyspirura* spp. in cranes. Rhinothecal overgrowth in psittacines, especially budgerigars, has been associated with liver disease (Figure 19.1).¹³³ The rhinotheca may overgrow in hardbills maintained in an indoor environment and provided soft foods. "Rubber bill," caused by insufficient mineralization of the upper beak, has been described with

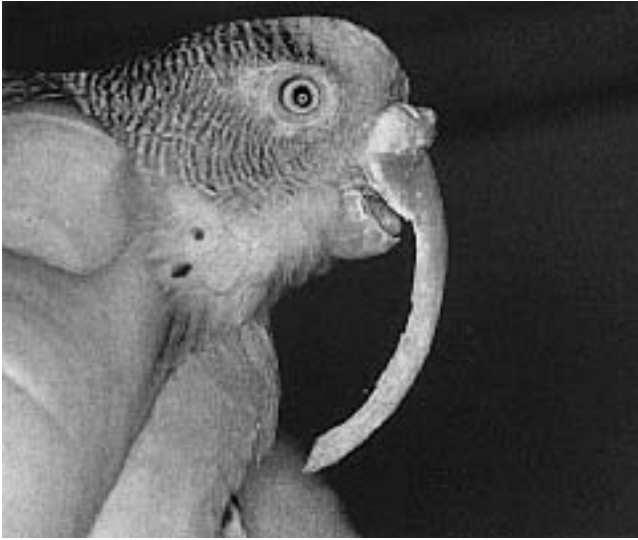


FIG 19.1 A two-year-old budgerigar, maintained on an all-seed diet in a strictly indoor environment, was presented for a beak trim. The bird was obese and was passing bile-tinged urates. This clinical presentation is suggestive of a hepatopathy.



FIG 19.2 An eight-year-old Umbrella Cockatoo was presented with a nine-month history of progressive sneezing and nasal discharge. The feathers around the beak were moist from a serous nasal discharge. The rhinotheca had a deep groove that extended from the nostril to the rostral commissure of the upper beak (arrows). The extent of this defect suggests that the germinative layer of the rhinotheca had been involved in a disease process for over six months. This bird belonged to a heavy smoker. A mixed population of gram-positive and gram-negative bacteria were cultured from a sinus aspirate. The bird responded to nasal flushing, systemic antibiotics, frequent exposure to fresh air and sunlight and being removed from a smoke-filled environment.

vitamin D and calcium deficiencies. Necrotic lesions at the commissure of the beak have been described with trichotecene mycotoxicosis, avian poxvirus and trichomoniasis (cockatiels).¹³⁷

Beak deformation consisting of loss of normal epithelium on the surface of the beak, upturning of the tomia and shortening of the upper beak have been reported secondary to photosensitization in ducks following ingestion of seeds from *Ammi visnaga*, *A. majus* and the plant or seeds from *Cynopterus watsonii* and *C. longipes*.¹⁵² Photosensitization has been suspected in many cases of vesicular dermatitis, but the precise etiologic agents are frequently undetermined.

Chronic rhinitis may lead to permanent defects in the adjoining germinative layer of the rhinotheca (Figure 19.2). Dysphagia, which may be recognized clinically as an accumulation of food under the tongue, can be an indication of rhamphothecal dysfunction.

The Oropharynx and Salivary Glands

Anatomy and Physiology^{49,101}

Birds lack an oropharyngeal isthmus, and the oral and pharyngeal cavities are combined to form an oropharynx. The walls of the oropharynx contain numerous mucus-secreting salivary glands (Figure 19.3). The palate contains a median fissure called the choana, which connects the sinuses to the glottis. Just caudal to the choana is the infundibular cleft, which is the common opening of the auditory tubes. Tongue anatomy varies widely among avian species. Parrots have intrinsic muscles in the tongue, while other birds have only extrinsic tongue muscles. Swallowing involves a rapid rostrocaudal movement of the tongue and the larynx, assisted by sticky saliva and caudally directed papilla on the tongue, laryngeal mound and palate. During swallowing, the choana, infundibular cleft and glottis are closed. The salivary glands secrete mucus and, in some species, amylase. During the breeding season, the salivary gland of swifts temporarily enlarges to produce an adhesive liquid used in nest construction. The nests of some of the cave swiftlets of Southeast Asia are made entirely of this edible secretion (birds' nest soup). The Grey Jay produces large quantities of mucus that are formed into boluses and stored on the sides of trees as a winter food supply. The mucosa of the oral cavity

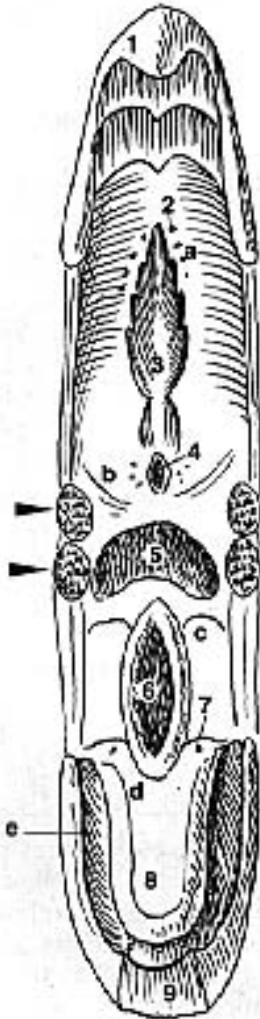


FIG 19.3 Anatomy of the oral cavity of an Umbrella Cockatoo. The depressor mandibulae muscles (arrows) have been transected bilaterally to allow the jaws to be opened, revealing the 1) upper beak, 2) openings of the seromucous glands, 3) choana, 4) rima infundibuli, 5) esophagus, 6) rima glottis, 7) salivary orifice, 8) tongue, 9) lower beak. Abscesses occur in multiple locations including a) perichoanal, b) pharyngeal, c) periglottal, d) lingual, e) lateral-ventral lingual and f) sublingual tissues.

in some passerine chicks is brightly colored, with distinctive markings that disappear when the chick is weaned. These markings appear to stimulate the parents to feed the chick.

■ Oropharyngeal Diseases

Table 19.3 lists common upper gastrointestinal tract diseases, the typical anatomic sites affected, the types of lesions induced and the common species affected.

Poxvirus

Poxvirus may cause proliferative caseous lesions (diphtheritic form) in the mouth and esophagus in a variety of avian species. Diagnosis can be achieved by identifying elementary bodies (Bollinger bodies) in impression smears prepared from lesions and stained with Wright's stain or by the Gimenez method. Trichomoniasis lesions may have a similar gross appearance.

Pigeon Herpesvirus Infection (Smadel's Disease)

Pigeon herpesvirus (PHV) infection has been associated with pharyngeal and esophageal diphtheritic membranes, which are attached to the underlying tissues. Lesions are most severe when secondarily infected with *Trichomonas* spp.¹⁰⁵ Other clinical signs include dyspnea, mucopurulent rhinitis and conjunctivitis. Histologic identification of basophilic and eosinophilic intranuclear inclusion bodies is suggestive.

Granulomas

Granulomas caused by *Mycobacterium* spp. or other bacterial or fungal agents frequently occur in the oral cavity. A diagnosis can be made by staining suspected material with the Gram's or Ziehl-Neelsen methods (see Table 19.2).^{50,71,202} Surgical removal in conjunction with appropriate antimicrobial agents is usually effective in resolving non-mycobacterial-induced granulomas. A case of malignant fibrohistiosarcoma located on the tip of the tongue in a seven-year-old Brown-throated Conure was successfully removed by radiosurgery.

Nematodes

Various *Capillaria* spp. may infect the mucosa of the tongue, pharynx, esophagus and ingluvies of Falconiformes, Psittaciformes, Galliformes, Passeriformes and Anseriformes.^{78,91,174} Characteristic lesions include hemorrhagic inflammation in the commissure of the beak and diphtheritic membranes in the pharynx and tongue. Parasites can be found embedded in inflammatory material. Typical bipolar eggs may be found in esophageal smears or ingluvial washings. In Strigiformes, *Synhimanthus (Dispharynx) falconis* has been reported in the oropharynx.¹⁰⁹

Spirurid infections have been reported in diurnal and nocturnal birds of prey. Lesions containing the adult nematodes can be found in the mouth, esophagus and crop. The embryonated eggs are thick-walled. Ascarides belonging to the genus *Contracaecum* have been found in fish-eating birds, and severe infections of the oral cavity have been documented in young Pelecanidae. In birds of prey, *Seratospiculum amacu-*

TABLE 19.3 Upper Gastrointestinal Tract Diseases^{22, 24, 51, 76, 191, 220, 221}

Organism	Location	Lesion Type	Species Susceptibility
<i>Candida</i> spp.	Oral cavity Esophagus	Ulcerative, necrotic, diphtheritic	Most species, particularly neonates, immunosuppressed animals
Duck enteritis virus	Oral cavity Sublingual salivary glands	Ulcerative	Ducks
Herpesvirus	Oropharynx, Esophagus Proventriculus	Diphtheritic	Owls
Lice (<i>Piagetiella peralis</i>)	Oral cavity	Stomatitis	Penguins
Leeches (<i>Theromyzon</i> spp.)	Nasal cavity, Conjunctiva Oropharynx	Hyperemia at attachment site	Anseriformes
<i>Mycobacterium</i> spp.	Tongue, Frenulum Hard palate	Granulomas	Psittaciformes, Falconiformes Galliformes
Neoplasias	All locations	Masses, ulcerative	Most species
Papillomas	Oropharynx, Esophagus Proventriculus	Masses	Psittaciformes
Pigeon herpesvirus	Pharynx, Esophagus	Diphtheritic	Pigeons
Poxvirus	Mouth Esophagus	Diphtheritic	Galliformes, Psittaciformes, Passeriformes, Raptors, Columbiformes
Trematodes (<i>Cathaemasia</i> spp. <i>Clinostomum</i> spp.)	Oral cavity	Stomatitis	Ciconiiformes
Trichomoniasis	Oral cavity, Esophagus Crop	Ulcerative Proliferative	Raptors, Psittaciformes, Columbiformes, Passeriformes

latum can cause lesions that resemble those of oral trichomoniasis.²¹² The adult worms are found in the air sacs. Eggs may be found in the oral mucus or feces.

Hypovitaminosis A

In psittacine birds, a typical clinical sign of hypovitaminosis A is metaplasia of the submandibular or lingual salivary glands and clubbing of the choanal papillae (see Color 8). Affected birds are usually fed all-seed diets with a large percentage of sunflower seeds. Treatment should include parenteral vitamin A and the use of a formulated diet. Keratogenic cysts in the lingual salivary glands should be differentiated from lingual abscesses by biopsy.

Lesions associated with hypovitaminosis A in gallinaceous birds first appear in the pharynx and are largely confined to the mucous glands and their ducts. The epithelium is replaced by a stratified squamous epithelium that occludes the ducts of the mucous glands, causing accumulations of secretions and necrotic debris. Small, white, hyperkeratotic le-

sions (up to 2 mm in diameter) may be seen in the nasal passages, mouth, esophagus, pharynx and crop.

Some authors suggest that hypovitaminosis A is unlikely in pigeons because these birds efficiently metabolize this vitamin.^{155,223} Other authors suggest hypovitaminosis A frequently occurs in pigeons but it is seldom recognized because the histologic lesions are limited to inflammation of the mucous glands.¹⁹⁴ As the condition progresses, the duct systems fill with masses of degenerate lymphoid and inflammatory cells, amorphous debris and mucus.

Sialoliths in Pigeons

Mucosal lesions that appear similar to those caused by hypovitaminosis A have been described on the palate of pigeons and are referred to as sialoliths (see Color 13).²²³ Sialoliths consisting of a proteinaceous substrate mixed with cellular debris are clinically recognized in approximately one percent of pigeons. The etiology of sialoliths remains unknown. However, based on their histologic, histochemical, chemi-

cal and physical characteristics, they are not thought to be caused by hypovitaminosis A.²²³ An association with pigeon herpesvirus infection has been suggested and seems plausible.^{110,206,223}

Foreign Bodies

Clinical signs of foreign bodies in the upper GI tract can include respiratory distress, head shaking, scratching the head with the feet, dysphagia or anorexia. A thorough oropharyngeal examination and radiographs may reveal the foreign body.

A string looped around the base of the tongue and passing down the esophagus has been associated with dysphagia and respiratory distress in gallinaceous birds.^{71,151} The string tightens and cuts further into the tongue with each swallowing movement, causing edema of the glottis. Ring-shaped foreign bodies (eg, tracheal rings of prey eaten by raptors) can become lodged around the tongue, causing avascular necrosis. Wooden, plastic or metal splinters



FIG 19.4 A mature Umbrella Cockatoo was presented for evaluation of palatine beak necrosis. The bird's feathers were in poor condition, and the bird appeared to be hungry but would eat reluctantly. The referring veterinarian had diagnosed PBFD based on the combination of beak necrosis and feather abnormalities. On physical examination, the defect in the palatine area of the beak appeared to contain a foreign body. Magnification indicated that the bird had a large wood splinter lodged in the occlusal surface of the upper beak. The splinter was removed and the wound was debrided and flushed. The bird's appetite improved within several days of presentation, and it began to preen normally within a week. The bird was DNA probe-negative for PBFD virus and polyomavirus.

(originating from toys or enclosures) may lodge in the mouth or esophagus of psittacine birds (Figure 19.4). Waterfowl frequently ingest baited fishhooks, which can be lodged anywhere in the upper or lower digestive tract. Fish bones may lodge in the pharynx or proximal part of the esophagus causing dysphagia. Plant hairs that lodge in the oral or esophageal mucosa can cause granulomas.

Stomatitis

Stomatitis in birds has been associated with the consumption of hot foods, ingestion of oil and ingestion of caustic substances.⁵¹ Birds that chew on silver nitrate sticks may have extensive chemical burns of the oropharynx and crop.¹³¹ Use of bipolar radiosurgery is superior to silver nitrate sticks for controlling hemorrhage of the beak and nails. Stomatitis may occur secondary to food accumulations caused by beak deformities (eg, PBFD) (Figure 19.5). Beak necrosis has been described in pigeons and gallinaceous

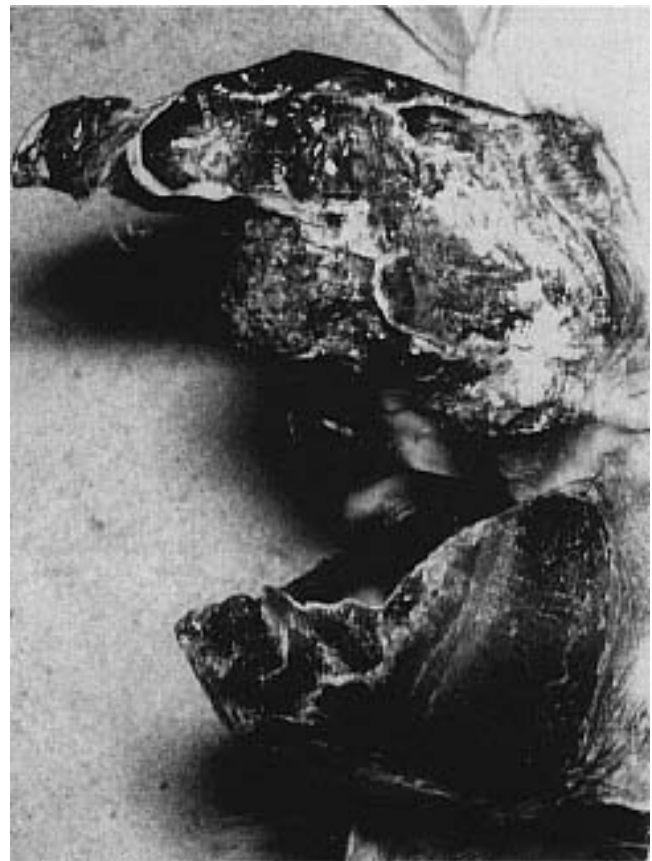


FIG 19.5 Palatine beak necrosis may occur in some birds with PBFD virus. Once a defect in the oral mucosa occurs, food may accumulate in the damaged tissues and create a nidus for secondary bacterial and fungal pathogens. In this Umbrella Cockatoo, the rhinotheca was completely necrotic and separated from the underlying bone. The bird had relatively minor feather pathology. This bird was euthanized.

birds fed a finely ground, high-gluten food. The gradual accumulation of fine particles of food along the inner edge of the lower beak leads to secondary infection and necrosis of the beak. Feeding pelleted rations prevents the problem. The tongue of turkey poults fed a finely ground mash may be curled backward by an accumulation of food on the floor of the mouth.¹⁵¹

Many trichotenes, notably T₂ toxin, can cause caustic injury to the alimentary mucosa. Yellow erosive and exudative plaques with underlying ulcers located near the salivary duct openings on the palate, tongue and buccal floor are characteristic lesions. Thick crusts of exudate may accumulate along the anterior margin of the beak. Anorexia is probably caused by the painful lesions in the beak.^{87,219}

Spirillum pulli may cause stomatitis in chickens.¹²⁷ The organism can be demonstrated cytologically in fresh scrapings from diphtheritic lesions or salivary glands. Experimental transmission occurs by inoculation of tissue suspensions and by direct contact. Moist, slick mucosal membranes have been described in pheasants with Newcastle disease virus and in chronic cholera.

