ANATOMY AND DISEASES OF THE PSITTACINE DIGESTIVE TRACT: 
IT GOES IN THE BEAK AND OUT THE VENT

Drury Reavill
Zoo/Exotic Pathology Service
2825 KOVR Drive
West Sacramento, CA 95605

Modified and used with permission from ABVP, 2009 Austin Symposium and VIN Library listings, 2010.

Although the basic design of the digestive tract is the same across many taxons, birds have developed variations in gastrointestinal anatomy and physiology that allows them to fill many of ecological niches and take advantage of a wide variety of foods. The following review will cover the anatomy, some function, and the diseases involving the digestive tract of psittacine birds. The review will start in the oral cavity and end with the cloaca.

ANATOMY OF THE ORAL CAVITY AND PHARYNX

In all birds, the oral and pharyngeal cavities form a common cavity called the oropharynx. The palate has a medial fissure called the choana and ridges that are lateral and rostral to this fissure. Birds do not have a soft palate. These ridges are used for holding seeds when removing husks. The toma (cutting edge) of the horny beak is the functional tooth structure. In psittacine birds, the thick round muscular tongue manipulates food in concert with the toma of the beak and the ridges of the palate. Psittacine birds also have another adaptation of the articulation of the upper mandible in the cranium at the naso-frontal hinge, which permits an increased gape and provides flexion to absorb some of the shock associated with seed/nut cracking.

The choana connects the oral and nasal cavities. Along the oral margins of the choana are small caudally-projecting papillae. Similar papillae are identified on the roof of the oropharynx as well as the infundibular cleft, tongue, and the laryngeal mound. The infundibular cleft is caudal to the choana on the roof of the oral cavity. This cleft connects the oral cavity with the middle ear. The cleft is the common opening of the auditory tubes and has abundant lymphatic tissue within the walls.

The tongue in psittacine birds is non protrusible, muscular, blunt, and dexterous. Psittacine birds have intrinsic muscles in the anterior regions of their tongues, independent of the hyoid apparatus that adds flexibility. Lories and lorikeets have relatively long tongues that end in fine, hairlike processes for the collection of sap or nectar. The laryngeal mound is at the base of the tongue, opening into the glottis. Birds do not have and epiglottis.

Salivary glands are poorly developed and are a diffuse layer of small compound tubular salivary glandular formations present beneath the epithelium of the oropharynx. Taste receptors in psittacine birds are located on the palate and posterior tongue. They are greatly reduced in number (350) compared to humans (about 9,000).
**Lesions of the Oral Cavity**

The lesions of trauma, infection, and tumours are probably the most common findings with oral cavity diseases. Most will result in some clinical signs referable to the oral cavity, including anorexia, dysphagia, gaping, rubbing at the beak, scratching at the oral cavity, or the presence of foul odours. These common diseases may appear as plaques or masses. In psittacine birds the specific diseases are the diphtheritic form of pox, bacterial granulomas, hypovitaminosis A-induced abscess or granuloma, foreign body reaction, papillomatosis, and neoplasia.

**Noninfectious disease**

Non-infectious oral lesions include various types of trauma from foreign bodies to chemical or thermal burns. In psittacine birds, the potential insults to consider are of ingesting over-heated foods or other caustic substances including silver nitrate sticks, and wood or plastic toys. Acute lesions may present as lacerations, abrasions, or general acute inflammation with variable haemorrhage. With chronicity there may be fibroplasia and the formation of irregular thickenings in the affected area. Foreign bodies may penetrate the mucosa of the oral cavity and tongue and serve as a nidus for severe chronic inflammation and granuloma formation. These can be difficult to differentiate from tumor masses.

Vitamin A deficiency was a common disease in cage birds. Historically, birds with vitamin A deficiency have been on an unsupplemented, all seed diet for many years. Vitamin A is essential for the integrity of mucous membranes and the epithelium. The absence of vitamin A results in squamous metaplasia of mucous glands, and the epithelium, in several organ systems. The small mucous glands in the oral cavity will fill with keratin and expand to form submucosal nodules that contain yellow-white and friable material. These lesions can be severe resulting in obstruction of the choanal slit. There may be secondary bacterial infections. Blunting of the choanal papillae is a subtler lesion seen in birds with the early stages of vitamin A deficiency.

