The Morphological Bases of Common Alimentary Tract Disorders Unique to Birds

David L. Graham[1]

The avian alimentary tract differs anatomically and functionally in several significant ways from those of mammals - which themselves demonstrate considerable anatomical and functional permutations. The avian beak, crop, stomach, yolk sac, small intestine, and cloacal (Fabricial) bursa provide examples of such departures from mammalian norms - anatomical and functional differences requiring specific awareness and consideration by the veterinarian dealing with avian alimentary tract disorders.

The Beak/Bill

The unique feature of the bird's oral prehensile equipment - the upper and lower bills or rostra (*rostrum maxillare* and *rostrum mandibulare*, respectively) - is the densely keratinized, hard, yet slightly flexible stratum corneum of the skin covering them - the rhamphotheca. The rhinotheca is the rhamphotheca of the upper bill, the gnathotheca that of the lower. In order to permit the least play or slack between the rhamphotheca and its underlying supporting bones its dermal fibrovascular stroma is thin and apposed directly upon bone periosteum; there is no demonstrable subcutis.

A clinically significant consequence of such close and unyielding encasement of the maxilla and mandible by the rhamphotheca is that inflammatory swelling of the thin dermis immediately compresses the delicate dermal vasculature. Dermal ischemia, unless of brief duration and rapid resolution, results in ischemic necrosis of not only the dermal fibrovascular tissue, but also of the suprajacent germinative epithelium dependent upon the former for its sustenance.
Loss of the dermal architecture has the same effect upon the skin of the beak as does such loss upon any other region of skin; although it may, at best, become re-epithelized, basic form and function are lost and a permanent, irreversible beak defect is the result. In the event of primary or secondary bacterial dermatitis of the beak skin it must be appreciated that lack of hemic perfusion precludes systemic antibiotics from reaching the affected site. Clearly, traumatic or infectious inflammatory damage to the

[1] **Schubot Exotic Bird Health Center, Dept. of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843-4467 United States of America**

periosteum and the osseous cortex of maxilla or mandible further complicate the healing process.

Esophagus and Ingluves (Crop)

The esophagus of many species of birds features a region of potential or permanent dilation - the ingluves - for the temporary storage and moistening of ingesta that will either be passed on to the ventriculus for further digestion or regurgitated for feeding of the young (of altricial species).

The esophagus, like the oropharynx, is well endowed with mucosal glands; the crop is aglandular and lined with non-keratinizing stratified squamous epithelium. During that period when altricial nestlings are fed regurgitated food the crop lining epithelium of columbiform species undergoes pronounced physiological parakeratotic thickening. The outer layers of the loose, parakeratotic lining are shed and regurgitated to feed the squabs as so-called "crop milk". It provides the nestling's total nutrition for several days, and subsequently supplements the regurgitated ingesta fed by the parents. Crop milk also contains immunoglobulins which in may, along with absorbed yolk antibodies, provide some degree of temporary passive immunity to the squab.

Dysphagia:
Lesions of the esophagus and crop are among the more common causes of dysphagia. Under the effects of hypovitminosis-A the mucosal glands undergo squamous metaplasia and the mucosal epithelium keratinizes and may progress to prominent hyperkeratosis. Loss of mucus lubrication is the result.

Endoingluvial mycosis, usually the result of infection by *Candida albicans*or other deuteromycotes, may occur in parent-fed and hand-fed nestlings, although the latter more commonly fall victim. Crop mycosis is often demonstrably secondary to one or another predisposing influence that has reduced the bird's resistance to infection or altered its normal flora (such as stress, treatment with broad-spectrum antibiotics, recurrent hypothermia, and the like). However, once established, the mycosis becomes a significant disease in its own right and requires diligence of diagnosis and treatment. Demonstration of large numbers of budding yeasts and pseudohyphae in a swab of the crop <u>mucosa</u> provides the diagnosis. Simply smearing out crop contents is inadequate as some cases of even severe crop mycosis can be missed if only the contents are examined. *Candida albicans* is a ubiquitous organism and is commonly observed in small numbers in the feces of normal birds; the mere presence of yeasts in the droppings does not warrant an *a priori* conclusion of alimentary tract mycosis.