Lacerations of the Tongue

Lacerations of the tongue have been encountered in psittacine birds and may be due to mate-induced trauma, automutilation during the excitement phase of post-anesthetic recovery or gnawing on sharp objects. The tongue is highly vascular and bleeds profusely if damaged. Anesthesia, magnification and radiocautery are usually necessary to control bleeding and repair the laceration.

Glossitis Gelatinosa Circumscripta

A gelatinous mass may be found on the dorsal aspect of the tongue in five- to twelve-week-old ducklings and goslings. The precise etiology is undetermined, but a multi-deficient diet has been suggested.¹⁰³

The Esophagus and Crop

Anatomy and Physiology^{31,49,101}

The esophagus lies immediately under the skin and to the right of the trachea (see Color 13).¹⁶ The internal surface of the esophagus is longitudinally folded, increasing its distensibility and allowing carnivorous and piscivorous birds to consume large food items. From a clinical perspective, the anatomy of the avian esophagus allows for easy introduction of instruments or endoscopes for foreign body removal from the esophagus, crop and proventriculus. In most birds, the esophagus is divided by the crop or ingluvies (some birds do not have a crop) into a cervical and a thoracic component.

In Galliformes and Falconiformes, the crop forms a ventral enlargement of the esophagus at the thoracic inlet. In Psittaciformes, the crop is stretched transversely across the neck. In canaries and ducks, the crop is absent, but there is a spindle-shaped swelling of the esophagus at the thoracic inlet. In pigeons, the ventral diverticulum of the esophagus that forms the crop is divided into two large lateral sacs. The esophagus is lined with partly kerat-

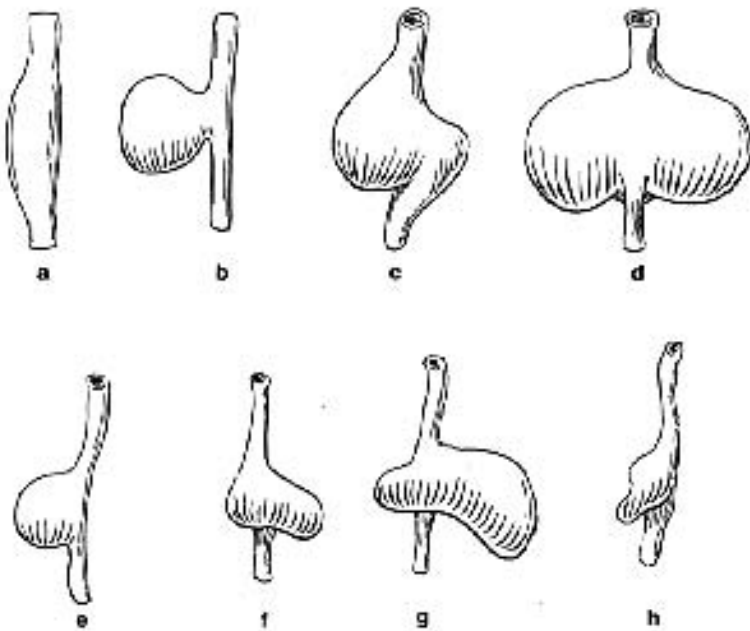


FIG 19.6 The shape of the crop varies dramatically depending on the species and the amount and shape of the ingesta. Some crop shapes include **a**) Great Cormorant, **b**) peafowl, **c**) budgerigar and **d**) domestic pigeon. **e-h**) Various shapes noted in a cockatoo. Modified in part from King and McLelland.¹⁰¹

inized stratified squamous epithelium. Mucous glands are located in the lamina propria, and are numerous in the thoracic esophagus. The structure of the crop resembles that of the esophagus, except that the mucous glands are restricted to an area adjacent to the esophagus. The function of the crop is to store food when the ventriculus is full (Figure 19.6). When the ventriculus is empty, food can bypass the crop and move directly into the proventriculus. The entire length of the esophagus can be used to store food in species that do not have a crop (eg, penguins and gulls). The esophagus and crop produce mucus, which softens and moisturizes the food in preparation for mechanical and chemical digestion lower in the gastrointestinal tract. Initial stages of carbohydrate digestion (mediated by salivary amylase) may occur in the crop of some species. The highly developed crop of the Hoatzin is unusual in having a large cervical component and two thoracic components. In this species, the ventriculus is small, and the muscular crop is the main site for the mechanical dissociation of food.

Adult pigeons of both genders produce a secretion called crop milk that is regurgitated and fed to the squabs during the first week after hatching. Other food items gradually replace the crop milk as the babies mature. Prolactin controls the production of crop milk, which consists of desquamated cells of the proliferated stratified squamous epithelium of the crop. Crop milk physically resembles mammalian milk and contains a high concentration of fat (6.9-12.7%) and protein (13.3-18.6%), but lacks carbohydrates and calcium. The crop of Psittaciformes and finches may also produce some secretions that are regurgitated and fed to neonates. The male Emperor Penguin feeds its chicks a fluid produced by desquamation of the esophageal epithelial cells, while the merocrine esophageal glands of both genders of Greater Flamingos produce a red nutritive juice that is regurgitated and fed to the young.

The crop of some birds may be involved in courtship behavior. Regurgitation is a common courtship behavior in some psittacine birds, particularly budgerigars, cockatiels and macaws. Males of some avian species (eg, pigeon, Great Bustard, ostrich, Sage Grouse) have inflatable esophageal diverticula that act as resonating chambers or display devices.

Investigative Methods

Clinical signs of esophageal or ingluvial disorders may include dysphagia, anorexia, retching, regurgi-



FIG 19.7 A two-year-old male budgerigar was presented with a three-week history of regurgitation. The bird was in overall good condition and would regurgitate when handled by either of the female members of the family. The bird would flick its head when it regurgitated, causing vomitus to land on the head feathers giving them a stiff, displaced appearance. Rhinorrhea can cause similarly appearing feather changes. The regurgitation in this bird was linked to courtship behavior.

tation or vomiting. In budgerigars, vomiting may be accompanied by a rapid flick of the beak, which frequently deposits vomitus on top of the bird's head (Figure 19.7).⁸ A history of recent drug administration, assisted feeding, poor hygiene or access to toxic compounds may suggest an etiology for esophageal problems.

The skin and feathers overlying the crop should be examined for abnormalities. Wetting of the feathers will help in visualizing lacerations, discolorations or necrosis of the crop. Hypermotility or hypomotility may occur with crop disorders.⁹⁶ In domestic fowl, peristaltic waves occur in the cervical esophagus at intervals of 15 seconds and in the thoracic esophagus, at intervals of about one minute. A psittacine crop that is partially filled with food should average one or two contractions per minute.

The esophagus and crop are thin-walled structures that are difficult to palpate unless abnormally thickened (Color 19.16). The crop can be palpated when it is full of food, fluid, air or abnormal masses. An enlarged crop with a dough-like consistency that fails to empty is suggestive of a crop impaction (Color 19.17). Occasionally, large deposits of fat, and in some cases lipomas, can occur near the crop and should not be misdiagnosed as a full or impacted crop. The use of improperly designed feeding cannulas to provide supportive nutrition to birds can result

in esophageal lacerations, with food being deposited into the subcutaneous, periesophageal tissues (see Color 30.8). Discolored necrotic areas, swelling and edema are common clinical findings.

For diagnostic purposes, an esophageal or ingluvial aspirate can be obtained by inserting a catheter and washing the mucosa with sterile isotonic saline solution. Luer-lock syringes should always be used when tube-feeding or collecting samples from psittacine birds to prevent them from swallowing the tube. Immediate microscopic examination of a wet mount slide is best for diagnosing trichomoniasis. Material aspirated from the crop should be centrifuged, and microscopic examination of the sediment may reveal nematode eggs or *Candida* spp. (see Color 30 and Color 10). Air-dried smears can be stained with Diff-Quik, Gram's stain, Wright's stain, Hemacolor or other stains for specific cytologic examination (see Chapter 10).

A fecal flotation and crop aspirate should be performed to detect parasite ova that might indicate an esophageal or ingluvial nematode or trematode infection. Flotation is more likely to detect low concentrations of eggs than a direct smear. Endoscopy is useful for examining the gastrointestinal mucosa and for removing some foreign bodies (Color 19.1).

Diseases of the Esophagus and Crop

Trichomoniasis

Trichomonas gallinae infections commonly occur in pigeons and raptors, and may also occur in Passeriformes (particularly canaries and Zebra Finches)¹¹⁵ and Psittaciformes (particularly budgerigars and cockatiels).^{8,61,137} In pigeons, the proliferative necrotic lesions caused by trichomoniasis are called "canker," while in falcons the disease is called "frounce." Trichomoniasis lesions appear similar to those caused by poxvirus; however, in cockatiels, poxvirus infections are uncommon.¹³⁷ Table 19.4 compares the clinical presentation of trichomoniasis in budgerigars and pigeons.

TABLE 19.4 Differences in Clinical Presentation of Trichomoniasis in Budgerigars and Pigeons

	Budgerigars	Pigeons
Anatomic Location	Esophagus, crop	Mouth, cloaca, umbilicus, liver, generalized
Lesions	Proliferative	Diphtheritic
Age	Mainly adults	Squabs

Samples for detecting *T. gallinae* can best be collected by introducing a slightly moistened cotton-tipped applicator into the esophagus, and moving it up and down several times against the mucosal lining. The cotton tip is then compressed between the thumb and index finger to produce one drop of fluid, which is placed on a slide for direct examination. A clinical diagnosis in Psittaciformes can be difficult because of low parasite numbers in the early stages of the disease¹³⁷ and the intracellular location of the parasite.⁸

Trichomoniasis can cause inflammation of the upper intestinal tract and mouth resulting in dysphagia or vomiting, and may be an underdiagnosed cause of ingluvitis in budgerigars. Historically, idiopathic vomiting, diarrhea and death with yellow caseous plaques and papilliform projections in the esophagus and crop have been defined in many budgerigars. These lesions resemble those caused by trichomoniasis.^{3,154} Trichomoniasis was documented in 70% of a group of budgerigars with clinical signs of vomiting and diarrhea.⁸ There was no gender predisposition, but the majority of cases occurred in birds from one to three years of age. The cervical part of the esophagus was most frequently affected, but many birds also had crop involvement. Histologic examination revealed that the increased thickness of the crop and esophagus was the result of an increase in depth of the mucosa. The intracellular occurrence of trichomoniasis has not been reported in other avian genera, and this unique feature of infection in budgerigars may contribute to the underdiagnosis of trichomoniasis as a cause of morbidity and mortality in Psittaciformes.

Trichomoniasis-induced ingluvitis and stomatitis have been reported in a cockatiel and budgerigar flock, respectively.^{138,162} Trichomoniasis was considered the cause for 80% morbidity and 72% mortality in a flock of 60 cockatiels exhibiting regurgitation, diarrhea and depression.²²² A large caseous mass in the distal trachea caused by *Trichomonas* sp. was reported in a ten-week-old Blue-fronted Amazon parrot.⁶¹

Trichomoniasis was diagnosed in neonatal cockatiels, Lilac-crowned Amazon Parrots, Mexican Red-headed Amazon Parrots, Sun Conures and Blue-crowned Conures that were fed from a single food source with three syringes. Clinical signs included necrotic dermatitis at the commissure of the beak, depression, crop stasis and white caseous plaques on the tongue and pharynx. Histopathology was negative in all cases. The diagnosis was made by identifying tricho-

Gastroenterology

Color 19.1

A >20-year-old Amazon parrot was presented with a six-month history of intermittent dyspnea that had become progressively worse. Survey radiographs indicated a soft tissue mass in the area of the caudal thoracic esophagus. Laparoscopy indicated a diffuse air sacculitis (that was not detected on radiographs), and the soft tissue mass was determined to be a dilated portion of the esophagus. Endoscopy of the trachea revealed thick mucus from which *Klebsiella* spp. and *Pseudomonas* spp. were recovered. The bird responded to systemic antibiotics but would relapse with the cessation of antibiotic therapy. Endoscopy of the esophagus revealed a large mass in the esophagus. Histologically, the surface of the mass contained hyperplastic mucus-producing cells. The etiology of the mass remains undetermined.

Color 19.2

An eight-year-old Sulphur-crested Cockatoo was presented with a five-week history of vomiting and progressive weight loss. Contrast radiographs indicated a mucosal filling defect in the proventriculus. Gastroscopy with a 3.5 mm flexible endoscope was performed. **a)** The opening from the crop into the thoracic esophagus is clearly visible. **b)** The opening from the proventriculus to the ventriculus is visible. Note the koilin layer (dark green areas), which can partially extend into the proventriculus. **c)** Bile-stained koilin layer of the ventriculus. **d)** Ulcers on the proventricular mucosa. Note barium sulfate is still adherent to some of the sites of ulceration. The bird did not respond to supportive therapy. Histopathology indicated marked myocardial degeneration and necrosis of undetermined etiology.

Color 19.3

A mature Scarlet Macaw was presented for intermittent regurgitation and weight loss. The bird was maintained in an outdoor environment and was frequently exposed

(every hour) to an automatic pesticide fogger. The bird had been in this environment off and on for several years. Abnormal clinical pathology findings included WBC=22,000, AST=750 and LDH=800. Radiographs indicated a rough appearance to the dorsal serosal surface of the proventriculus, suggestive of inflammation. The client chose to treat the bird at home with only antibiotics. The bird died several days after presentation. At necropsy, a large perforating ulcer was present in the proventriculus.

Color 19.4

Hemorrhagic, necrotizing, gram-negative bacterial colitis in an Umbrella Cockatoo with a chronic PBFV virus.

Color 19.5

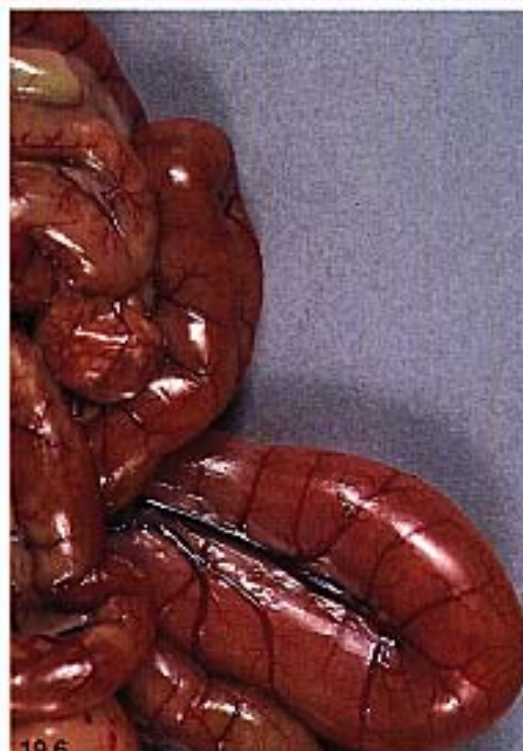
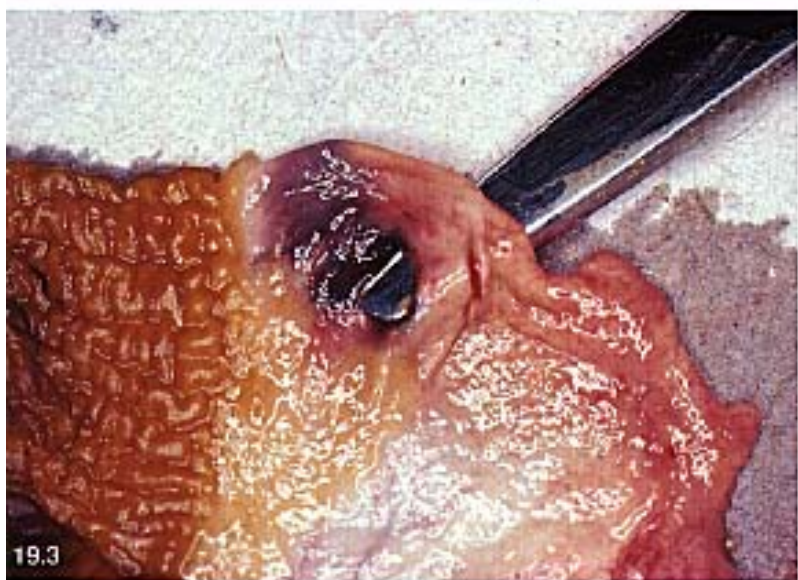
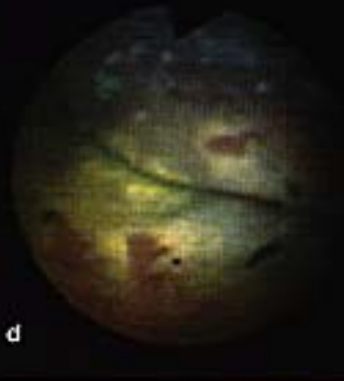
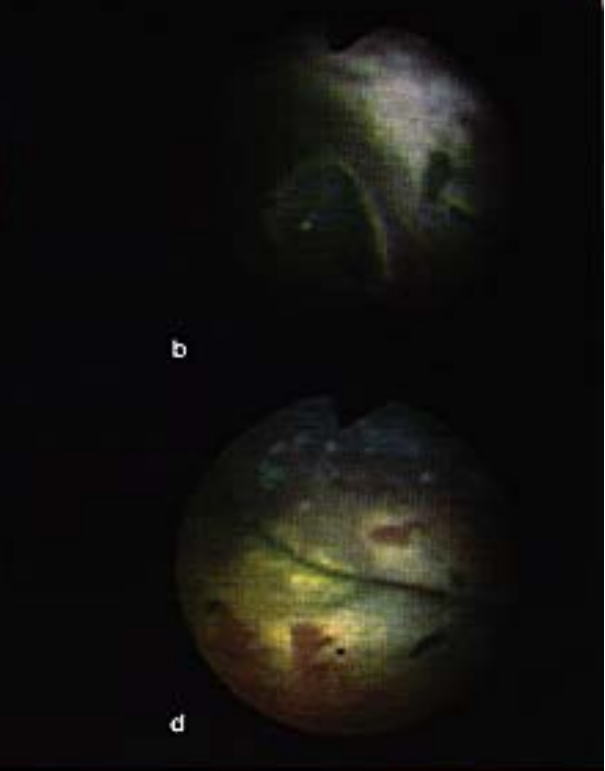
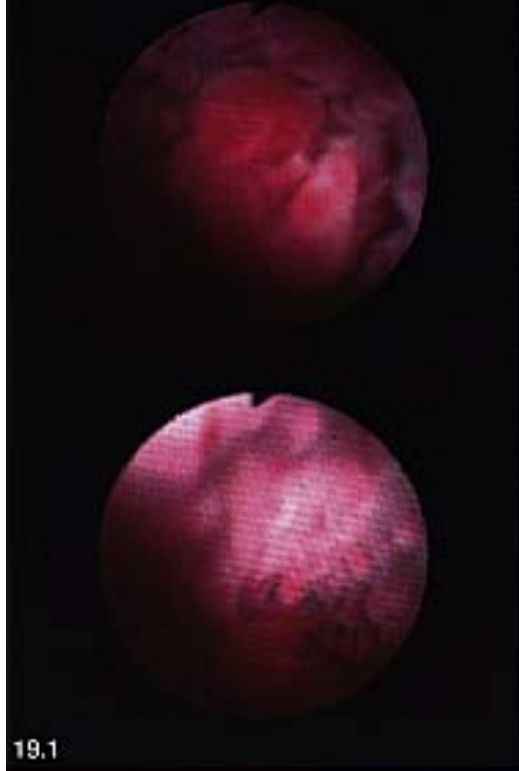
Several different nematodes can cause nodular-type lesions and mucosal hypertrophy in the proventriculus and ventriculus. In this case, proventricular hypertrophy was caused by *Dyspharynx* sp. in a crane that died following a period of weight loss, depression and anemia (courtesy of Robert E. Schmidt).