**Infectious disease**

Infectious agents causing oral disease in psittacine birds include viruses, bacteria, fungi, flagellates, and nematodes.

Nearly every bird family or group has its own poxvirus that produce the typical skin and/or mucosa lesions. Agapornis (lovebird) pox produces lesions in the oral and nasal cavities, and on the palpebrae, axillae, shoulders, and/or abdomen. These lesions are dark, discolored areas of skin, and when secondary bacterial infections exist, are very pruritic.

Amazona (neotropical) pox enjoys a wide host range in South American psittacine birds. Historically, this disease was a major problem in wild caught blue fronted Amazons (*Amazona aestiva aestiva*) and pionus nestlings held in quarantine. The disease may present in either the dry (cutaneous) or wet (diphtheroid) forms. The cutaneous form involves the non-feathered areas of skin with development of papules or raised scab like lesions around eyes, beak, nares, tibiotarsus, and feet. Lesions will eventually desquamate, usually without leaving a scar. The wet form affects the mucous membranes and generally results in a high mortality rate. The associated lesion is a depigmented, raised plaque covered by a diphtheritic membrane and appears on conjunctiva, oral membranes, tracheal mucosa, and within bronchi. Histologically mucosal epithelial cells are swollen with ballooning degeneration and contain the typical eosinophilic cytoplasmic inclusion bodies. Differential diagnoses for this lesion
on mucosal membranes includes vitamin A deficiency, bacterial infections, particularly with *Pseudomonas* sp., candidiasis, and trichomoniasis.

Transmission for all the poxviruses is by fomites or arthropod vectors, as well as direct transmission. The virus can remain viable for sixteen to nineteen days within mosquito salivary glands. A vaccine is available for some species.

Bacterial infections cause necrosis of the oral mucosa and glands. These infections must be differentiated from poxvirus lesions and may be secondary to vitamin A deficiency. Acute infections can be hemorrhagic, but chronic infections with gram-negative bacteria or mycobacteria will lead to granuloma formation. These can be difficult to differentiate from tumor masses grossly.

Cockatiel “lock jaw” syndrome results from extensive skeletal muscle fibrosis and inflammation in the muscles of the jaw that presents clinically as “lock jaw” (temporomandibular rigidity). This syndrome, with its distinctive clinical sign of the inability to open the beak, also includes rhinitis, sinusitis, and moderate to high mortality of affected birds. *Bordatella avium* and *Enterococcus* have been identified as causative agents of this disease. Associated superinfection with other gram-negative bacteria or yeast is a common finding. Once clinical signs develop, treatment is usually unrewarding.

The primary mycotic infection of the oral cavity is *Candida albicans*. Possibly due to their immature immune systems, Candidiasis is more prevalent in young birds, and may progress to a fatal systemic disease. Diagnosis of candidal infections is by clinical signs, history, lesions, and laboratory samples. Culture alone will not diagnose a yeast infection. Therapy must address the underlying conditions leading to infection.

Oral and digestive tract trichomoniasis, caused by *Trichomonas gallinae*, is rare in captive-raised psittacine birds. Grossly, yellow-white nodules and plaques characterize oral trichomoniasis. In the live bird, the organisms are readily seen on a wet mount. They die rapidly after the bird’s death.

**Neoplastic disease**

The most common neoplasm of the oral cavity is oral papillomatosis, primarily in the New World psittacine birds such as macaws, conures, Amazon parrots, and hawk-headed parrots. Oral lesions are particularly common in the larger macaws, but are relatively rare in Amazon parrots. Papillomas are most commonly located along the margins of the choanal fissure, at the base of the tongue, and on the glottis. They may become large enough to obstruct the airways. The papillomatous lesions are white to pink, raised, and most have the typical cauliflower-like appearance. Other reported sites of occurrence include the oropharynx, choanal cleft, conjunctiva, larynx, oesophagus, crop, proventriculus, ventriculus, nasal mucosa, nasolacrimal duct, bile ducts, pancreatic ducts, and cloaca. Papillomas vary in size and may become secondarily infected, with haemorrhage and necrosis noted. Recent research links these tumours to a viral infection with PsHV genotypes 1, 2, and 3 (psittacine herpesvirus). In psittacine birds, there is a suggested association between papillomatosis and bile duct and pancreatic duct tumours.