Atony of the crop (failure to "put over" or pass on ingesta after a brief postprandial interval) may result from several causes: 1. crop mycosis (see above), 2.) overdistention of the crop from overfeeding with secondary atony of the myingluvium, 3.) feeding the nestling a formula of excessively thick consistency, 4.) thermal injury to the crop wall from overheated or inadequately cooled feeding formula, and 5.) defects of the

autonomic innervation of the myingluvium.

Caveat gavagetor

An all-too-common and avoidable circumstance is that of inadvertent puncture of the oropharyngeal or esophageal mucosa and wall with a stiff or rigid feeding tube followed by deposition of feeding formula into the paraesophageal / paraingluvial adventitia and cervical subcutis. The resulting cervical swelling is usually perceived by the hand-feeder and/or veterinarian to be a full crop. Inability to reduce the swelling by aspiration or crop lavage should suggest the nature of the lesion. The prognosis is poor to grave because most such cases are not diagnosed until the bird is *in extremis.* Sepsis, rapid decline, and death are the inevitable results if the swelling is not quickly recognized for what it is, drained via a skin incision, lavaged with saline, and lavaged with antibiotic solution. The site of puncture may be closed by the time of diagnosis, but should be sought and sutured if still patent.. The affected bird also requires treatment with systemic antibiotics and appropriate nutritional support (by gentle, competent intubation or via pharyngostomy tube).

Ingluvial gas gangrene

Ingluvial emphysema (gas gangrene of the crop wall) has been observed in two lories that died unexpectedly. *Clostridium perfringens* (untyped) was identified in the lesions but its source was not discovered. The affected birds had been fed a diet containing honey which was suspected of having provided a concentrated nutrient source promoting the growth of the anaerobe and toxin production.

Papillomas

Internal papillomatous disease is sometimes manifested by numerous endoesophageal and endoingluvial papillomas in the absence of oral and cloacal lesions. Progressively severe dysphagia and attendant loss of condition in combination with small nodules to larger masses palpable in the esophageal and crop wall suggest that papillomas should be included in the differential diagnoses of dysphagia..

The Avian Stomach

The avian stomach is bi-cameral and both portions are entirely glandular. The first segment, the proventriculus, has superficial, shallow, mucous glands and multiple, complex submucosal glands which produce digestive enzymes and hydrochloric acid. The second portion, the ventriculus, (gizzard) has simple, tubular mucosal glands which produce the koilin that lines the lumen and provides a firm substrate upon which its muscular walls can act to grind or knead ingesta or, in some species, to compact indigestible material prior to its regurgitation.

Proventriculus:

The proventriculus is continuous with the ventriculus via the terminal portion of the former - the intermediate zone - which is possessed of only simple superficial glands and is devoid of submucosal glands.

In many species of granivorous birds a large, rod-shaped bacterium comprises the exclusive autochthenous flora of the intermediate zone. These large, Gram-positive, rod-shaped organisms, originally mistaken for fungi, are commonly termed "megabacteria" and exist in intimate association with gland cells; indeed, the deep ends of some organisms are embedded within and closely enveloped by invaginations of the epithelial cell membrane. The gland cell so involved shows no microscopic or ultramicroscopic degenerative changes.

The term "megabacteria" is purely descriptive and should not be construed a taxonomic designation and thus is not herein italicized The organism (or organisms) have not yet been assigned a scientific binomial. Cultural characteristics, nutritional requirements, substrate utilization kinetics, and cell wall fatty acid constituents indicate that megabacteria isolated from budgerigars in the United States are either a member of the Genus *Lactobacillus* or are related closely thereto. It is perhaps more than coincidental that aviculturists have long recognized that birds evincing unthriftiness following a long course of broad spectrum oral antibiotics can be "brought around" by dosing them orally with live-culture yogurt which, incidentally, contains *Lactobacillus acidophilus.*

The nestling receives its inoculum of megabacteria with its meals of parental regurgitus which includes not only crop contents, but proventricular secretions and contents as well. Having colonized the mucosal glands of the intermediate zone, megabacteria proliferate and the excess population is passed down the alimentary tract with digesta and chyme and are consistently shed in the feces of birds they have colonized.