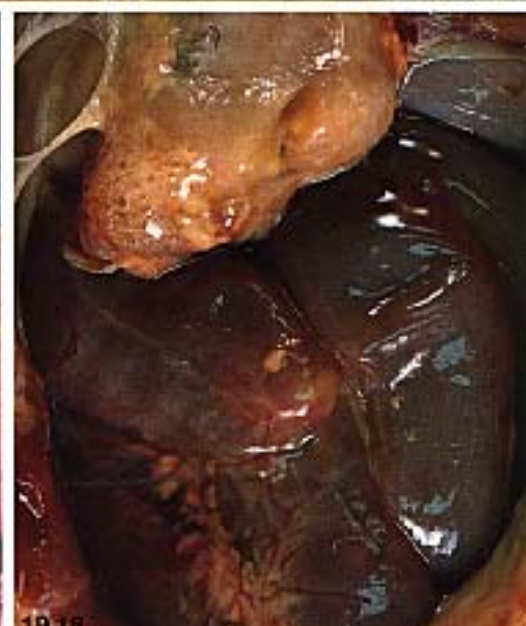
Color 19.6

A cockatoo with PBFV virus was found dead in its enclosure. The bird was in excellent overall condition and was of normal weight. Necropsy findings included hyperemia of the intestinal tract with distended vessels. Histopathology indicated a gram-negative bacterial enteritis and septicemia.

Color 19.7

A mature Yellow-collared Macaw hen died following a period of unseasonably low temperatures well below freezing. The bird had exhibited several days of depression before death. At necropsy, the cloaca and lower intestinal tract were filled with blood-tinged, poorly digested food. Histopathology indicated severe necrosis of the villi and crypt cells with a minimum inflammatory response. An etiologic agent was not identified.





Gastroenterology

Color 19.8

The pericloacal area of birds is normally dry and the feathers are of normal color. Accumulations of excrement or bile-stained feathers are indications of enteritis or cloacal dysfunction. In this case, a mature cockatiel had biliverdinuria from hepatopathy and enteritis associated with giardiasis.

Color 19.9

a) A cloacal papilloma appears as multiple, pink-to-red masses on the cloacal mucosa. Large lesions may protrude from the cloaca and be misidentified as a cloacal prolapse. Five percent acetic acid will cause papillomatous tissue to appear white while normal mucosa will stay pink. Stage cauterization with silver nitrate may be the easiest and most efficacious way to remove papillomatous tissue. Large masses should be removed with repeated treatments several weeks apart to prevent excessive damage to the cloacal mucosa. **b)** The silver nitrate stick is placed in direct contact with the papillomatous tissue and moved gently over the lesion. When the tissue turns grey, the silver nitrate is inactivated by flushing with water. The undiluted silver nitrate must not be allowed to run inside the cloaca, or nonspecific chemical burns will occur.

Color 19.10

A mature pigeon was presented for depression and anorexia of two days' duration. The feathers around the vent and lower abdomen were covered with excrement and fly larvae. The contaminated feathers were removed. The cloaca was impacted with dry, firm feces and urates. The feces were removed from the cloaca with forceps, and the cloaca was flushed with dilute chlorhexidine solution. A Gram's stain of the rostral cloacal wall revealed a moderate number of short, gram-negative bacteria. The bird responded to parenteral enrofloxacin.

Color 19.11

A female Amazon parrot was presented three days after egg laying when she was found on the bottom of the enclosure. The bird was depressed and emaciated. A cloacal prolapse had occurred, and the mucosa had multifocal, dry, necrotic areas. The necrotic tissues were cleansed with a dilute chlorhexidine solution and surgically removed. The cloaca was placed back in the abdomen and was held in place with a cloacopexy. The bird was placed on oral enrofloxacin, and the cloaca was flushed BID with dilute chlorhexidine solution followed by the installation of Preparation H. The bird responded to the therapy. The prolapse was considered to have occurred following the passage of an unusually large egg.

Color 19.12

A mature male budgerigar was presented with a three-week history of intermittent

regurgitation. Several masses (ingluvioliths) were palpated and were removed surgically from the crop.

Color 19.13

A mature budgerigar was presented for a swelling in the thoracic inlet area. The crop was severely distended with food, and the bird had an audible click on inspiration. Goiter was the presumptive diagnosis and the bird responded to iodine therapy (courtesy of Elizabeth Hillyer).

Color 19.14

A >25-year-old rosella died acutely. The gastrointestinal tract was dilated and contained poorly digested ingesta. The bird died from severe atherosclerosis.

Color 19.15

Typical "turkish towel" appearance of the crop in a bird with a severe *Candida* spp. ingluvititis. Compare to Color 19.16 in which the crop is transparent.

Color 19.16

In Psittaciformes, the normal crop and esophagus are thin, milk-colored, partially transparent membranes with a few distinguishable, small blood vessels. Note that the trachea can be seen through the crop.

Color 19.17

A 15-week-old Hyacinth Macaw was presented for necropsy following a brief period of depression. The client had noticed that the bird's crop had not emptied over a twelve-hour period. The bird was being fed a commercial hand-feeding formula supplemented with a liquid vitamin and mineral supplement. At necropsy, the crop was hyperemic and distended with food, and the vessels were congested. The kidneys were calcified. Macaws appear particularly sensitive to hypervitaminosis D₃. This bird's diet was estimated to have four to six times the necessary level of vitamin D₃.

Color 19.18

A 23-year-old Scarlet Macaw hen was presented two weeks after the referring veterinarian had surgically removed an impacted egg from the distal uterus. The bird was severely depressed and emaciated, and the abdomen was distended. Radiographs indicated gaseous distention of the intestinal tract. Abnormal clinical pathology findings included WBC=45,000 (toxic heterophils), PCV=25, TP=2.2 and AST=900. Fluid collected by abdominocentesis was characteristic of an exudate (SpGr=1.025, Protein=4.5 g/dl and numerous heterophils). An exploratory laparotomy was performed, a section of strangulated colon was removed and a side-to-side anastomosis was performed. The bird did not recover from the surgery. Note the congested, distended bowel loops and the presence of granulation tissue on the abdominal wall.

monas in impression smears of the lungs of a bird that died one hour before examination.¹³⁷

In another case, six Yellow-naped Amazon Parrot babies were affected. Clinical signs included whitish gray plaques on the tongue, choana and pharynx. Wet mount examination of scrapings from these lesions, crop lavage and transtracheal wash were negative. Postmortem examination of one of the Amazon parrots revealed a caseous plaque partially occluding the syrinx and lungs and filled with a yellowish, clear exudate containing abundant trichomonads that were demonstrated by a wet mount impression smear.¹³⁷ These cases suggest that trichomoniasis is a severely underdiagnosed cause of upper gastrointestinal disease in psittacine birds. Histopathologic examination is of limited diagnostic value because the causative organisms float away from the tissue in formalin.¹³⁷ Nitroimidazole drugs like metronidazole, ronidazole, dimetridazole and carnidazole are usually effective in treating trichomoniasis; however, nitroimidazole-resistant strains of trichomoniasis occur in The Netherlands because of the improper use of these drugs by pigeon fanciers.⁵⁶

Nematode and Trematode Infections

Many nematodes including *Capillaria* spp., *Echinura uncinata*, *Gongylonema ingluvicola* and *Dis-pharynx nasuata* can invade the esophageal or crop mucosa. The thorny-headed worm (*Oncicola canis*) has been reported in turkeys.^{78,174}

Ingluvial/Esophageal Stasis and Dilatation

The suggestive causes of crop stasis include heavy metal toxicity, crop impaction, callus formation after a coracoid fracture, thyroid enlargement, atonic crop, sour crop, overstretching of the crop, esophagitis (candidiasis, trichomoniasis, capillariasis, ser-ratospiciliasis), ingluvioliths and esophageal stenosis (Color 19.13). If the fluid in the crop remains stagnant, it will decay and have a foul odor (often referred to as sour crop). Regurgitation of proventricular fluid may be a contributing factor. Feeding a liquid formula to granivorous birds can induce crop stasis, possibly as a result of a lack of mechanical stimulation.

In turkeys, there is thought to be a hereditary predisposition to developing a pendulous crop after increased liquid intake during the first wave of seasonal hot weather. The majority of affected birds do not recover, but continue to have pendulous crops. It has been shown that feeding cerelose as a substitute for starch increases the incidence of pendulous crop

in gallinaceous birds. Affected birds had large quantities of the gas-producing yeast *Saccharomyces tel-lustris*. Large quantities of gas may have initiated the crop dilatation.¹⁵¹

Crop Impaction

Crop impaction is occasionally seen in Galliformes and Anseriformes that have sudden access to an abundant supply of lush grasses and sprouted grains.^{79,122} Dried oatmeal and soybeans swell when they absorb water and can impact the crop. This is a major cause of mortality in free-ranging Canada Geese.^{80,92} About 2000 Canada Geese died from impaction of the cervical esophagus accompanied by necrosis and ulceration of the esophageal mucosa following the ingestion of fox tail grass (*Setaria lutes-cens*). Lead poisoning, acute fowl cholera and ventricular worm infections can cause similar clinical signs.¹²² Crop impactions can occur in birds provided ad libitum grit. Food substances that are difficult to digest, such as raw potatoes, beets, apple skins, sausage skins and large pieces of animal tissues, may also cause crop impaction. In captive raptors, inglu-vial impaction may occur when roughage is suddenly added to a low roughage diet, or when the moisture content of the diet is inadequate. Crop impaction may be complicated by secondary *Clostridium per-fringens* infection in the European Kestrel.⁹¹

Impacted material in the crop can be softened by the administration of warm water followed by massaging the crop. However, an ingluviotomy will generally be the method of choice for removing impacted material. Foreign bodies may be removed endoscopically. Expressing the ingluvial contents through the mouth by turning the bird upside down is a dangerous procedure that may lead to irritation of the nasal mucosa, sinusitis or aspiration pneumonia. Packing the choana with cotton and intubating with an endotracheal tube will help eliminate this problem.

Ingluvioliths

Ingluvioliths have occasionally been reported in birds (Figure 19.8).^{3,10,12} Urate (excreta) calculi with seed husk centers were described in the crops of several budgerigars.¹² It was speculated that the calculi originated from the ingestion of excreta and seed husks in birds provided no food. Other ingluvioliths have been found to contain potassium phosphate, oxalate and cystine, and were not considered to have occurred secondary to urate ingestion (Color 19.12).³

Foreign Bodies

Unweaned psittacine neonates (especially macaws and Eclectus Parrots) frequently ingest foreign bodies.¹¹ Any ingested foreign body, including rubber or metal feeding tubes, should be removed immediately from the ingluvies before it has an opportunity to align with the thoracic esophagus and pass into the (pro)ventriculus. The type of foreign body is dependent on the species (Figure 19.9). Fishing hooks are common in waterfowl. Perforating or obstructing bones are encountered in raptors.

Crop and Esophageal Lacerations and Fistula

Penetration of the pharynx or esophagus by feeding cannulas, or esophageal-ingluvial burns caused by

ingestion of overheated feeding formulas or caustic materials can result in deposition of food subcutaneously and lead to extensive foreign body reactions. In birds of prey, sharp bones from prey animals may cause an esophageal or ingluvial fistula.^{20,153} Traumatic lacerations of the chest including the ingluvies are often seen in racing pigeons. The most likely cause for these lacerations is a collision with antenna wires. Bite wounds from mates or cats and dogs may also cause crop lacerations. A bird with a crop fistula may be presented with weight loss despite a ravenous appetite. The feathers surrounding the fistula are usually matted with dried food. Subcutaneous pockets of food should be surgically drained and frequently flushed. A feeding tube can be passed from the esophagus directly into the proventriculus to allow enteral feeding while the esophagus and crop heal (see Chapters 15, 16 and 41).⁸¹

Esophageal Stricture

A ten-year-old Hyacinth Macaw developed a stricture in the thoracic esophagus after ingesting several large pieces of hard plastic. The most obvious clinical sign was regurgitation of mucus. A barium study revealed a filling defect in the caudal esophagus. Endoscopic examination through an ingluvial incision revealed an annular ring of exudate and hyperplastic tissue. The stricture was resolved by periodic mechanical dilation.²⁰³ Esophageal strictures may also occur secondary to burns. A cockatoo with severe self-mutilation syndrome damaged the perieso-



FIG 19.8 An ingluviolith is evident in these radiographs of a cockatiel (courtesy of Jean Paré).

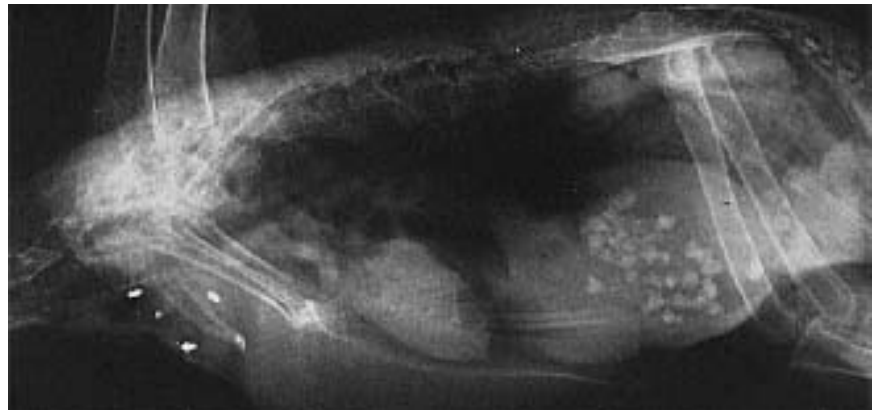


FIG 19.9 An adult female Umbrella Cockatoo was presented with an acute onset of lethargy and regurgitation. Several small solid masses could be palpated in the crop. The bird was producing small amounts of excrement and had tenesmus. The bird was maintained in an earthen-floored flight enclosure and was fed wild bird seeds. Abnormal clinical pathology findings were limited to hypokalemia and mild anemia (PCV=34%). Radiographs indicated several metallic densities in the crop. In addition, an excessive number of mineral densities (small rocks, grit) were present in the ventriculus and intestinal tract. The metallic densities (pieces of wire) were removed using an endoscope and forceps. The ventriculus was flushed with copious amounts of warmed LRS, which resulted in the removal of about 20 to 30 small rocks. The bird was given corn oil by crop tube three times a day for three days, and rocks were noted in the stool on the day after the first corn oil treatment. The bird's diet was corrected and no further problems were noted.

phageal skin to such a degree that the esophagus was occluded and the bird died from asphyxiation.

The Proventriculus and Ventriculus

Anatomy and Physiology^{55,101}

The avian stomach consists of a cranial glandular part (proventriculus) and a caudal muscular part (ventriculus). The proventriculus in birds is situated in the left dorsal and left ventral regions of the thoraco-abdominal cavity, and is covered ventrally by the fat-laden posthepatic septum (see Color 14). The pyloric part of the ventriculus joins the duodenum and is located on the right side of the midline. In granivorous, insectivorous and herbivorous birds, the muscular wall of the ventriculus is highly developed and is clearly distinct from the proventriculus. The two organs are divided by an intermediate zone, or isthmus, which can be seen grossly as a constrictive band (Figure 19.10). The ventriculus can be palpated in granivorous birds on the left ventral side of the abdomen just caudal to the sternum.