Squamous cell carcinoma (SCC) is a malignant tumor of squamous epithelial cells. SCC has been described in the oral cavity, oesophagus, crop, and cloaca. These neoplasms are often locally invasive and destructive with have a relatively high potential for local recurrence; however, they have a relatively low potential for metastasis. Severe secondary infections are common.
Mesenchymal tumours reported in the oral cavity include fibroma, fibrosarcoma, and lymphosarcoma. Gross differentiation of these lesions from each other or from granulomas is difficult. Histologic evaluation is recommended. Benign tumours can be removed surgically.

**ANATOMY OF THE OESOPHAGUS AND INGLUVIES (CROP)**

The oesophagus is generally divided into the cervical oesophagus, ingluvies (crop), and the thoracic oesophagus. Both the cervical and thoracic oesophagus are lined by a relatively thick stratified squamous epithelium. The esophageal wall is thin-walled but has a greater diameter than similarly sized mammals. There are subepithelial mucous glands. The cervical oesophagus generally runs on the right side of the neck. In the caudal neck area, it opens into a sack-like highly distensible structure called the crop.

The crop in psittacine birds is well-developed. This is used as a storage area and is lined by a stratified squamous epithelium like the oesophagus. No mucous glands are present within the crop. In the parrot, it has bulges first to the right and then left across the midline, lying transversely across the neck.

**LESIONS OF THE OESOPHAGUS AND INGLUVIES (CROP)**

**Noninfectious disease**

Primary noninfectious lesions of the crop include crop burns, foreign body penetration, and vitamin A deficiency. The crop can be burned by overheated formula or by contact with a heating pad or heat lamp over the skin covering the crop. Formulas over 43.5°C will burn tissue. The burns are usually found in the cranial-ventral portion of the crop. Grossly there may be reddening and edema. Blistering or necrosis through the crop wall and the overlying skin occurs in severe cases. Mild burns may heal without intervention. Moderate to severe burns will result in tissue sloughing and require a surgical closure.

Foreign bodies may penetrate the wall of the pharynx, oesophagus, or crop leading to necrosis. Food material can then migrate in the subcutis of the neck resulting in wide spread inflammation and necrosis. Use of a syringe for hand-feeding and an overly eager baby results in pharyngeal punctures to the buccal tissue to the right of the tongue. The use of a metal or rubber feeding tube can result in esophageal punctures at the mid-point of the neck where the oesophagus naturally narrows. Crop perforation is most common in birds that are being tube fed as nestlings or in the hospital. During feeding, large boluses of food may be injected into the tissues surrounding the oesophagus and/or crop. Small amounts may become walled-off but larger amounts will go onto develop into a cellulitis with toxaemia. These patients require immediate surgery to open, drain, and flush the pockets of food from the tissues. An attempt should be made to locate the original puncture. Typically the food migrates in a ventral direction.

**Infectious disease**

Proventricular dilatation disease (PDD) is defined by lymphoplasmacytic inflammatory infiltrates within the peripheral and/or central nervous system. A viral aetiology is associated with PDD (Borna virus by two research groups in 2008); however, the mode of transmission, incubation and disease development is unknown. Biopsy of the crop is a commonly used antemortem morphologic diagnostic tool for PDD because of the ease of access of the crop. However, histologic lesions are not
as consistently found in crop as they are in the proventriculus and ventriculus.

Bacterial infections of the crop and oesophagus can be primary or secondary. In immunosuppressed birds, there may be bacterial colonization and overgrowth in the mucosa. This overgrowth is not observable grossly and little or no inflammatory response is seen histologically. Bacterial and yeast overgrowth of the crop is also common in nestling birds with crop stasis. Failure of the crop to empty is a nonspecific sign and may be secondary to many digestive and systemic disorders. In severe infections of the crop, yellow-white nodules and plaques, haemorrhage, necrosis and a variable fibrinopurulent exudate affect the mucosal surface.