The proventriculus has two types of glandular epithelial cells. Mucus-producing, columnar epithelial cells line the proventricular mucosa and the lumina of the ducts from the proventricular glands. These cells contain numerous periodic acid Schiff (PAS)-positive mucin granules. The multilobular glands of the proventriculus are lined by oxynticopeptic cells. These cells have ultrastructural features similar to both the parietal (acid-secreting) and the peptic (enzyme-secreting) cells of the mammalian stomach, and secrete both pepsinogen and hydrochloric acid. The ducts from these glands empty from numerous papillae that can be found in the proventricular wall. These oxynticopeptic cells possess a very eosinophilic and somewhat granular cytoplasm and do not contain PAS-positive mucin granules.

The wall of the ventriculus is composed of smooth muscle arranged into four semi-autonomous masses. The caudodorsal and cranioventral thick muscles and the craniodorsal and caudoventral thin muscles attach to the right and left tendinous centers in the lateral walls of the ventriculus. The asymmetric arrangement of the muscles enables rotary and crush-



FIG 19.10 The isthmus (arrow) is distinguished as a constrictive band separating the proventriculus (p) from the ventriculus (v). The proventriculus can be seen laying dorsal to the caudal thoracic air sac. This bird died from myocardial degeneration.

ing movements during ventricular contractions. The thin muscles form the cranial and caudal blind sacs. The caudal blind sac is a good point for gastrotomy incision in granivorous birds because incisions in the thick muscles heal poorly. The inner surface of the ventriculus of granivorous birds is lined by a carbohydrate-protein complex (koilin layer or cuticle). This koilin layer is composed of vertical rods secreted by the mucosal glands of the lamina propria and a horizontal matrix, which is a secretion of the surface epithelium that hardens after spreading around the rods. Desquamated cells of the surface epithelium are trapped within the horizontal matrix. Hydrochloric acid from the proventriculus causes precipitation of the protein complex to form a tough, water-resistant lining. The brown, green or yellow color of the koilin is caused by regurgitation of bile through the pylorus.

The intermediate zone that divides the proventriculus and ventriculus has histologic characteristics

similar to both organs. Compound glands are absent, and the internal surface is relatively smooth. The columnar epithelium lining the proventriculus gradually changes into ventricular glands. There is a mixture of proventricular-like mucoid secretions and ventricular-like glandular secretions. Most gastric neoplasms in birds occur in the intermediate zone, and use of PAS stains appears useful in determining the types of epithelial cells that have been transformed.^{113,161}

The ventriculus is the site of gastric proteolysis, and in many species also of mechanical digestion. In carnivorous and piscivorous birds, the proventriculus is more adapted for storage than for physical digestion. In these species, the ventriculus is thin-walled and sac-like, and the ventriculus and proventriculus are difficult to differentiate grossly. The koilin layer is relatively thin and softer than in granivorous birds. The muscularis is relatively thin. In raptorial species (eg, owls), the ventriculus is involved in the formation and regurgitation of pellets or “castings,” which are composed of undigestible fur, feathers or bones. Intermediate forms of proventricular and ventricular differentiation are found in many avian species including frugivorous (fruit-eating) and testacivorous (shellfish-eating) birds.

In certain frugivorous pigeons, the koilin layer is composed of rows of hard, pointed, conical projections that facilitate crushing firm fruits such as nutmeg. In some species (magpie and starling), massive shedding and excretion of the koilin layer occur periodically. Male hornbills may regurgitate the koilin layer as a seed-filled sac that is fed to the nesting female.

The proventriculus in an ostrich is a large, thin-walled structure. In contrast to other birds, the oxynticopeptic cells are restricted to a patch on the greater curvature. The distal extremity of the ostrich proventriculus passes dorsal to the ventriculus and empties on the caudal aspect of this organ. The ostrich ventriculus is a thick-walled muscular organ. The isthmus between the proventriculus and ventriculus is large, which makes it easy to remove foreign bodies from the ventriculus through a proventricular incision. In emus and nandus, the proventriculus is large and spindle-shaped, and the ventriculus is slightly larger and more lightly muscled than that of the ostrich.

Proventricular and Ventricular Diseases

Most diseases of the proventriculus or ventriculus produce similar clinical signs and make differentiation difficult. For example, an enlarged proventriculus may be found in many of the diseases in Table 19.1. The following is a discussion of some of the diseases that affect the proventriculus and ventriculus.

Megabacterial Proventriculitis

“Going light” syndrome in budgerigars, a disease characterized by emaciation, weakness, high morbidity and low mortality, has been described in canaries and budgerigars. Vomiting of slimy material is seen in advanced stages of the disease.^{64,177,197,201} Postmortem findings include proventriculitis and proventricular dilatation. Histologically, gram-positive, PAS-positive, acidophilic (with Giemsa), rod-shaped bacteria can be identified, especially in the area between the proventriculus and ventriculus. The organisms have been characterized as large bacteria (not fungi), hence the name “megabacteria” (see Figure 33.13).

A diagnosis can be made by cytologic demonstration of the organisms in a proventricular washing. The pH of the proventriculus is markedly elevated in affected birds. The pH of the proventriculus from normal canaries was found to range from 0.7 to 2.4 compared to severely infected canaries in which the pH was 7.0 to 7.3. In birds with moderate numbers of megabacteria, the pH of the proventriculus ranged from 1.0 to 2.0.²⁰¹ The most important differential diagnosis is trichomoniasis.

Proventricular and Ventricular Nematodes

Many nematode species have been reported to occur in the proventriculus (*Echinura uncinata*, *Gongylonema ingluvicola*, *Cyrnea* spp., *Dyspharynx nasuta* and *Tetrameres* spp.). *Amidostomum* spp., *Cheilospirura spinosa* and *Epomidiostomum uncinatum* are found under the horny layer of the ventriculus (Color 19.5).^{60,174,179} Lesions vary considerably depending on the host and the parasite, and may be quite extensive (see Chapter 36). Clinical signs may be absent or include emaciation, anemia and mortality. Diagnosis can often be made by detecting parasite eggs using a fecal flotation technique. Treatment can be attempted with levamisole (20 mg/kg orally, or 10 mg/kg parenterally) or ivermectin (200 µg/kg parenterally). It should be stressed that experience with ivermectin in many avian species is absent. Acute death has been reported after the use of ivermectin in some mammalian and reptilian species.

Neuropathic Gastric Dilatation and Encephalomyelitis of Psittacines

Neuropathic gastric dilatation (NGD)¹²³ has been given many names since it was first recognized as a clinical entity in 1971 (Table 19.5).

The descriptive term myenteric ganglioneuritis and encephalomyelitis of Psittaciformes best defines the following histologic lesions that can be observed in affected animals: lymphocytic and monocytic infiltration of intrinsic and extrinsic splanchnic nerves of the muscularis tunics of the alimentary tract; in some cases, leiomyositis in organs innervated by affected nerves and non-suppurative encephalitis, myelitis and radiculoneuritis have been described.⁶⁸ Many psittacine species can be affected, including macaws, cockatoos, conures, African Grey Parrots, Senegal Parrots, Amazon parrots, Eclectus Parrots, Thick-billed Parrots and cockatiels.⁶⁸

Clinical signs are related to (pro)ventricular and sometimes neurologic dysfunction, and may include anorexia, regurgitation, undigested seeds in the feces and weight loss. The occurrence of neurologic signs is variable. In advanced cases, proventricular dilatation can be visualized on abdominal radiographs, with or without contrast media (Figure 19.11). In the clinical patient, a tentative diagnosis of NGD can be made based on clinical signs and radiographic findings. It should be stressed that other diseases can mimic NGD and should be ruled out before a definitive diagnosis is considered (Table 19.6).^{69,90,197} NGD

TABLE 19.5 Synonyms for Neuropathic Gastric Dilatation*

- Psittacine proventricular dilatation syndrome (PPDS)
- Macaw wasting syndrome
- Myenteric ganglioneuritis
- Psittacine encephalomyelitis
- Proventricular dilatation of psittacines
- Proventricular dilatation and wasting syndrome
- Myenteric ganglioneuritis and encephalomyelitis of psittacines
- Wasting/proventricular dilatation disease (WPDD)
- Proventricular dilatation disease
- Proventricular dilatation syndrome
- Macaw fading syndrome

* References 37,45,68,89,94,121,123,157,167,172,195,221

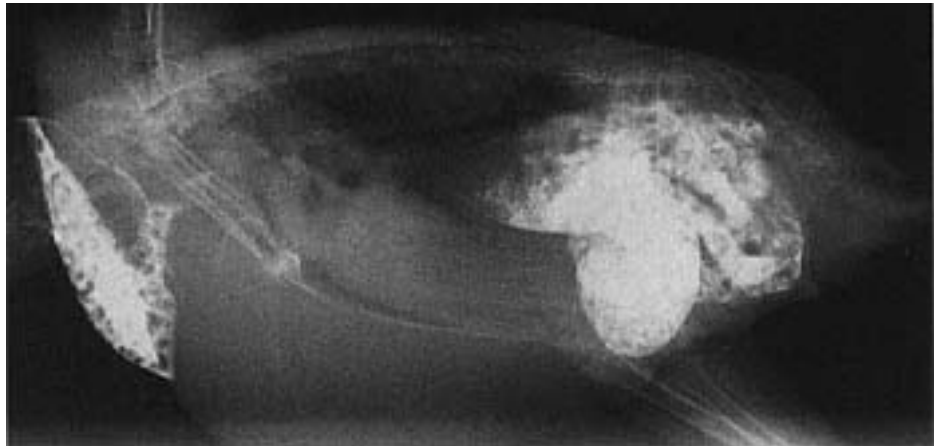


FIG 19.11 A three-year-old female cockatiel was presented with a two-week history of passing undigested seeds. Survey radiographs suggested a dilated proventriculus. Barium contrast study indicated a slowed gastric emptying time (VD and lateral radiographs eight hours after the administration of barium by crop tube). Note the numerous filling defects (ingesta and undigested seeds) in the crop, proventriculus and intestines. The client chose to have the bird euthanized. Histopathology confirmed a diagnosis of neuropathic gastric dilatation.

TABLE 19.6 Differential Diagnosis of Neuropathic Gastric Dilatation

- Heavy metal poisoning
- Fungal infection of ventriculus or koilin mycosis
- Nematode infection of the (pro)ventriculus
- Megabacteria infection of the proventriculus
- Gastric impaction
- Pyloric obstruction by a foreign body
- Ventriculus perforation by a foreign body
- Myoventricular dysgenesis
- Proventricular foreign body
- Koilin dysgenesis
- Myoventricular calcinosis
- Intestinal papillomatosis
- Proventricular and ventricular neoplasia
- Vitamin E and selenium deficiencies

can be confirmed by histologic identification of characteristic lesions in the splanchnic nerves from a ventricular biopsy (Figure 19.12). However, a negative result derived from a small biopsy of the ventricular wall does not rule out NGD.⁶⁸

Although the etiology is presently unclear, a viral etiology has been suggested because of epidemiologic histories of affected aviaries and the demonstration of intranuclear and intracytoplasmic inclusion bodies in affected tissue of some birds.^{26,63,84,123} Paramyxovirus-like inclusions have been described by some researchers¹²³ but could not be identified by others.⁶⁸ The “avian viral serositis” virus produces experimental lesions that are similar to those described with NGD (see Color 32).

Affected animals invariably die after a more or less protracted course of the disease. Controlled studies to document the effects of treatment on the prognosis of NGD have not been reported. General therapy including supportive care, a liquid diet, vitamin supplementation and treatment of secondary diseases has been recommended. It has been suggested that birds can survive on a liquid diet,⁷⁸ but no case reports could be found that document long-term survival of birds confirmed by ventricular biopsy to have NGD. Treatment of NGD should be considered with caution, given that the disease may be caused by an infectious agent.

It has been suggested that a virus may induce an autoimmune reaction that would be responsible for the lesions observed in NGD.⁶⁷ The inciting virus would no longer be present when the disease became clinically obvious or was diagnosed at necropsy. If this scenario is true, then administration of an anti-

inflammatory dose of corticosteroids might be indicated.¹⁶¹

Ganglioneuritis and Encephalitis in Geese

Proventricular impaction with non-suppurative encephalomyelitis and ganglioneuritis morphologically similar to NGD in psittacine birds has been reported in two Canada Geese.⁴³

Proventriculitis in Chickens

Proventricular enlargement and infiltration of the proventricular interglandular tissue with mononuclear cells has been described¹⁰⁶ as part of the infectious runting and stunting syndrome in broiler chickens.

Gastric Impaction and Gastric Foreign Bodies

Gastric impaction is common in psittacine babies that consume bedding material such as crushed walnut shell, ground corncob, shredded paper pulp, styrofoam packing, kitty litter and excess grit. These bedding materials should not be used with neonates.^{35,39}

Ventricular impaction secondary to litter ingestion has been reported to cause high mortality during the first three weeks of life in turkey poults. Affected animals were emaciated and had empty intestinal tracts, distended impacted ventriculi and foreign bodies in the first part of the duodenum.¹⁷⁰

Gastric obstruction caused by phytobezoars or detachment of the koilin layer of the ventriculus has been reported in chickens, a goose, ducks and a pigeon.⁵¹ Gastric rupture may occur secondary to an impaction. In raptors, ventricular impaction caused by a hard mass of fur can also occur. Gastric impaction is more common in captive raptors fed early during the day in hot weather. Those fed late in the day are less likely to retain their casting.⁶⁶

In ostriches, (pro)ventricular impaction is a serious management problem.²⁰⁹ The condition occurs usually two to three weeks after moving the birds to a new environment containing a novel substrate or food. Most affected birds are under six months of age, but the condition can also occur in adults. Clinically the birds are sluggish and produce small, firm fecal balls. Some affected birds may be lame. Cloacal prolapse may occur in young birds several days to four weeks old. Early diagnosis through abdominal palpation and radiographs followed by immediate surgical correction are imperative for successful resolution.^{88,187} A 17-inch string of beads could be palpated

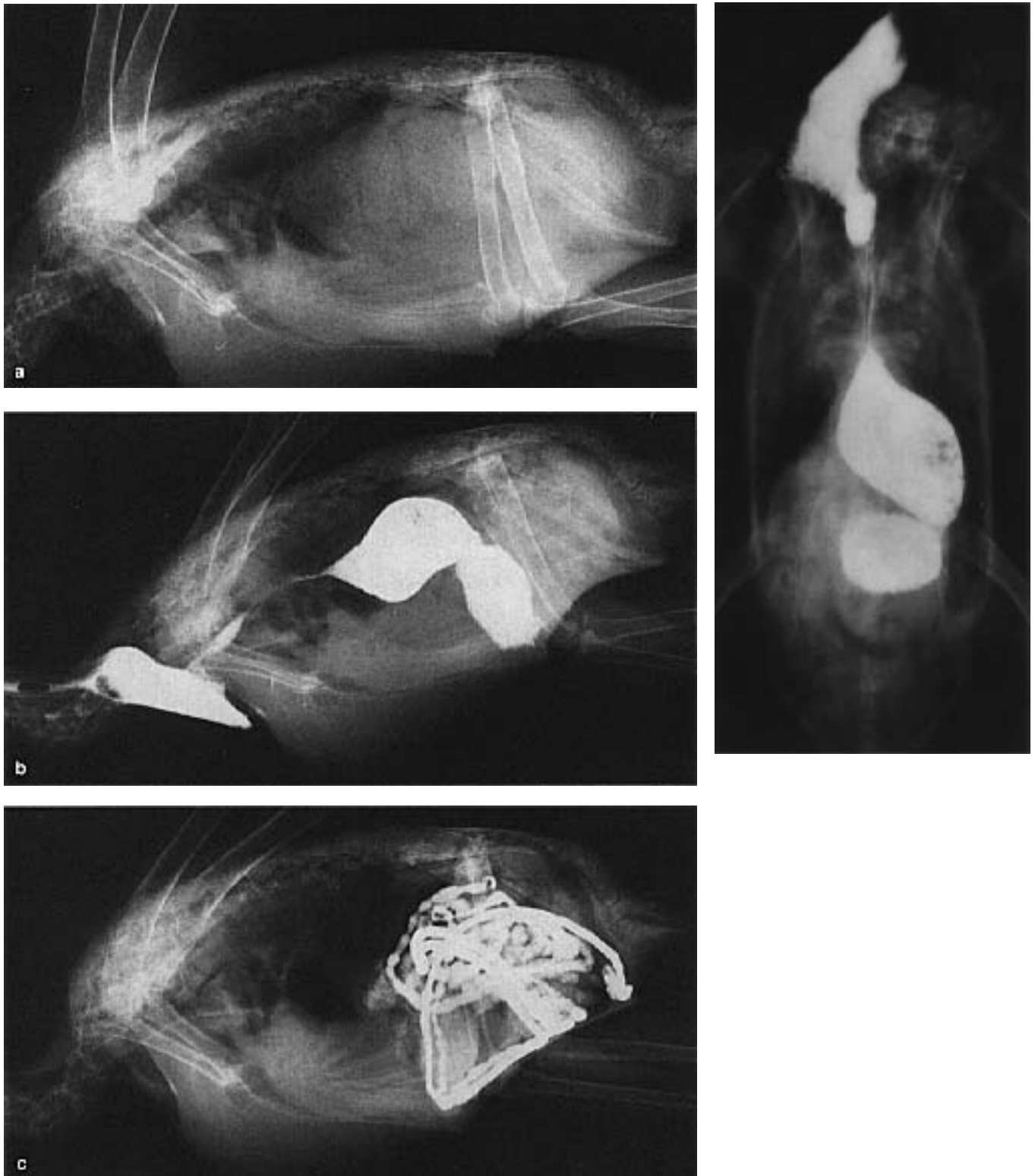


FIG 19.12 A one-year-old male Eclectus Parrot was presented for severe emaciation, depression, diarrhea and a distended abdomen. The bird had a ravenous appetite. Abnormal clinical pathology findings included AST=1637, LDH=4049 and WBC=3000. **a)** Survey radiographs indicated a severely dilated proventriculus and hepatomegaly. The bird was fasted for eight hours and a barium contrast study was performed. **b)** Ventrodorsal and lateral radiographs were taken two hours post-barium administration and **c)** lateral radiograph represents eight hours post-barium administration. The proventriculus was markedly reduced in size following fasting. Contrast studies indicated a delayed gastric-emptying time, hepatomegaly (causing the proventriculus to be displaced dorsally) and retention of barium in the proventriculus, suggestive of mucosal inflammation. The bird responded to treatment with broad-spectrum antibiotics and lactulose. It should be stressed that the survey radiographs were suggestive of NGD; however, a dilated proventriculus and slowed gastric-emptying time are not pathognomonic for NGD. A biopsy is necessary to confirm a diagnosis.

in the crop of an Amazon parrot. Barium contrast radiographs were used to confirm that the string of beads passed into the small intestines. The string was extracted through the mouth using sponge forceps.²¹³

Although poorly documented in birds, emetics might be useful to remove foreign bodies that would not damage the gastrointestinal mucosa during the regurgitation process. Apomorphine induced emesis in 55% of treated birds³⁶ and in another study, a 0.5% solution of tartar emetic was effective.¹¹⁴

Metallic Foreign Bodies: Traumatic Gastritis, Heavy Metal Poisoning

Ingestion of metallic foreign bodies is relatively common in Galliformes, Anseriformes, Columbiformes, Gruiformes, Pelecaniformes, Psittaciformes and ratites.^{51,71,205,209,210} In captive Psittaciformes and free-ranging Anseriformes, ingestion of lead is extremely common. Paralysis of the intestinal tract from nerve damage may occur secondary to lead poisoning. In Anseriformes, this is clinically recognized as esophageal and proventricular dilatation. In the other orders, ingestion of ferrous metal objects, such as nails, wire, hairpins and needles, account for the majority of cases. This is particularly common in gallinaceous birds.¹⁵¹ In one case, each affected bird had a piece of one-inch wire penetrating the ventriculus (see Figure 45.2). It was discovered that the owner used a wire brush to clean the water container.

Ingestion of ferrous objects may cause perforation of the ventriculus (majority of cases) or proventriculus, leading to an acute, generalized, purulent peritonitis or to a local peritonitis with abscess formation on the serosal surface of the (pro)ventriculus or duodenum. The powerful contractions of the ventriculus muscles in the domestic fowl can result in a pressure of 100-200 mmHg, which can easily force sharp objects through the tough muscular wall. Occasionally, penetration of a large (hepatic) artery or vein can result in fatal hemorrhage. In some rare cases, the ferrous foreign body will be resorbed by the inflammatory reaction without permanent deleterious effects. Cases have been documented in which a foreign body

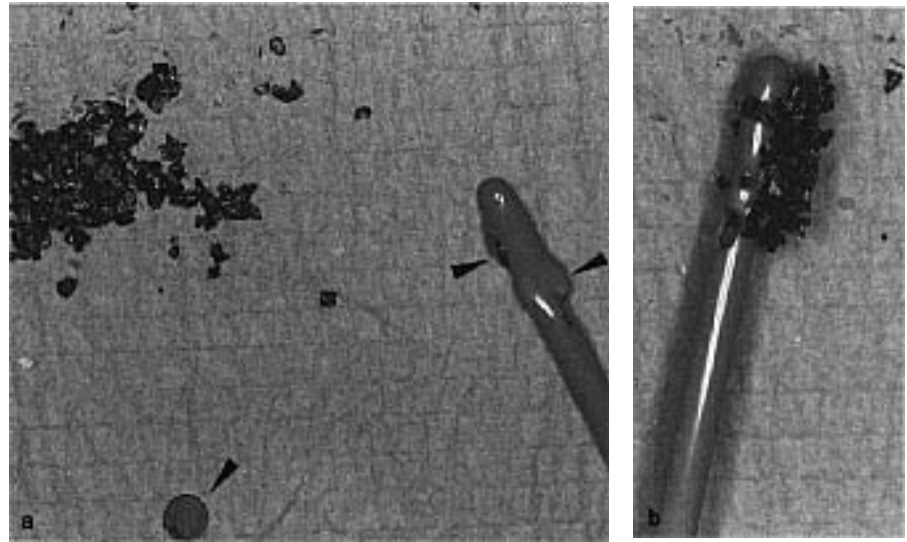


FIG 19.13 A mature African Grey Parrot was presented for evaluation of depression and anorexia several days after destroying a panel of chicken wire placed over a screened-in-porch. Radiographs indicated that the ventriculus was full of metallic densities. Because the wire was composed of a ferrous metal, **a**) the foreign bodies were removed by repeatedly inserting a red rubber feeding catheter equipped with several neodymium-ferro-borium alloy magnets (arrows). **b**) The metal removed from the bird is firmly attached to the magnet contained within the tube.

was exteriorized by penetration through the body wall.^{51,205}

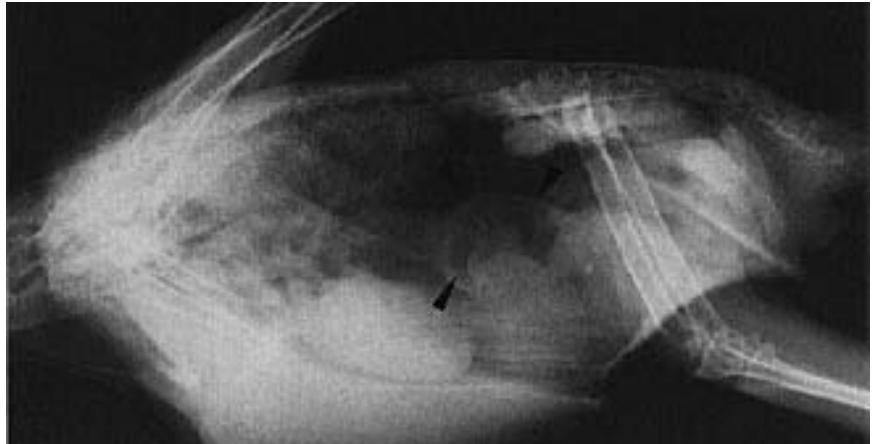
Foreign body penetration of the ventricular wall causes a decrease in ventricular contraction and an insufficient digestion of food, which may be recognized clinically by the passage of undigested seeds in the feces. In the racing pigeon, passing undigested seed is considered pathognomonic for a traumatic gastritis. In chronic cases, anorexia, weight loss and a palpable abscess on the left side of the abdominal wall may be noted.²⁰⁵ Radiology is the method of choice to confirm a tentative diagnosis.

Noninvasive treatments for removal of gastric metal foreign bodies should be attempted before (pro)ventriculotomy. Ferrous metals may be removed from the (pro)ventriculus using a powerful magnet of neodymium-ferro-borium alloy (The Magnet Store 1-800-222-7846) attached to a small-diameter polyvinyl catheter with a removable steel guide wire (Figure 19.13).¹²⁰ The size of the polyvinyl probe and magnetic disk can be varied according to the size of the animal. A probe with a length of two meters, a diameter of 18 mm and an attached cylinder magnet of 17 x 70 mm (derived from a bovine cage magnet) has been used to remove thirteen large staples from the ventriculus of an ostrich. Fluoroscopy or endoscopy can be used to guide grasping forceps in the

removal of gastric foreign bodies.^{117,119} Most cases of lead and zinc ingestion can be managed medically and do not require surgery.

Myoventricular Dysgenesis

Proventricular dilatation secondary to ventricular abnormalities caused by feeding finely ground food low in fiber is commonly observed as an incidental finding in chickens. The enlarged proventriculus has a distended thin wall and is full of food. The ventriculus in affected birds is poorly developed, and there is no sharp demarcation between the proventriculus and ventriculus.¹⁶⁹ The postmortem findings are similar to those with pyloric obstruction or gastroesophageal paralysis (ie, dilatation of the food-filled ventriculus, proventriculus, esophagus and crop). In the latter condition, the ventricular muscles are of normal thickness.



Vitamin E and Selenium Deficiencies

Vitamin E and selenium deficiencies may cause degenerative lesions in the smooth muscle of the ventriculus of domestic and free-ranging Anseriformes.^{135,178,219}

Gastric Ulceration, Ventriculus Erosion, Gastritis, Koilin Dysgenesis

Idiopathic proventricular ulcers do occur in psittacine birds. Many affected animals are “high strung” or in what could be called stressful environments (Figure 19.14); (Color 19.3).

High dietary levels of certain types of fish meal or finely ground, low-fiber diets can cause erosions and ulcers in the koilin layer of gallinaceous birds.^{71,144} Infectious and parasitic agents may damage the ventricular wall causing dysfunction. Penetration of foreign bodies may cause localized lesions. Zoalene (DOT) toxicosis may cause gastric erosion.⁷¹ Ventriculus erosion with a heavy infiltration of heterophils has been reported with zinc poisoning.^{46,48,216}

Copper Poisoning

Excessive dietary copper leads to roughening and thickening of the koilin layer. The marked thickening and folding may have a wart-like appearance. Hemorrhages may be seen under the koilin layer.^{53,216}



FIG 19.14 An eight-year-old Sulphur-crested Cockatoo was presented with a five-week history of vomiting and progressive weight loss. Survey radiographs indicated gaseous distention of the proventriculus (arrows). Contrast radiographs indicated mucosal filling defects in the proventriculus. Gastroscopy with a 3.5 mm flexible endoscope indicated numerous proventricular ulcers (Color 19.2). The bird did not respond to supportive therapy. Histopathology indicated marked myocardial degeneration and necrosis of undetermined etiology.

Neoplasias^{2,68,74,113,161}

The clinical effects of (pro)ventricular neoplasia may vary, depending on the size of the tumor and the presence or absence of active bleeding from the ulcerated tumor. Clinical signs may include weight loss, vomiting, passing of whole seeds in the feces, regenerative anemia, hypoproteinemia and melena. Although hypoproteinemia may occur, the albumin/globulin ratio is not affected, which together with the anemia and melena is strongly indicative of gastrointestinal blood loss (Table 19.7). Death usually ensues when massive gastric bleeding occurs following erosion of a major vessel. Contrast radiography using both positive and negative contrast may be helpful in outlining the (pro)ventricular neoplasm.¹⁹⁶ Endoscopic-guided biopsy may be used to confirm a tentative diagnosis.

Tumors are frequently located at the isthmus on the boundary between the proventriculus and ventriculus. Gross lesions in the (pro)ventriculus may be subtle, and histologic examination is needed to differentiate tumors from other causes of ulceration or hypertrophy. The use of specific staining methods (Alcian blue and periodic acid Schiff) facilitates differentiation between tumors of proventricular and ventricular origin.^{113,161}

Presently, no reports of successful treatment of (pro)ventricular tumors have been published, but it has been suggested that early diagnosis and surgical excision are feasible.¹⁶¹

TABLE 19.7 Documented Cases of Gastrointestinal Neoplasias in Psittaciformes (P), Galliformes (G) and Other (O).

	Adenoma/ Adenocarcinoma ⁵⁷	Basal Cell Carcinoma	Fibroma/ Fibrosarcoma	Hemangiosarcoma	Histiocytic Sarcoma ^{86, 71}	Islet Cell Carcinoma	Leiomyoma/ Leiomyosarcoma ¹⁵⁶	Lymphangioma	Myxoma/ Myxosarcoma	Papillomas ^{71, 129, 69, 112, 200, 145}	Sarcoma	Spindle Cell Carcinoma ⁷¹	Squamous Cell Carcinoma ^{18, 131, 139, 163, 74, 74, 196, 94, 80}
Rhamphotheca	P	P	P								P	P	
Oropharynx			P		G,P					P	G	G	G,P
Esophagus	G				G					G,P			G,P,O
Crop	G						P			G,P			
Proventriculus	G,P,O			G						P		G	
Ventriculus	G,P,O		G	G	G		G			P	G		
Intestines	G,P		G,P	G			G,P	G	G			G	
Rectum	O		G										
Cloaca	G,P,O		P							P	P		
Pancreas	G					G					G		

Squamous cell carcinomas are the most common tumor in the oral cavity of Galliformes. Oropharyngeal tumors may be painful, ulcerative and infiltrative but rarely metastasize. Clinical signs may include dysphagia, regurgitation, vomiting, diarrhea, tenesmus and cloacal prolapse. A definitive diagnosis can be made by biopsy. Prognosis depends on the location of the tumor, the degree of tissue infiltration and the occurrence of metastasis. Many connective tissue tumors in gallinaceous birds may be caused by the leucosis sarcoma group of viral infections.¹⁴⁹ There is a high incidence of pharyngeal and esophageal squamous cell carcinoma in humans and chickens in northern China suggesting a common etiology.^{34, 180} The incidence of gastric neoplasia seems higher in psittacine birds compared to otherspecies. Intestinal tumors must be differentiated from metastatic oviductal or ovarian tumors by demonstrating that no primary tumors of these organs exist and that the tumor originates from intestinal mucosal epithelium or glands, rather than growing inward from the serosal surface.²⁹ Obstruction of the pancreatic ducts can occur with pancreatic neoplasias. Islet cell carcinomas can cause diabetes mellitus.^{74, 196}

The Small and Large Intestines

■ Anatomy and Physiology¹⁰¹

In the majority of avian species, the duodenum is a narrow, U-shaped organ that originates from the pylorus on the right side of the ventriculus. The pale pink or yellow lobulated pancreas is located between the proximal descending and distal ascending duodenal loops (see Color 14). In some species (eg, White-tailed Sea Eagle and Jackass Penguin), secondary folds are present in the duodenum. In the Black Stork, the duodenum is twisted, while in other species (eg, Northern Fulmar and Gannet), the duodenum consists of more than one loop. The bile and pancreatic ducts often open near each other at the distal end of the duodenum. There are one, two (ducks and geese) or three (domestic fowl) pancreatic ducts and two bile ducts. When a gallbladder is present, this organ drains the right liver lobe via the right hepatocystic duct and empties into the duodenum via the cysticoenteric duct. In species where a gallbladder is absent (most pigeons, many parrots and the ostrich), the right liver lobe drains directly into the duodenum via the right hepatoenteric duct. In gallinaceous birds, the common hepatoenteric duct drains bile from both liver lobes to the duodenum. The jejunum and ileum are arranged in a number of loops, and are suspended by a long, distal mesentery on the right side of the abdominal cavity.

The vitelline diverticulum (Meckel's diverticulum), which is the remnant of the yolk duct, is located opposite the distal end of the cranial mesenteric artery. The duct of the yolk sac opens into the small intestine on a small papilla. The yolk provides nourishment, minerals, fat-soluble vitamins and maternal immunoglobulins to the embryonic bird and to the chick during the first few days of life. Just before hatching, the yolk sac is internalized and the umbilicus is closed. Precocial birds have a yolk sac that ranges from 10 to 25% of body weight at hatching, while in altricial species these values range from 5 to 10% of body weight.⁹⁹ Precocial birds must learn to eat during the first few days of life, while altricial birds are fed by their parents. In altricial species, resorption of the yolk is faster than in precocial species and takes about four days.

In gallinaceous birds, the yolk sac should not persist beyond six to nine days and should not be larger than pea-size between six to eight days of age.⁷¹ In the ostrich, yolk sac absorption may require eight or more days. In the emu and cassowary, a yolk sac can be palpated for at least one week, but it should be reduced in size. It should be noted that at hatching, the liver of some birds is a bright yellow color due to absorption of pigments from the yolk sac (see Color 30). The liver gradually changes to the mahogany color of the adult between eight and fourteen days of age in gallinaceous birds.¹³⁰ In the adult chicken, the vitelline diverticulum has been found to contribute to extramedullary myelopoiesis, and it has been suggested to have a lymphoepithelial function.¹³⁰

The large intestine usually consists of paired ceca and a short, straight rectum. The ceca arise at the ileorectal junction. The anatomy of the ceca varies among species. In Galliformes, the ceca are large and curve in a caudodorsal direction. Tetraonids (eg, capercaillies grouse that eat branches and twigs of trees) have the largest ceca of any species because of their high cellulose diet.⁴ The ostrich has a large sacculated cecum while other ratites have only vestigial ceca. Passeriformes, Columbiformes and some Psittaciformes also have vestigial ceca. Budgerigars have no ceca. Vestigial ceca usually contain large amounts of lymphoid tissue. In Galliformes, the lymphoid cecal tissue is located in the proximal part of each cecum and is called the cecal tonsil. Large ceca are involved in the bacterial fermentation of cellulose, and are also important in water reabsorption from ureteral urine. The type of food consumed by a bird influences intestinal length. Red Grouse fed a pelleted ration have a cecal length 50% shorter than those of free-ranging birds.¹³⁶ This is extremely important when one considers the problems with digestion that could occur if captive-raised birds were released into the wild.