The primary mycotic infection of the crop is *Candida albicans*. Alteration of normal bacterial flora, in the integrity of the alimentary mucosa, or in immunocompetence will permit overgrowth (and possible dissemination) of resident yeast in the crop. Gross changes are similar to those seen in the oral cavity, with necrosis and a grey to yellow-white exudate on the mucosal surface. Often the crop wall appears significantly thickened and is thrown into broad folds. Tissue invasion, which occurs with colonization and penetration of a disrupted epithelial surface, is indicated by the presence of mycelial forms. Factors predisposing avian species to infection include: prolonged antibiotic therapy, hypovitaminoses (especially Vitamin A), feeding spoiled, stale, or sour foods, a stressful environment with moist floors, dirty nests, and faecal contamination, malnutrition, and co-existing bacterial or viral infections. Human error (overfilling the crop or feeding formula at an improper temperature) can predispose to crop stasis and candidal overgrowth in hand-fed psittacine birds. Cockatiels, parakeets, and lovebirds appear to be highly susceptible, making yeast infection appear to be a primary disease in these species.

Trichomonad infulvitis is an extremely rare disease of psittacine birds in the United States; however, it is common in budgerigars in Australia. Caused by *Trichomonas gallinae*, it can produce granulomatous and ulcerative lesions of the crop and occasionally disseminated disease involving the respiratory tract.

**Neoplastic disease**

Neoplasms of the oesophagus and crop include papillomas and squamous cell carcinomas. These are similar to those described in the oral cavity. Tumours of smooth muscle origin (leiomyomas and leiomyosarcomas) result in large space-occupying masses that may become necrotic and haemorrhage, although they can be asymptomatic when small.

**ANATOMY OF THE STOMACH (PROVENTRICULUS AND VENTRICULUS)**

The stomach is composed of two parts, the proventriculus and the ventriculus. In psittacine birds, it lies left dorsal and ventral. The proventriculus itself is a thick spindle-shaped organ. The mucosa is composed of compound tubular glands. These glands produce both pepsinogen and hydrochloric acid. There is an intermediate zone between the proventriculus and the ventriculus. This is a transitional area that opens into the ventriculus.

The ventriculus is responsible for mechanical digestion. There is a thick external muscularis, which supports the mucosa secreting the koilin lining layer. These ventricular glands secrete a carbohydrate-protein complex, which produces the dense thick serrated layer that will completely line the ventriculus. This koilin varies in strength with the koilin formed within the crypts of the mucosa softer and wearing faster than koilin from the gland tips, producing a serrated surface. The
koilin hardens in the middle to upper sections due to exposure from the hydrochloric acid from the proventriculus. The ventriculus, because of its muscular arrangement with the four semiautonomous smooth muscle masses that attach to extensive aponeuroses acts as a food grinder with both rotary and crushing movements. The greenish or brownish color of the koilin is due to reflux of bile pigments from the duodenum.

**Lesions of the Proventriculus and Ventriculus**

**Noninfectious disease**

Gastric impactions that some times result in perforation are most common in young psittacine birds who ingest foreign material. Impacted birds have proventricular dilatation and the wall is flaccid. Perforation or rupture is characterized by haemorrhage and accumulation of ingesta in and around the affected area.

Severe mineralization of the proventricular mucosa is seen in birds. One etiology is dietary and is believed to be secondary to excessive dietary calcium and may possibly occur in birds ingesting excessive vitamin D₃. The lesion has also been associated with renal failure and subsequent soft tissue mineralization.

**Infectious disease**

Infectious diseases affecting the proventriculus include viral, bacterial, mycobacterial, fungal, and parasitic.

Proventricular dilatation disease (PDD) is a disease of the central and peripheral nervous systems. A viral etiology is associated with PDD (Borna virus by two research groups in 2008); however, the mode of transmission, incubation and disease development is unknown. It will be discussed in this section because one of the most common lesions associated with this disease occurs in the proventriculus. The disease has also been called neuropathic gastric dilatation, myenteric ganglioneuritis and splanchnic neuropathy. African grey parrots, macaws, conures, and cockatoos are the most commonly affected psittacine species.