The rectum lies in the dorsal part of the abdominal cavity and is a continuation of the ileum. It is usually a short, straight organ, but in some species, including the rhea, the rectum is looped or folded. In the emu, the rectum is adapted to preserve water. The high resorptive capacity may be related to increased folding of the mucosal surface, which increases the surface area by a factor of five. The emu has a limited renal concentrating ability with a maximal urine:plasma osmotic ratio of only 1.4:1.5.¹⁸²

Intestinal Diseases

Enteritis

Many infectious agents can cause enteritis. Table 19.1 lists some infectious causes of diarrhea. Infectious stunting syndrome (ISS) in chickens (probably of viral etiology) is associated with an enteritis and inflammation of the pancreatic ducts. Most affected birds recover completely after a period of diarrhea. However, some birds develop exocrine pancreatic deficiency secondary to blockage of the pancreatic ducts (Colors 19.4 and 19.6).⁹³

Ileus

Ileus (intestinal obstruction) can be defined as a condition wherein the passage of intestinal contents is arrested or severely impaired. The cause of intestinal obstruction may be physical or it may be due to impaired motor function (paralytic ileus) (Color 19.18). Physical causes may be located within the lumen, in the intestinal wall or outside the intestine. Occlusion of the intestinal lumen may be caused by foreign bodies, enteroliths or parasites. Intestinal wall lesions that have been reported to cause stenosis



FIG 19.15 A twelve-week-old Blue and Gold Macaw chick was presented for anorexia, depression and regurgitation of three days' duration. The abdomen was severely distended and doughy. Heart sounds were muffled dorsally, and severe dyspnea occurred following minimal exercise. A modified transudate was collected by abdominocentesis. Abnormal clinical pathology findings included: WBC=4500, AST=800 and LDH=1200. Radiographs indicated gaseous distention of the bowel. The bird did not respond to supportive care. At necropsy, the abdomen was filled with yellow fluid, and the bowel loops were distended with gas and were blue-black in coloration. Histologic findings were suggestive of avian viral serositis. The bird's sibling died following a similar clinical disease.

in birds include tumors, granulomas and strictures (eg, cicatrization tissue induced by foreign bodies). Extraluminal compression may occur from intussusception, volvulus mesenterialis, volvulus nodosus, incarcerated hernia mesenterialis, pseudoligaments and adhesions due to tumors or peritonitis. Vascular causes of ileus include embolism and thrombosis of a splanchnic artery or vein with infarction of a bowel segment.

Neurogenic causes (paralytic ileus) include lead poisoning, peritonitis, neuropathic gastric dilatation and enteritis (Figure 19.15).

Once the intestine is obstructed it dilates, and fluid is collected in the intestinal lumen and lost from the circulation. Clinical signs depend on the site and severity of the obstruction. The birds become rapidly dehydrated and are severely depressed. In many conditions ischemic necrosis of the intestinal wall occurs, leading to increased permeability and protein loss into the intestinal lumen. Resorption of intestinal contents, including endotoxins released from gram-negative bacteria, can cause shock. Usually complete intestinal obstruction in birds caused by intussusception or volvulus is fatal within 24 to 48 hours. A more protracted course is common with other causes of intestinal obstruction. Vomiting is usually present in complete mechanical obstruction, although this sign may be absent when the obstruction is in the caudal part of the intestinal tract. The passage of feces is diminished or absent. Diarrhea may be present with partial obstruction. Emaciation is seen when the obstruction occurs gradually from a progressive disease.

Plain radiographs may show the extent and location of the gas-filled intestinal loops. A barium enema or upper GI contrast study may be used to determine the exact location of the obstruction. The use of double contrast techniques facilitates visualization of lesions in the intestinal wall. Early diagnosis and rapid surgical correction may successfully resolve many intestinal obstructions. Birds should be stabilized with fluids and antibiotics before surgery.

Intussusception

Intussusception of the distal part of the small intestine is occasionally reported in young gallinaceous birds secondary to enteritis or spasmodic antiperistalsis caused by a nematode infection or coccidiosis. The affected part of the intestine rapidly becomes necrotic, inducing adhesions with neighboring tissues. Death usually occurs from peritonitis and toxemia.⁹³

In 19 cases of intestinal intussusception in gallinaeous birds and ducks, 15 cases involved the small intestine, three cases involved the ceca and one case involved the rectum. The middle and distal parts of the small intestine were usually involved. The beginning of the intussusception was usually located about 30 cm proximal to the ileocecal junction in chickens and 10-20 cm proximal in ducks. In one bird the distal end of the small intestine was invaginated into the rectum, while in another bird, 5 cm of the small intestine was prolapsed through the cloaca. In one case of cecal intussusception, the cecum passed through the rectum, and the black-red color of the apex could be seen in the cloaca. In the other cases of cecal invagination, part of the cecum was invaginated into itself. Invagination leads to partial obstruction with accumulation of fluid and gas proximal to the affected site. Rectal intussusception, which can lead to rectal prolapse, has been reported in gallinaceous birds and has been noted in Psittaciformes.⁵¹

Volvulus Mesenterialis

Volvulus mesenterialis can be defined as the twisting of a portion of the intestine around its mesenteric attachment. Volvulus mesenterialis jejuni has been reported in chickens, ducks and pigeons.⁵¹ The presence of stalked tumorous egg follicles with associated adhesions and stalked mesenteric cysts are common predisposing factors. In one case (pigeon), a heavy ascarid infection was present. In the other cases, enteritis was present, which may have predisposed the birds to mesenteric torsion. In one case, a fecalith caused obstruction and torsion of the rectum just proximal to the junction with the cloaca. The entire rectum was filled with fecal material and gas. In the ostrich, torsion of the large bowel has also been reported.²⁰⁹ It is speculated that an abrupt change in feed may be a predisposing factor.

Volvulus Nodosus

Volvulus nodosus can be defined as twisting of an intestinal loop around itself. Volvulus nodosus is usually seen in conjunction with a volvulus mesenterialis jejuni but may occur independently. Predisposing factors include adhesions and antiperistaltic movements induced by intestinal ulcerations, heavy ascarid infestations, accumulation of sand, foreign bodies and tumors.

Occlusion of the bowel can occur if intestinal loops herniate through tears in the mesentery or through the abdominal wall.⁵¹ Abdominal herniation is particularly common in egg-laying hens, and may be

associated with trauma, abdominal masses, egg binding, tenesmus or endocrine abnormalities.

Other reported causes of intestinal obstruction include a stone lodged near the boundary between the duodenum and jejunum, sloughed koilin layer in a pigeon obstructing the distal part of the rectum where it joins the cloaca, and phytobezoars lodged in the distal part of the ileum or distal part of the rectum. Acute death was caused by rupture of an intestinal diverticulum in two of these cases and intestinal hemorrhage in one case. Other affected birds died eight to ten days after developing clinical signs, probably due to shock and absorption of intestinal toxins.⁵¹ Sarcoma of the small intestine, an abscess or a cyst in the distal part of the rectum, diphtheritic enteritis with obstruction in the distal part of the small intestine and a stenosis caused by circular cicatrization tissue in the small intestine or rectum have also been reported as causes of intestinal obstruction.

Persistently feeding voluminous feedstuffs of poor nutritional value caused intestinal impaction in a group of Galliformes, Anseriformes and Columbiformes. In some cases, the entire gastrointestinal tract was involved. In others, the small intestine, rectum or cecum was the major site of impaction. Obstruction of the small intestine, which may progress to rupture, may be caused by ascarids or cestodes in Galliformes, Anseriformes, Falconiformes, Psittaciformes or Passeriformes (see Figure 36.30).^{91,93,154} Esophageal and intestinal perforations in turkeys occurred following the ingestion of grasshoppers.²¹⁵ Intestinal perforation by fir cones and glass have been reported in Tetraonidae.⁴

Coligranuloma: Hjarre's Disease

A coliform-induced granulomatous disease (Hjarre's disease) has been reported in captive⁷⁵ and free-ranging Galliformes. Clinical signs are not specific and include emaciation and diarrhea. Large lesions may cause intestinal obstruction. Granulomas may also occur in the liver; ceca (which may be very large), duodenum and mesentery. The lesions should be differentiated from leukotic neoplasms and tuberculosis.

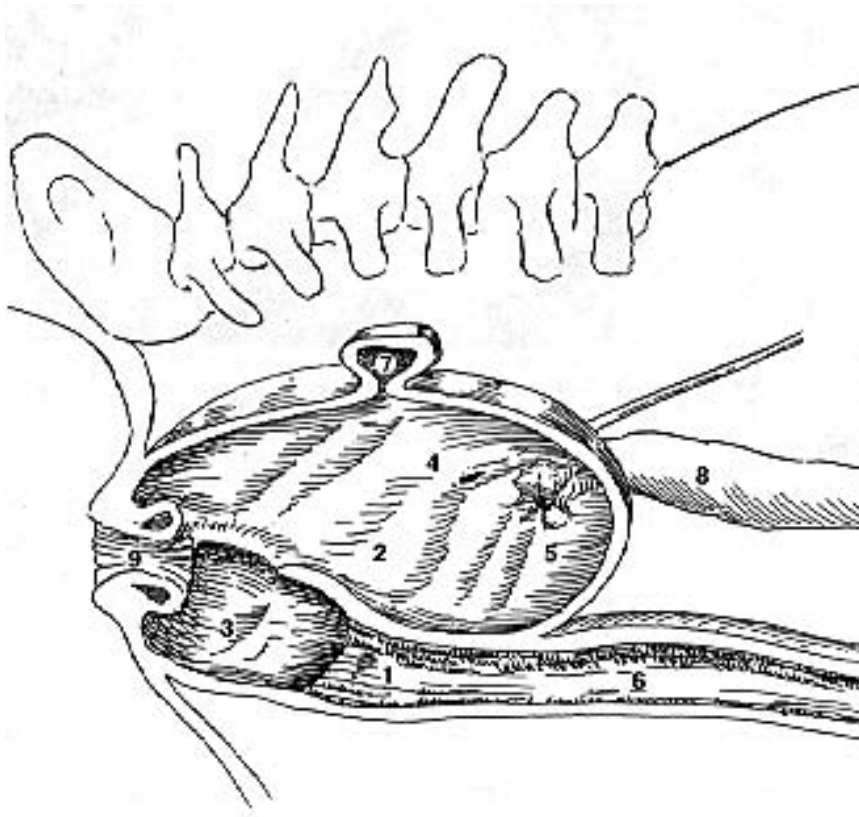


FIG 19.16 The cloaca is composed of the 1) coprodeum, 2) urodeum and 3) proctodeum. The 4) openings of the ureter and 5) vagina can be seen in the urodeum. Other structures associated with the cloaca include the 6) rectum, 7) cloacal bursa, 8) oviduct and 9) vent.

The Cloaca

Anatomy and Physiology

The cloaca consists of three compartments: the coprodeum, which is directly continuous with the rectum; the urodeum, which contains the openings of the ureters and genital ducts; and the proctodeum, which opens to the outside through the lips of the vent.¹³⁰ The coprodeum is separated from the urodeum by the coprourodeal fold, while the urodeum and proctodeum are separated by the uroproctodeal fold (Figure 19.16). A striated sphincter muscle controls the action of the vent. In the cock, the phallus, if present, lies on the crest of the ventral lip of the vent. It consists of a median phallic body flanked on either side by a lateral phallic body and lymphatic folds. Tumescence of the phallus is due to

lymph, which flows from the paracloacal vascular bodies that lie in the ventrolateral walls of the urodeum. In the detumescent state, the phallus is directed toward the interior of the cloaca. This type of phallus is called non-intromittent, because it does not enter the cloaca of the female but is merely applied to the protruded oviduct of the female. In adult male ducks and ratites, a distinct phallus is present that is inserted into the female cloaca during coitus. The male Vasa Parrot also has a large copulatory organ that swells considerably during the breeding season. This physiologic phenomenon in the Vasa Parrot should not be confused with cloacal pathology.

The cloacal bursa (bursa of Fabricius) is a dorsomedian pear-shaped diverticulum of the cloacal wall (see Figure 5.6). In chickens, it reaches its maximum size at six weeks when it measures 3 x 2 x 1 cm and weighs about 4 grams. It then begins to regress at about 8 to 12 weeks of age, and by 20 months it weighs only 0.5 g. In the adult, a nodular remnant of the bursa can be identified. In ratites, the neck of the bursa has a wide lumen, which does not occur in other avian species. In these birds, the proctodeum and cloacal bursa form one single cavity. The unusually wide entrance to the bursa is often incorrectly identified as a urinary bladder. The bursa is the site of differentiation of B-lymphocytes, which play an important role in the humoral defense system of the body (see Chapter 5).

During defecation, the coprourodeal fold protrudes through the vent to prevent fecal contamination of the urodeum and proctodeum. Similarly, the uroproctodeal fold protrudes through the vent during egg laying. Urine deposited in the urodeum moves retrograde into the rectum (and ceca, if present), where reabsorption of water takes place. Often birds will have watery excreta when they are excited, because they defecate before water reabsorption is complete.

Clinical Examination

Clinical signs indicative of cloacal disorders may include flatulence, tenesmus, soiled pericloacal area,

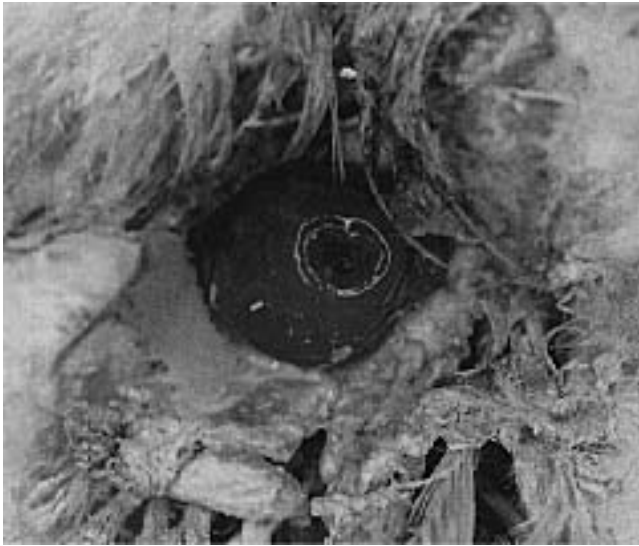


FIG 19.17 A two-year-old Umbrella Cockatoo was presented with a four-day history of tenesmus. The bird was fed a seed-based diet and was constantly demanding affection from its owner. Physical examination findings included the accumulation of excrement around the vent and protrusion of a smooth, glistening, pink cloaca. The bird had a moderate number (15%) of gram-negative bacteria in the feces and was placed on injectable enrofloxacin and medroxyprogesterone acetate. The mucosa of the cloaca was coated with apple cider vinegar to check for papillomas. None were detected. The bird responded to the antibiotic and hormone therapy as well as to a change in diet to a formulated product and behavior modification. The tenesmus stopped, and there were no further problems with cloacal prolapsing.

protruding tissue from the cloaca and foul-smelling feces. Examination of the cloaca should start with the feathers and skin around the vent. Normally these structures should be clean, and there should be no signs of inflammation (Color 19.8). An abnormal acidic smell can be a sign of cloacitis, which is often associated with cloacal papillomatosis.

■ Cloacal Diseases

Cloacal Prolapse

A prolapse involving the cloaca may contain intestines, oviduct and one or both ureters. The appearance of smooth, glistening, pink tissue is an indication that the cloaca has prolapsed, which may be caused by sphincter problems, chronic irritation of the rectum or tenesmus. A cloacal prolapse may cause severe constipation and toxemia (Color 19.10). In gallinaceous birds, cannibalism by cage mates may result in cloacal rupture and evisceration of the affected individual.

Acute cloacal prolapse associated with egg laying generally responds to manual reduction, followed by

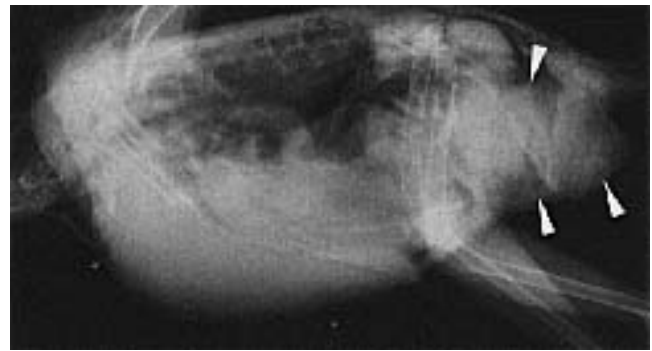


FIG 19.18 A four-year-old male Amazon parrot was presented with a history of straining to defecate and emitting a foul odor. A large pericloacal mass was palpable on physical examination. The mass was ulcerated, hyperemic, moist and associated with a yellowish-green discharge. Cytologic evaluation of samples collected from within the mass revealed gram-negative rods, degranulating heterophils and macrophages containing bacteria. Radiographs showed an extensive pericloacal mass that was causing cloacal distention (arrows) and cranial displacement of the intestines. The mass was surgically debrided, and the bird was successfully treated with piperacillin.

application of two simple transverse stay sutures perpendicular to the vent. Postoperative straining can be prevented by applying xylocaine gel in the cloaca BID. The cause of the straining or increased abdominal pressure should be corrected to prevent further prolapsing.

In cockatoos, chronic cloacal prolapse may be associated with sexual behavior in the presence of the owner, or can be caused by idiopathic straining (Figure 19.17). A combination of ventral cloacopexy¹⁷³ and cloacal mucosal “reefing” has been used to correct chronic cloacal prolapse (see Chapter 41).

Cloacitis

A sporadically occurring, chronic inflammatory process of the cloaca with a very offensive odor, commonly known as “vent gleet,” may occur in laying hens and occasionally in males. A yellow diphtheritic membrane may form on the mucosal surface, and urates and inflammatory exudate contaminate the skin and feathers around the vent. The cause is unknown. Treatment consists of cleaning the area and applying a local antibiotic ointment.⁹³

A similar condition has been reported in ducks. Scarring, which reduces the elasticity and diameter of the cloaca and may prevent egg laying and, in extreme cases, defecation, is a complication of cloacitis (Figure 19.18).^{71,93}

Neisseria, *Mycoplasma* spp. and *Candida albicans*

Bacteria (especially *Neisseria* and *Mycoplasma* spp.), and *Candida albicans* have been associated with a venereal disease affecting ganders.^{9,14,188} It seems likely that the cloacitis observed in drakes has a similar etiology, but an association has not been confirmed.

Cloacal infections may occasionally be observed in other species and may result from trauma, surgery or infectious diseases. Uroliths or fecaliths may form during the process. Cloacitis is often seen in psittacine birds suffering from cloacal papillomatosis.

Phallus Prolapse and Venereal Disease in Anseriformes

The phallus may not retract into the cloaca in some sexually mature drakes. The problem is usually associated with an extensive infection in the erectile tissue at the base of the phallus. It has been suggested that the etiology of this condition is traumatic, because the incidence is higher under conditions where the drakes have to mate with the females out of the water.⁷¹ Drakes with females that have cloacitis may have a phallus infection, suggesting that an infectious agent can play a role in phallus prolapse.

Cloacal Stricture

Infections, surgical manipulation of the cloaca (particularly for removal of papillomas) and trauma may cause stricture of the vent, requiring surgical recreation of an opening and appropriate aftercare to prevent a recurrence.¹⁷³

Cloacal Impaction

Cloacal impaction may occur from foreign bodies (eg, potato chunks in Galliformes), fecaliths, concretions of urates and retained necrotic eggs (Figure 19.19). Uroliths can vary from six to eight millimeter-thick concretions on the cloacal wall to solid masses the size of a chicken egg. In any case of cloacal impaction, passing excrement is difficult or impossible and can cause congestion of the ureters and dilatation of the intestines as far proximal as the duodenum. Renal failure and visceral gout may occur if the ureters are blocked.⁵¹

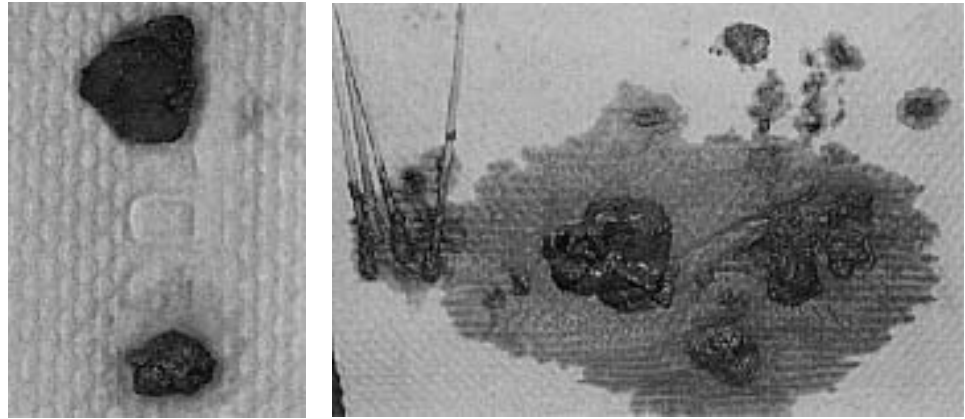


FIG 19.19 A mature Amazon parrot was presented with a two-day history of depression, anorexia and straining to defecate. The abdomen was distended. When a cotton swab was inserted into the cloaca, excrement would “squirt” out. When the swab was removed, no excrement would be released. Several uroliths that were functioning as a valve were identified at the opening of the cloaca. When the uroliths were removed, the bird was able to defecate normally.

Cloacoliths composed of urates have been observed in numerous psittacine birds, particularly macaws and Amazon parrots.¹⁷³ The etiology is unknown. Treatment consists of segmenting and removing the concretions. Cloacal impaction may also occur secondary to cloacal infections and cloacal stricture (Figure 19.20).

Cloacal papillomatosis is a well known disease in psittacine birds and is recognized clinically as a glistening red or pink cauliflower- or strawberry-like mass rising from the cloacal orifice (Color 19.9).^{42,67,86,112,173,190,196,200} Early lesions are characterized by a rough-appearing mucosa at the mucocutaneous junction of the cloaca. Other presenting signs may include tenesmus, melena, foul-smelling feces, flatulence, pasting of the vent and cloacoliths. The abnormal odor is likely to be caused by bacterial proliferation in the crypts caused by the papillomas. The incidence of disease is higher in New World parrots, but Old World parrots may also be affected.⁶⁹ The condition is frequently misdiagnosed as a cloacal prolapse. Applying an acetic acid solution (apple cider vinegar) to cloacal epithelium will change the color of papillomatous tissue to white. A definitive diagnosis can be made after histopathologic examination of a biopsy. Cloacal papillomas are often associated with similar lesions in the oropharynx, choana, esophagus, crop, proventriculus, ventriculus and occasionally mucosa of the eye and nose.⁶⁷ The etiology is presently unknown. There seems to be a high correlation between neoplasia of bile ducts and pancreatic ducts and papillomatosis in psittacine birds.^{41,69,86,159}

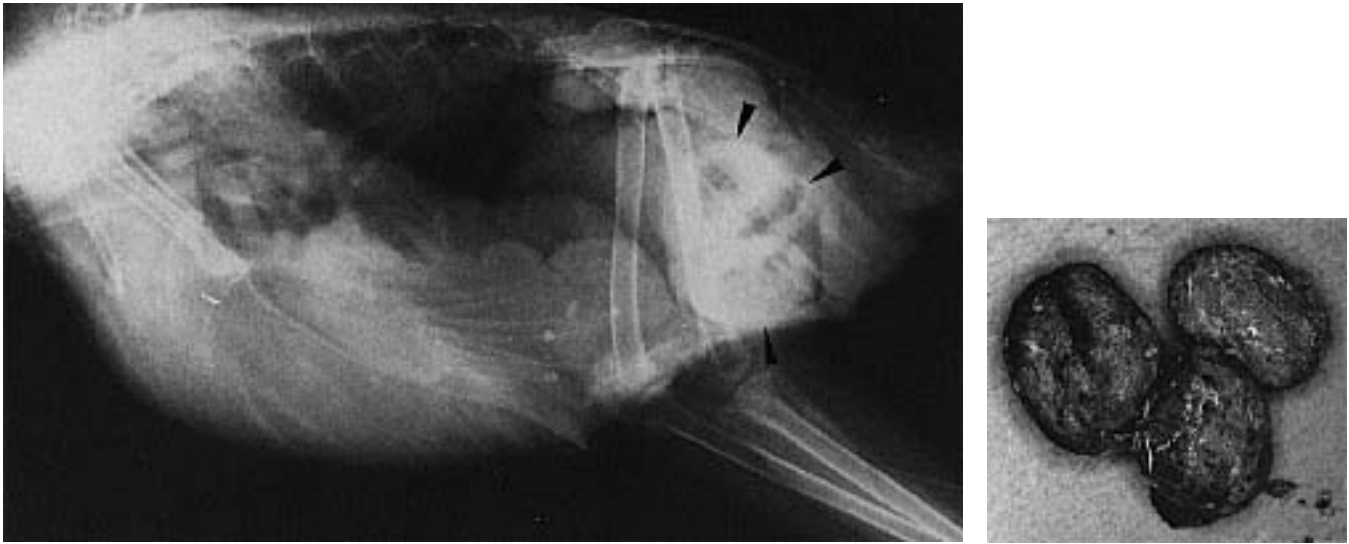


FIG 19.20 A ten-year-old Blue-fronted Amazon Parrot was presented with depression, failure to defecate and a distended abdomen. The referring veterinarian had placed a purse-string type suture to correct what was diagnosed as a cloacal prolapse three days before presentation. The abdomen was distended and tight. Unproductive tenesmus was noted by a constant “winking” of the cloaca. The purse-string suture was removed and the bird released a large quantity of excrement; however, the tenesmus continued. Survey radiographs revealed several elongated mineralized masses in the cloacal area (arrows). Three large uroliths were removed from the cloaca with forceps. The cloacal mucosa was hyperemic and ulcerated. A large cloacal papilloma was evident in the left lateral wall of the cloaca. The papilloma was cauterized by specific application of silver nitrate, and the cloaca was flushed with a dilute povidone-iodine solution. Removal of the papilloma required two further treatments. Papillomas that protrude from the cloaca must not be confused with a cloacal prolapse, as initially occurred in this case.

Various techniques have been used to treat cloacal papillomas, including cryosurgery, chemical cauterization, radiosurgery and autogenous vaccination,¹⁷³ but the reported spontaneous remissions and intermittent nature of the disease^{67,69,129,200} makes evaluation of the various treatments difficult. The introduction of birds with papillomas to a breeding facility should be prevented by performing a thorough physical examination at the beginning and end of the quarantine period. Of 41 papillomatous lesions, growth was benign in 40, but one single case was diagnosed as carcinoma *in situ*.¹⁹⁰ Other tumors should be considered in the differential diagnosis.^{13,58,15,163} Papillomas are most easily removed from the cloaca with careful, staged cauterization with a silver nitrate stick. The silver nitrate must come in contact only with the tissue intended to be removed to prevent severe burns of normal cloacal mucosa (Color 19.9).

The Pancreas

Anatomy and Physiology

The pancreas is situated on the left ventral side of the abdominal cavity between the descending and ascending loops of the duodenum. There are three lobes: dorsal, ventral and splenic. The dorsal and ventral lobes are usually connected (except in the Mallard and pigeon). The splenic lobe extends cranially from the dorsal or ventral lobe. There are one, two or three pancreatic ducts, which usually drain the pancreatic secretions into the ascending part of the duodenum. The exocrine pancreatic enzymes that are present in the duodenum include amylase, lipase, trypsin and chymotrypsin, which facilitate degradation of carbohydrates, fats and proteins, respectively. Trypsin and chymotrypsin are secreted as inactive precursors, and they become active only when they enter the duodenum. The activator is the locally produced enzyme, enterokinase, which changes trypsinogen to trypsin. This prevents the pancreas from being digested by its own enzymes.⁴⁹

Diagnostic Considerations

The pancreas has both endocrine and exocrine functions. The former are discussed in Chapter 23. Many postmortem lesions have been reported in avian pancreata.⁷⁰ There are two major clinical manifestations of pancreatic disease. If no pancreatic enzymes are available in the duodenum, maldigestion and passing of feces with excessive amylum and fat will occur. Affected animals may have voluminous, pale or tan, greasy feces (see Color 8). Fat in the feces can be demonstrated by Sudan staining. Interpretation of microscopic examination of feces for undigested food such as fat, starch grains and muscle fibers is complicated by variation in diets and by changes due to intestinal causes of malabsorption. Measurement of fecal proteolytic activity can be performed in several ways. The X-ray film gelatin digestion test is an unreliable assay of fecal proteolytic activity.²¹⁷ A test for fecal proteolytic and amylase activity is probably more reliable for use in birds.²¹⁴

Pancreatic Diseases

Acute Pancreatic Necrosis/Acute Pancreatitis

The pathogenesis of acute pancreatitis involves the activation of pancreatic enzymes in and around the pancreas and in the bloodstream, resulting in coagulation necrosis of the pancreas, and necrosis and hemorrhage of peripancreatic and peritoneal adipose tissue (see Color 14). Affected birds may be in shock, and radiographs may show loss of abdominal detail due to peritonitis and fluid accumulation in the peritoneal cavity. Dilatation of the small intestine may be visible due to an accompanying ileus. Increased plasma amylase activity (secondary to destruction of exocrine pancreatic cells) has been reported in chronic active pancreatitis in birds. Elevated plasma amylase may also occur with occlusion of the main pancreatic duct,¹⁶⁴ and might be a component of the infectious stunting syndrome in chickens.¹⁸¹ Lipemia may be present. A diagnosis of pancreatitis can be confirmed by endoscopy or exploratory laparotomy.

Obesity seems to be a predisposing factor of pancreatitis in birds. Treatment should include withholding food and oral medication for 72 hours, correction of fluid and electrolyte balance and prophylactic use of antibiotics. Dietary fat intake should be restricted to decrease the secretory load of the pancreas.^{70,158}

Chronic Pancreatic Fibrosis/Chronic Pancreatitis/Pancreatic Exocrine Insufficiency

A decrease in pancreatic glandular tissue or fibrosis may occur as the result of a chronic inflammatory process and cause clinical changes suggestive of pancreatic exocrine insufficiency (PEI).^{70,160} Further studies on the relation between pancreatic disease and plasma amylase and plasma lipase activities in birds are needed to facilitate clinical diagnosis; however, birds with a malabsorption syndrome and high amylase and lipase levels may respond to therapy with pancreatic enzymes, suggesting PEI. Frequently, the cause of PEI is undetermined. Pancreatic fibrosis was reported in two psittacine birds with chronic chlamydiosis.⁷⁰

High dietary levels of zinc may cause dilation of acinar lumina and degenerative changes in acinar cells including depletion of zymogen bodies, cytoplasmic vacuolization, the presence of hyaline bodies and other electron-dense debris, necrosis of individual acinar cells and fibrosis.^{46,48,98,204,216} Excess levels of zinc also interfere with exocrine pancreatic function.¹¹⁸

Pancreatic atrophy and fibrosis accompanied by impaired fat digestion have been reported in chickens on a selenium-deficient diet.^{192,193} It has been shown that addition of 0.1 ppm selenium to the diet could reverse the clinical signs and cause complete pancreatic acinar regeneration.⁷²

Infectious Stunting Syndrome (ISS)

Infectious stunting syndrome (ISS)^{93,1709} has many synonyms including runting and leg weakness,¹⁰⁴ runting syndrome,¹⁴⁷ infectious stunting,^{19,171} pale bird syndrome,^{65,116,142} malabsorption syndrome,¹⁴² brittle bone disease,¹⁹⁹ diarrhea and stunting,⁹³ runting and stunting syndrome,¹⁶⁵ and stunting and runting syndrome.¹²⁵ Clinical signs include growth retardation, steatorrhea, polyphagia, coprophagia, soft bones, swollen tibial epiphyses, rachitic ribs and abnormal feather development. The latter is possibly related to decreased circulating concentrations of thyroxine (see Chapter 23).^{93,170}

Although the etiology of this disease is not known, a virus is likely to be the etiologic agent,¹⁷⁰ but mycotoxins, other toxins, *Campylobacter* spp. and spirochetes may also be involved.^{93,189} Chickens, turkeys and guineafowl have all been documented with this disease. Most birds develop enteritis and inflammation of the pancreatic ducts and recover completely after a prolonged period of diarrhea.⁹³

Pancreatic lesions are thought to be incited by inflammatory reactions in the pancreatic ducts, which may result in complete blockage of the pancreatic ducts in a small proportion of affected birds. Blockage leads to vacuolization and shrinkage of exocrine cells and atrophy of the acini. As the disease progresses, much of the exocrine tissue is obliterated by fibroplasia. The changes are similar to those induced after experimental ligation of the pancreatic ducts.^{124,126} The resulting exocrine pancreatic insufficiency leads to maldigestion,¹⁷³ steatorrhea and inappropriate absorption of fat-soluble vitamins. The skeletal changes are likely to be related to reduced absorption of vitamin D and calcium, the latter being bound to the excessive amounts of fat and excreted in the feces.⁹³

Paramyxovirus Infections in Psittaciformes and Passeriformes

Paramyxovirus type III is a common infection in *Neophema* spp. and *Platyserca* spp., and is also encountered in some passerine birds, especially Estrildidae.^{184,198} Torticollis, other neurologic signs, cachexia and death are common. Some infected birds develop a yellow-to-white chalky stool that contains large amounts of starch. In almost all cases, a pancreatitis can be detected. Histologically, the lesions can vary from a few lymphoid follicles to massive infiltration with lymphocytes and plasma cells. In some cases, this is clinically manifested as pancreatic exocrine insufficiency.

Campylobacter infections in Estrildidae cause similar discoloration of the feces. Histologically, atrophy of the microvilli of the small intestine, which may cause a malabsorption syndrome, can be found.⁴⁷

Pancreatic Tumors

A high correlation between neoplasia of bile ducts and pancreatic ducts and internal papillomatous diseases in psittacine birds has been suggested.^{41,69,86,159}

The Pleuro-peritoneum

Anatomy and Physiology^{101,130}

The coelomic cavity in birds is subdivided by peritoneal, pleural and pericardial reflections into eight different cavities, excluding the eight cavities that are formed by the air sacs. Birds do not have a

diaphragm. They have two pleural cavities and five peritoneal cavities (Table 19.8). Clinically, these cavities dictate the location and spread of pathologic processes within the coelomic cavity and are important when considering surgical approaches to the abdominal organs (see Color 13, Anatomy Overlay).