The clinical signs may vary between psittacine species but generally include; depression, weight loss, constant or intermittent regurgitation, passage of undigested seed in the feces, ataxia, abnormal head movements, seizures, and proprioceptive or motor deficits. Many birds develop a complete ileus of the digestive tract and as a result are severely wasted with little to no body fat by the time that they die. The primary gross lesions seen in PDD are flaccidity and dilatation of any portion of the gastrointestinal tract with the proventriculus, ventriculus, and crop being most commonly affected. The proventriculus may be so dilated as to fill much of the left side of the coelomic cavity and to displace the ventriculus to the right and cranially. If the bird has been on a seed diet, the proventriculus and ventriculus will be packed with seeds. Atrophy of the muscles of the ventriculus and thinning of the proventricular mucosa is common. Multifocal ulceration of the proventricular mucosa also occurs. The diagnosis currently relies on histopathology and identifying the typical inflammation involving the peripheral and central nervous system.

An organism that is frequently referred to as “megabacteria” is commonly found on the mucosal surface of the gastric isthmus of budgerigars, lovebirds, cockatiels, neophema parakeets, rosellas, canaries, finches, parrotlets, ostriches, and less frequently poultry. Recent work shows that this
organism is actually a previously undescribed yeast, *Macrorhabdus ornithogaster*. The organisms are relatively large (2 X 20-40 µm), Gram positive, PAS-positive, and stain strongly with Calcaflour White MR2 (a chitin stain). They are found densely packed, on the surface of the isthmus and often penetrate down between the glands of the isthmus. They may also extend into the ventriculus and penetrate deeply into the koilin.

In adult birds, weight loss and depression mark the infection. It may be a component of the normal upper alimentary tract flora of budgies and canaries, as many birds infected with this organism do not have clinical signs or histologic lesions. The mode of transmission is unknown. The organism may be detected with multiple wet mount preparations of the feces, but not all infected birds shed the organism. Amphotericin B has been suggested as an effective therapy based on in vivo studies.

Zygomycete fungi are a cause of proventriculitis as well as ventriculitis in several avian species. The morphology these fungal organisms are suggestive of Mucor, Absidia or Rhizopus fungal groups although tissue cultures or chromosomal analysis would be necessary for specific identification. Gross lesions are similar to bacterial infections and erosions or ulcers are common. It is unknown what initiates this type of fungal infection; however, this disease entity has proven difficult to diagnose antemortem and treatments are not described. The majority of cases are in young (<1 year) large psittacine birds.

Cryptosporidiosis of the proventriculus is seen in a variety of psittacine birds. Infections are recognized in cockatiels, lovebirds, parrotlets, and finches, they appear responsible for debilitating diarrhoea. Often no gross lesion is reported, but there may be excessive mucus production and variable mucosal hypertrophy. Although they are generally opportunistic and secondary invaders, they have been reported as primary pathogens producing respiratory and/or intestinal disease in birds. Transmission is by ingestion of infective sporulated oocysts.

**Neoplastic disease**

Gastric carcinomas and adenocarcinomas are described in *Brotogeris* species, budgerigars, lovebirds, cockatiels, parakeets, conures, Amazon parrots, cockatoos, African grays, macaws, and Eclectus. These tumours produce a thickening and irregularity of the proventricular and/or ventricular wall with a variable amount of inflammation, haemorrhage, and necrosis. Most are found at the proventricular-ventricular junction. Clinical signs reported include weight loss, weakness, inability to perch, head tilt, melaena, undigested seeds in the feces, beak overgrowth, and polyuria. Common hematology changes are anaemia and hypoproteinaemia. These tumours metastasize infrequently.

**Ventriculus**

**Noninfectious disease**

Trauma to the ventricular koilin and mucosa may be secondary to ingested foreign bodies. Depending on the species and thickness of the ventricular wall, there may be associated perforation. Grossly erosions, ulcers, and haemorrhage are noted, and foreign material may be identifiable in the lesion.

Xanthomatosi is rare finding in the ventricular musculature. If the lesion is large enough there is a nodular irregularity to the ventricular wall. Microscopically the xanthoma is similar to that seen in the skin and subcutis, with numerous large foamy macrophages surrounding cholesterol clefts.
The infectious and neoplastic diseases are similar to those described in the proventriculus.

**ANATOMY OF THE INTESTINES AND CLOACA**

The intestines of psittacine birds are relatively simple. They are arranged in loops with the first loop being the duodenal loop that encircles the pancreas. The remnant of the yolk sac, the vitelline diverticulum, is considered the junction between the jejunum and ileum, although this is of little physiologic significance. Psittacine birds do not have ceca. The microscopic structure of the intestines is very similar to that of mammals. Submucosal lymphoid tissue is found normally in many species of birds particularly in the distal ileum. This tissue can significantly alter the shape of the villi.