TABLE 19.8 Reference Guide to the Coelomic Cavities in Birds

- Right pleural cavity (RPC)
- Left pleural cavity (LPC)
- Pericardial cavity (PC)
- Left ventral hepatic peritoneal cavity (LVHPC)
- Right ventral hepatic peritoneal cavity (RVHPC)
- Left dorsal hepatic peritoneal cavity (LDHPC)
- Right dorsal hepatic peritoneal cavity (RDHPC)
- Intestinal peritoneal cavity (IPC)

As in mammals, both the parietal and the visceral pleura are reflected over the lungs, but in birds a large surface of the parietal pleura is joined to the visceral pleura by fibrous strands. The degree of obliteration of the pleural space depends on the species. In some birds, including the domestic fowl, extensive areas of pleural cavity persist in the adult (in the chicken, dorsolateral). In these areas the lungs can collapse inward when the pleural cavity is opened. Some fluid can accumulate in these areas under certain disease conditions.

The pericardium in birds is essentially similar to that in mammals. Because the lungs are situated dorsally and there is no diaphragm, the heart is not enclosed by the lungs. The liver lies on both sides of the heart, and the parietal pericardium becomes continuous with the peritoneum. During embryonic development, the pleural cavity becomes separated from the peritoneal cavity by the pulmonary fold, which is a double-layered sheet formed by the parietal pleura (dorsal) and the parietal peritoneum (ventral). The cranial and caudal thoracic air sacs develop as dilatations from the bronchi and penetrate into this double-layered sheet, splitting the dorsal from the ventral layer. The dorsal layer becomes fused with the adjacent wall of the thoracic air sac, becomes tough and tendinous and acquires fascicles of striated muscle along its lateral edge that attach to the ribs (costoseptal muscle). This is called the horizontal septum, saccopleural membrane or pulmonary aponeurosis. The ventral layer becomes fused with the ventral walls of the thoracic air sacs and is called the oblique septum, which occurs bilaterally.

Unlike the horizontal septum, the oblique septae or saccoperitoneal membranes remain thin and look like air sac walls.

The partitions in the peritoneal cavity proper are formed by five sheets of peritoneum which, apart from the mesentery, do not occur in mammals.

The combined dorsal and ventral mesentery form a continuous midline vertical sheet from the dorsal to the ventral body wall as far caudally as the ventriculus. Caudal to this level, only a dorsal mesentery is present supporting the intestines. Cranially, the mesentery is continuous with the pericardium.

The posthepatic septum is composed of a left and a right double-layered sheet. It extends caudal to the liver from the last thoracic vertebra in a ventro-caudal direction to the caudal wall of the peritoneum. The posthepatic septum has connections with the visceral peritoneum enclosing the liver.

The ventriculus is enclosed between the two layers of the left sheet of the posthepatic septum. The principal peritoneal fat depot is located between the two peritoneal layers of the posthepatic septum. The lateral layers of the right and left sheet form the medial wall of the right and left hepatic peritoneal cavities. Because the right and left sheets are fused cranially and unite in the midline ventrally, the liver is separated from the rest of the viscera by this posthepatic septum. The posthepatic septum (together with the mesentery) divides the peritoneum into three principal cavities: the intestinal peritoneal cavity dorsomedially, and two lateral hepatic cavities that enclose the liver. The left and right hepatic cavities are further subdivided by the left and right hepatic ligaments, which run horizontally and are continuous with other peritoneal sheets (medially with the mesentery, cranially with the parietal peritoneum, caudally with the posthepatic septum and laterally with the oblique septum), thus forming the LDHPC, LVHPC, RDHPC and RVHPC, respectively. The LVHPC and RVHPC are large, elongated cavities extending from the left and right liver lobe, respectively, to the caudal body wall. The LDHPC and RDHPC are much smaller and are in contact with the craniodorsal aspects of the left and right liver lobes. The IPC is enclosed between the left and right hepatic cavities and extends from the liver to the vent. The intestines and gonads are suspended by mesenteries within the IPC. Each peritoneal cavity is blind and has no connections, except for one that exists between the IPC and the LDHPC. The peritoneum

plays an important role in the defense system of the body by closing perforations, containing infection and providing blood supply. The peritoneum heals rapidly after damage. Peritoneal injuries normally heal without the formation of adhesions, but in the presence of infection, ischemia or foreign bodies, fibrous adhesions may occur. The peritoneal surface allows the passive diffusion of water and solutes of low molecular weight between the peritoneal cavities and the subperitoneal vasculature. Larger molecules and particulate matter enter the bloodstream via the lymphatics. Healthy birds have a thin film of fluid in the peritoneum, which facilitates organ movement. The presence of free fluid in the peritoneal cavities is considered pathologic.

From the anatomic relationships outlined above, it is clear that diseases associated with the female genital tract (ovarian adenocarcinoma with implant metastasis on the peritoneum, egg-related peritonitis) are often confined to the IPC and LDHPC. Peritonitis from gastric perforation may be restricted to the LVHPC. Rupture of the liver can lead to accumulation of blood in one of the hepatic peritoneal cavities. Accumulation of transudate is most often seen in the LVHPC and the RVHPC and the PC, although the dorsal hepatic and peritoneal cavities may sometimes be involved. It is even possible to see some fluid accumulate in the RPC and LPC.

■ Ascites

Ascites is defined as the accumulation of serous fluid within one or more of the peritoneal cavities and may be caused by peritoneal and extraperitoneal diseases. Accumulation of fluid in one or more peritoneal cavity can result in abdominal distention. Large amounts of ascitic fluid may compress the pulmonary air sac system, causing dyspnea. During physical examination, abdominal distention can be recognized by the increased distance from carina to pubic bones. A bird with ascites should be handled carefully to prevent rupture of the air sacs, which can lead to immediate asphyxiation. Clinical signs may or may not occur with abdominal fluid accumulations. In liver disease, yellow or green feces may be seen. In neoplastic or liver disease, palpable masses may be present in the abdomen (Figure 19.21).

In ascites associated with liver disease (including hepatic congestion due to cardiac disease), increased portal venous hydrostatic pressure and decreased portal venous colloid osmotic pressure are important factors. Increased subperitoneal capillary permeabil-



FIG 19.21 A mature caique was presented with a two-week history of progressive dyspnea and abdominal swelling. Note the distal protrusion of the abdomen. Fluid collected by abdominocentesis was yellow and was characterized as a transudate (SpGr=1.012, Protein=1.8 mg/dl, few cells). The liver was swollen and the blood was lipemic. An exploratory surgery was performed, at which time a soft-shelled egg was removed from the uterus. Soft-shelled eggs are most often related to a primary etiology of malnutrition, which results in the fatty liver, lipemia and dystocia leading to possible egg-related peritonitis.

ity, decreased peritoneal lymphatic drainage and leakage from disrupted abdominal viscera (bile, urine) may cause non-liver-related ascites. Sometimes the definition of ascites is restricted to non-inflammatory transudate,⁴⁴ but the distinction between transudate and exudate is not always clear under clinical conditions. Conditions where an inflammatory exudate is present can be defined as peritonitis.

Although it has been suggested that chylous ascites (ascites due to the presence of lipoproteins and chylomicrons in the peritoneal cavity) can occur in birds,⁴⁴ current information on avian physiology sug-

gests that the absorption of fat from the intestine in birds is different from what occurs in mammals. The lymphatic system is not as well developed in birds as in mammals. The lymphatic vessels are small, the largest being hardly more than 1 mm in diameter, and the thoracic duct is only 1.5 mm across.¹⁴⁸ Because there is no functional intestinal lymphatic system in birds, absorbed lipids enter the portal system as large, very low-density lipoproteins. These lipoproteins have been defined as “portomicrons” in contrast to “chylomicrons” (the fat-rich particles that are absorbed by mammals).¹⁶ Chylous peritonitis, which occurs in mammals secondary to rupture of the lymphatic vessels or lymphatic congestion, is therefore theoretically not possible in birds.

Blockage of lymph drainage can be an important factor in the development of ascites in birds. For example, implantation of oviduct carcinoma on the intestinal peritoneal cavity rapidly induces ascites from portal hypertension secondary to pulmonary hypertension. Right ventricular failure with valvular insufficiency results in increased pressure in the vena cava where the lymph ducts connect to the circulatory system.⁹³

Pseudo-chylous ascites is the condition whereby turbid or milky abdominal fluid is seen. This may be caused by cellular debris and is associated with abdominal malignancies and infections.⁵⁷ A goose with a severe fatty degeneration of the liver accompanied by extreme hepatomegaly and hyperlipemia developed severe dyspnea secondary to pseudo-chylous ascites of unknown origin. A milky-appearing ascitic fluid was demonstrated in the ventral hepatic peritoneal space by laparotomy. Removal of 0.5 liter of ascitic fluid and diuretic therapy decreased the dyspnea. The concentration of total triglycerides in the ascitic fluid was 55 mmol/l, while total triglycerides in the plasma was 77 mmol/l. The ascitic fluid:plasma triglyceride ratio in this bird was opposite to what would be expected in chylous ascites, although the physical characteristics of the fluid were highly suggestive for this condition.

Edema may occur in organs and tissues in conjunction with ascites caused by hypoalbuminemia. It may be recognized clinically as edema of subcutaneous tissues of the abdomen or pitting edema on the feet (ducks with amyloidosis).

Diagnostic Methods

Radiographically, ascites is characterized by a diffuse, ground-glass haziness in the abdomen, and specific organs are often impossible to delineate. Administration of furosemide for several days or abdominocentesis will increase the diagnostic value of the radiographs. The cardiohepatic silhouette may appear widened, and the air sacs may appear narrowed laterally on the ventrodorsal view. Occasionally, ileus or enlargement of the heart, liver, spleen or other abdominal organs may be detected, providing information with respect to the primary disorder. Ultrasonography is a noninvasive technique that is valuable in the differential diagnosis of abdominal enlargement and ascites (see Chapter 12).^{27,100}

Abdominocentesis provides diagnostic information in birds with ascites (see Chapter 10).³³ Peritoneal lavage is possible, but extreme caution should be practiced to prevent iatrogenic puncture of the pulmonary air sac system and asphyxiation of the bird. Abdominocentesis should be performed when one is certain that free fluid is present to prevent inadvertent organ puncture. Transudate is characterized by a clear to pale-yellow color, a low specific gravity (<1.020), low protein (1 g/dl) and a low cellularity. Exudate is characterized by a high specific gravity (>1.020), a high protein content (3 g/dl) and possible presence of many inflammatory and mesothelial cells. Exudates may clot during sampling and may require an anticoagulant for proper cytologic examination. Septic exudates contain intracellular bacteria. Identifying a wide variety of bacteria suggests perforation of the gastrointestinal tract or the abdominal wall.

Clinical Biochemistry and Hematology

Laboratory investigations in birds with ascites should include plasma chemistries for hepatic and renal disease (AST, CPK, LDH, bile acids, protein electrophoresis, uric acid, urea). Renal protein loss should be evaluated by a quantitative determination of protein in the urine. Additionally, a PCV and total WBC and differential are indicated. When peritonitis is present, a marked leucocytosis can be observed, with the predominant cell type being heterophilic leukocytes. Juvenile heterophils (band cells) are normally not present in the peripheral blood and indicate severe inflammation. Granulomatous diseases and avian tuberculosis are often associated with monocytosis.

In neoplastic disease, exfoliated neoplastic cells may be encountered. Hemorrhagic effusions may have the

appearance of peripheral blood and have leukocyte and erythrocyte numbers comparable to peripheral blood. Chronic hemorrhagic effusions may show signs of erythrophagocytosis. Urine in the abdominal cavity can be recognized by the presence of spherical urate crystals.

In mammals, LDH and the ascites serum protein and LDH ratios are helpful in differentiating between exudates and transudates.¹⁵ The ascitic fluid amylase:plasma amylase ratio is useful for the diagnosis of pancreatic ascites. Ascitic fluid with a milky appearance and a triglyceride:plasma triglyceride ratio <1 is suggestive of pseudochyloous ascites because chyloous ascites does not occur in birds.

Differential Diagnosis^{44,62,71,89,97,112,170}

Abdominal enlargement due to ascites should be differentiated from other causes of abdominal enlargement such as obesity, neoplasia, herniation, egg-related peritonitis, granuloma, gravid uterus, gastrointestinal dilatation, hepatomegaly, splenomegaly and renomegaly. A cystic right oviduct can also pose a diagnostic challenge to the clinician because these fluid-filled cysts may reach a size up to 10 cm in diameter and may compress the abdominal viscera, mimicking ascites.

Chronic liver disease can cause ascites through intrahepatic portal hypertension due to hepatic fibrosis (aflatoxicosis, coal tar poisoning, plant toxins from *Crotalaria* spp. or rapeseed, bacterial or viral cholangiohepatitis). The accompanying hypoalbuminemia contributes to ascites formation. Blood chemistries, bile acids, low plasma albumin and liver biopsies are useful diagnostic techniques. Ascites from chronic liver disease is common in Anseriformes with amyloidosis and in mynahs, toucans and birds of paradise with iron storage disease.

Ascites may occur as part of generalized edema secondary to hypoalbuminemia caused by chronic liver disease, nephrotic syndrome and protein-losing enteropathy.

Neoplasias, particularly abdominal carcinomas (especially ovarian adenocarcinoma with implants on intestinal peritoneal cavity), may block lymph drainage, causing ascites with a high-protein content that contains neoplastic cells.

Congestive heart failure (right ventricular failure; RVF) is a common cause of ascites in gallinaceous birds and ducklings that are raised at high altitudes

and forced to grow at a rapid rate. Low environmental temperature and high-sodium diets have been associated with RVF and ascites in chickens and turkeys. Furazolidone causes cardiomyopathy in turkeys, ducks and chickens. *Fusarium moniliforme* var. *subglutans* produces the mycotoxin moniliformin that induces myocardial degeneration with associated hydropericardium and ascites in chicks, ducklings and turkey poults. RVF and ascites may also be associated with ricketts. Congenital ventricular septal defect can cause a left to right shunting of blood and result in RVF and ascites. Numerous drugs have been shown to cause cardiovascular malformations in chick embryos.

Exposure to chlorinated biphenyls, dioxin (toxic fat syndrome), creosol and coal tar products can damage the endothelial lining of blood vessels, causing hydropericardium and ascites.

Viral infections such as Marek's disease tumors may occur in the heart, and viruses of the leukosis-sarcoma group can cause various tumors associated with ascites (hemangioma and hemangiosarcoma of mesentery, erythroblastosis, mesotheliomas). Other viruses including avian polyomavirus and avian viral serositis can cause myocarditis and pericarditis leading to RVF and ascites.

Bacterial endocarditis and myocarditis may result in cardiac insufficiency (RVF) and ascites. *Staphylococcus*, *Streptococcus* and *Erysipelothrix* spp. have been associated with endocarditis, while bacterial myocarditis may occur in listeriosis, pullorum disease, fowl typhoid and other bacterial infections. *E. coli* peritonitis may be associated with ascending infections from the female genital tract.

Mycobacterium spp. infections can cause blockage of lymph drainage in some cases. Acid-fast (Ziehl-Neelsen) staining organisms may be noted in ascitic fluid. Peritonitis can occur from foreign bodies penetrating the intestinal tract and secondary to infections in the lungs, air sacs, pericardium, female reproductive organs and gastrointestinal tract. *E. coli*, staphylococci and streptococci can often be isolated in serofibrinous peritonitis in females as a result of ascending infections from the uterus. Aspergillosis air sacculitis can also involve the peritoneum.

Penetrating or nonpenetrating trauma to the abdomen can cause urate ascites, bile ascites, pancreatic ascites and hemoperitoneum (rupture of liver, spleen or kidney). Enlargement of an organ or other space-

occupying masses can block lymph drainage, resulting in ascites.

Cystic right oviduct occurs if the right Muellerian duct does not regress normally. The oviduct remnant is attached to the cloaca by a narrow stalk. Ultrasonography can differentiate between free fluid and fluid encapsulated within a cyst (see Chapter 12).

Therapy for ascites should be aimed at the primary disorder. Therapeutic removal of ascitic fluid is indicated only if ascites is accompanied by a life-threatening dyspnea. If hypoproteinemia is present, abdominocentesis will remove protein from a bird that may have compromised liver or kidney function. Diuretic therapy (furosemide) can be administered to effect. Low-sodium diets may be helpful.

Accumulation of fat in the peritoneal cavity can cause dyspnea through compression of the thoracic and abdominal air sacs. Obesity is an important differential diagnosis in birds with dyspnea and abdominal enlargement. It is commonly seen in parrots, cockatoos and pigeons on high-energy diets with restricted exercise, but many other species can be affected.

Ventral abdominal hernias are common in budgerigars and racing pigeons (particularly hens). A causal relationship with hyperestrogenism, which causes weakening of the abdominal muscles, has been suggested. The hernia may contain fat, loops of bowel or other abdominal organs. Incarceration of the intestinal tract is a rare but possible complication. A diagnosis can be made by physical examination and radiology. Treatment involves surgical closure of the abdominal hernia. Removal of excess fat that is primarily located between the sheets of the posthepatic septum facilitates the procedure. A perineal hernia containing a persistent right oviduct was observed by the author in a budgerigar.

The most common causes of peritonitis in birds are foreign bodies (from alimentary tract or through abdominal wall) and egg-related peritonitis. The latter condition can be the result of oviduct or ovarian dysfunction. Conditions such as false layer, internal layer, impaction of oviduct and torsion of egg yolk followed by infarction should be considered. Abdominocentesis is indicated to collect samples for further examination (cytology, culture).

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