The cloaca is the combined outflow tract of the digestive, urinary, and reproductive tract. The colorectum enters into the coprodeum, the ventral aspect of the cloaca. Dorsally and separated by a horizontal fold from coprodeum, is the urodeum into which the ureters empty. The oviduct in the female enters the urodeum from the left lateral wall. The deferent ducts enter the urodeum in the male. The urodeum opens into the proctodeum.

**LESIONS OF THE INTESTINES**

**Noninfectious disease**

Noninfectious diseases of the intestinal tract of pet birds include trauma secondary to foreign bodies, ingested toxins, and infrequently torsion or intussusception. Trauma and torsion are usually obvious at necropsy, but histologic examination of affected tissue may be helpful to rule out underlying disease. Torsion leads to distension to sections of the intestine, and edema fluid may be present in the lumen. True intussusception must be differentiated from agonal telescoping of a portion of the intestine. In true intussusception there is edema and congestion with subsequent inflammation and fibrin deposition and adhesion formation.

Impaction of the intestines is usually the result of improper diet, ingestion of foreign material, and, in some cases, dehydration. The impacted section is dilated and firm and foreign material and ingesta are present in the lumen. Because the ventriculus is designed to hold ingested items until they are small enough to be digested by the intestines, intestinal foreign bodies are rare in pet birds.

**Infectious disease**

A variety of bacteria cause enteritis in psittacine birds. Gram negative pathogens can be primary or secondary invaders. Salmonellosis is of concern in all species of birds. The disease in pet birds was a significant problem in wild caught birds that were closely confined in quarantine stations. Currently, it is more likely to be seen in birds from aviaries that have a significant rodent infestation. *Salmonella* sp. and most pathogenic enteric bacteria are generally invasive, resulting in significant systemic lesions. The gross lesions of a bacterial enteritis include redness, exudation, and in some cases ulceration of the mucosa. Gas or fluid may distend the intestine. Faecal soiling of the feathers of the vent is a common lesion consistent with diarrhoea.

Clostridial overgrowth of the intestines may result in fatal enteritis. Lesions are most severe in the small intestine and vary from focal to diffuse haemorrhage, necrosis, and fibrin deposition. Although severe haemorrhage may be present, no other histologic changes are noted in some cases. Numerous large gram-positive spore-forming rod-shaped bacteria are seen in the lumen and mucosa of affected
birds. The lack of histologic lesions may be due to acute death from systemic enterotoxaemia. *Clostridium tertium* has been reported in a cockatoo with megacolon. At necropsy there was severe dilatation of the colon, and a severe lymphoplasmacytic inflammatory reaction was noted histologically.

Mycobacterial infections occur sporadically in many species of pet birds and generally results in a chronic wasting disease. Amazon parrots, brotogerids, Pionus, and finches/canaries are most commonly infected. Birds are most commonly infected with *Mycobacterium avium*, *M. intracellulare* and *M. genevensis*. *M. avium* and *M. intracellulare* share many characteristics and are frequently grouped as *M. avium*-intracellulare (MAI) complex. Infection is usually oral through ingestion of the organism. Clinically with the intestinal form, muscle wasting and emaciation is the usual presentation, along with eventual decrease in appetite. Gross lesions include diffuse and/or nodular thickening and opacification of the intestinal wall.

Diagnosis of mycobacteriosis can be difficult. Clinical presentation, severe leukocytosis, organomegaly, and cytologic or histologic presence of acid fast bacteria gives a high index of suspicion. While the leukogram indicates a severe response, the serum chemistries are usually unremarkable except liver enzymes which may be mildly elevated if the liver is involved. Serum protein electrophoresis may aid in the diagnosis early in the course of the disease. Radiographic examination may reveal hepatosplenomegaly and gas dilated loops of bowel. Laparoscopic examination and biopsy is extremely useful in obtaining a diagnosis. Faecal acid fast examination is usually not rewarding. PCR technology has promise for detecting the organism in faecal samples.

*Giardia* and *Spirochaulus* (previously classified as *Hexamita*), are two flagellates that are considered to cause intestinal disease in psittacine birds. These organisms may be associated with only minimal gross changes. Excessive fluid and mucus as well as mucosal hyperaemia is seen in some birds. Flagellates disappear from the intestine rapidly if the intestines are not immediately preserved.

*Encephalitozoon hellem* is an obligate intracellular single celled parasite. It may be found incidentally in the intestine or be associated with serosal and mucosal haemorrhage. This microsporidia infection also targets the liver, kidney, spleen, and less commonly the eye. Infection occurs most commonly in young lovebirds and budgies (less than one year). Infected birds die; survivors are stunted, unthrifty, and have diarrhoea. Gross necropsy findings include pasted vents, pale voluminous feces, watery intestinal contents, and undigested seeds in the feces. Both asymptomatic and clinical infections occur in budgerigars and lovebirds and other species. The transmission is probably by ingestion of contaminated material. The spores are highly resistant in the environment and can remain infective for months.

Metazoan parasites are infrequent causes of clinical disease in well-managed aviaries or in household pets. Cestodes are occasionally diagnosed at necropsy. There is usually no associated gross or histologic lesion. Cestodes are more common in Australian finches and wild caught African grey parrots and cockatoos. These parasites will survive in their hosts for many years and have the potential for causing intestinal obstruction in rare cases.

A number of nematodes have been reported in cage birds. With numerous parasites blockage of the intestine may occur. Of particular concern are the roundworms, of the genus *Ascaridia*. Roundworms are particularly common in Australian grass parakeets (*Neophema* sp.) and cockatiels with access to the ground. *Ascarid* infestations generally result in weight loss, anorexia, diarrhoea, and death. Rare reports in psittacine birds describe tissue migration of ascarid adult and/or larvae. The lesions include
intestinal obstruction with ascarids, hepatic nematodiasis, adult nematodes in the pancreatic and bile ducts and a disseminated bacterial infection. Faecal examination of contact birds is recommended although may not be definitively diagnostic as not all birds will shed eggs. Ascarids have a direct life cycle and environmental control is important.

Neoplastic disease

Primary neoplasms of the intestine include carcinoma, and several types of sarcoma. Carcinomas are infrequent. They present as variably-sized masses that may be ulcerated on the mucosal surface.

Lesions of the Cloaca

Noninfectious disease

Impaction of the cloaca can result from a variety of causes including failure to pass an egg, intrinsic disease of the cloacal wall and loss of muscle tone due to viral-induced ganglioneuritis [PDD]. Grossly there is dilatation of the cloacal wall and it may be irregularly thickened. The lumen may contain an egg, impacted faecal material or products of inflammatory disease. Abscesses of the cloacal wall may partially obstruct the cloaca or rupture into the lumen of the cloaca.

There are a variety of causes of cloacal prolapse although these can be challenging to find the etiology. Cloacal prolapse is especially common in tame umbrella cockatoos. It is speculated that behavioral factors contribute to the prolapse. The prolapsed mucosa will appear nodular or proliferative and there may be areas of necrosis and haemorrhage. Histologic examination may be necessary to differentiate prolapse and associated inflammation from cloacal papillomatosis. The prolapsed mucosa will be somewhat thickened and inflamed.

Infectious disease

Infections affecting the cloaca are the same as those seen in the upper intestinal tract and the gross and histologic features are similar.

Neoplastic disease

Papillomatosis is a common tumor of the cloaca and has been recently demonstrated to be induced by a herpesvirus. These proliferations can spontaneously regress and then recur. They can be identified anywhere in the gastrointestinal tract although are more frequently recognized within the cloaca and choana.

Cloacal carcinoma is an infiltrative tumor that leads to thickening of the cloacal wall. Carcinomas are usually firm and grey-white.

Anatomy of the Pancreas

The pancreas is divided into three lobes, dorsal, ventral, and splenic. The largest portion of the pancreas lies within the loop of the duodenum. This portion of the pancreas extends cranially and may come in contact with the spleen. A portion of the pancreas, in psittacine birds, parallels the abaxial side of the right duodenal loop. The normal pancreas is yellow to yellowish pink with a finely lobulated surface. Histologically it contains both exocrine and endocrine tissues that resemble its
mammalian counterparts. Islets are not uniformly distributed in the pancreas of all birds, and multiple sections from different portions of the pancreas must be made if the islets are to be seen and evaluated. The pancreatic ducts generally drain into the distal part of the ascending duodenum. The enzymes secreted by the exocrines pancreas include amylase, lipase, proteolytic enzymes, and sodium bicarbonate.

**Lesions of the Exocrine Pancreas**

**Noninfectious disease**

Prolonged caloric deficiency in birds will lead to pancreatic atrophy. Grossly the change may not be visible, but histologically there is acinar epithelial atrophy associated with normal islet’s of Langerhans.

The pancreas is the target organ in cases of zinc toxicity. Gross lesions may not be noticeable; however, slight parenchymal mottling is occasionally seen. The primary microscopic lesion is vacuolation and degranulation of acinar cells.

Acute pancreatic necrosis is seen in psittacine birds, particularly Quaker parakeets. Many of these birds die suddenly without significant clinical signs. Gross lesions include a firm pale pancreas, variable haemorrhage and adjacent fat necrosis characterized by firm yellow-white foci. The exact cause is unknown; high-fat diets have been implicated.

**Infectious disease**

Viral and bacterial agents reported to cause pancreatitis, include herpesvirus, polyomavirus, adenovirus, paramyxovirus [PMV-3], poxvirus, a variety of gram negative bacteria, and chlamydiophila. Gross lesions vary from none to haemorrhage and necrosis and there may be a purulent exudate in cases of bacterial pancreatitis. The viral infections usually result in inflammation and necrosis.

**Proliferative and neoplastic disease**

Bile duct hyperplasia and papilloma formation are seen in some birds with internal papillomatosis. They may accompany bile duct changes or occur independently of them.

Carcinomas are usually infiltrative with poorly defined borders. Grossly there may be obvious infiltration of the adjacent small intestine, and in severe cases most of the normal pancreatic architecture is lost. There may be severe adhesion formation binding the intestines and other organs into a solid mass. These tumours occur most commonly in cockatiels. Abdominal effusion is another common manifestation of this disease.

**Avian Digestive Physiology**

Feeding strategies of psittacine birds can be subclassified to include granivory (grain- or feed-based diets typical of budgerigars and cockatiels), frugivory (fruit-based diets typical for many macaws), and nectarivory (nectar-based diet of lorikeets and lories).

Starting in the mouth, touch receptors are found in rich supply on the tongue, oral cavity, and beak.
Avian taste buds are generally located in the oral cavity on the floor of the pharynx and at the base of the tongue. Nectarivores are able to differentiate between sugar solutions based on composition and concentration. Tannins can be detected in certain plant foodstuffs in birds that are folivores (plant-based diets). In studies using cockatiels, it appears that these birds have the ability to taste quinine, a secondary plant compound, near the same threshold as humans, but they are much less sensitive than humans to fructose.

Movement of food through the gastrointestinal tract of birds is similar to that of mammals, going in the general direction from the mouth to the vent. However, in many birds, it has been recognized that retrograde movement of digesta will occur between the proventriculus and the ventriculus, the small intestine and the ventriculus, the colorectum and the small intestine, and the cloaca and the colorectum. The reflux of the digesta between the proventriculus and ventriculus optimizes the action of enzymatic and mechanical digestion. The reflux from the cloaca to the rectum helps to resorb protein, salt, and water present within the urine.

Food leaves the oral cavity and enters the oesophagus, which widens to the crop. The crop allows for storage as well as softening of food for more efficient digestion. The softening is due to hydration by saliva added to the food during swallowing and any mucus that may be secreted into the crop. As food passes through the proventriculus, it is coated with acid and pepsin. It rapidly moves to the ventriculus, and contractions of the ventriculus will crush the food into smaller particles. Larger food particles may reflux back into the proventriculus for addition of fresh pepsin and hydrochloric acid. Food moving into the small intestine undergoes enzymatic digestion. Protein starches and nucleic acids are hydrolyzed within the lumen of the small intestine by the pancreatic enzymes. Intestinal lactase has not been identified in those birds tested. This suggests that most birds are unable to digest lactose-containing foods. Some frugivorous species and nectarivorous species have exceptionally long intestinal microvilli, which is presumed to aid the absorption of the free sugars that are found in their food.

**Recommended Reading**