In the event of decreased food intake and decreased passage of ingesta thru the proventriculus, the numbers of megabacteria increase in and on the mucosa of the intermediate zone to the point that the excess population is grossly visible as a light grey to off-white tenacious coating on the mucosal surface. Their tendency to grow with continued exuberance in birds that are hyporexic or anorexic for one of a wide variety of reasons has led to their circumstantial incrimination as the etiological agent of a variety of ills. Attempts to fulfill Koch's postulates proving the pathogenicity of megabacteria have, to date, failed; no definitive evidence of the putative pathogenicity of megabacteria has been reported.

It is this writer's experience that megabacteria are consistently present in healthy adult and nestling budgerigars in better-producing aviaries and are often lacking or present in lower numbers and in fewer birds in many less successful operations. The possibility that megabacteria may be not merely a normal commensal, but rather, a symbiont that

specifically promotes improved proventricular function or provides more pervasive beneficent effects upon alimentary tract health and nutritional status to the host it colonizes warrants strong consideration.

Proventricular adenocarcinoma

Gastric carcinomas are rare in birds; the exception occurs in the Genus *Brotogeris* in which avian proventricular adenocarcinomas are most frequently diagnosed. As is the case with most avian carcinomas, proventricular carcinomas are locally invasive, but rarely, if ever, metastasize.

Ventriculus/Gizzard

The ventriculus is characterized in granivorous species by a *tunica muscularis* having two, thick, apposed muscle masses which impart a stout, roughly lenticular shape to the organ. The mucosal glands produce secretions that harden to form the tough, biphasic, koilin lining. Protokoilin produced within the deep regions of the gland crypts hardens into columns or rods. The tough koilin rods are more dense and more resistant to surface abrasion than is the softer matrix within which they are embedded and which is produced by the epithelium of the surface and of the necks of the glands. The result is that the koilin's lumen surface is something of a self-sharpening file; the ends of the tough rods project above the surface of the softer, eroded matrix and impart a "bite" or non-slip surface to the koilin anvils giving them a better grip on the ingesta being milled.

In frugivorous and carnivorous species the koilin is relatively thin, of a much softer consistency, and less organized than that of grain-eaters and of some species that eat molluscs and crustaceans. The ventriculus of lories is almost vestigial compared with that of more consistently granivorous parrots.

Dysfunction of the ventriculus in seed-eaters is clinically characterized by either the passage of intact seed kernels in the feces, by ventricular impaction and failure to pass ingesta on to the intestine - often accompanied by retrograde distention and impaction of the proventriculus - or both.

"Seeds In The Droppings"

The differential diagnoses of ventriculus dysfunction include:
<u>Degenerations of the tunica muscularis</u>
myocalcinosis (vitamin-D toxicosis)
nutritional myopathy (vitamin-E/Selenium deficiency)
lymphocytic myoventriculitis (Avian encephalomyelitis virus infection)

<u>Impaired innervation / atrophy of the tunica muscularis</u>

lymphocytic splanchnic neuropathy/gastric ganglioneuropathy/ “wasting disease”)
lead toxicosis

Koilin dysplasia
endoventriculitis (nematodiasis, bacterial infection)
endoventricular papilloma; an occasional component of internal papillomatous disease

Koilin degeneration
laminar calcinosis (vitamin-D toxicosis)
phycomycosis / zygomycosis
candidiasis (esp. in Estrildidae)

Vitelline (Yolk) Sac

The yolk sac, lined by the vitelline membrane, contains the yolk which provides nutrients to the growing embryo. The nearly depleted yolk sac is withdrawn into the coelom just before or at the time of pipping and is a source of nutrients for the newly hatched chick for the first few days after hatching. Indeed, the new hatchling, if the product of normal incubation, requires no food for the first two to three days after hatch.

Bacterial contamination of the yolk sac before it is internalized or of the umbilicus before it is sealed can result in septic omphalo-vitellitis which frequently generalizes as a multisystemic, fatal infection. Omphalo-vitellitis occurs most commonly in hatchlings that were “helped from the egg” a bit prematurely - before the yolk sac had been fully internalized. The aviculturist may report having “...poked it inside and put a bit of adhesive tape over the navel.” The results of bacterial cultures from several such cases from the particular aviary will indicate the common bacterial species most likely to be involved in new cases. *E. coli, Salmonella, Pseudomonas,*and *Staphylococcus* are among the more common genera identified.

“Retained yolk sac” in ratites

So-called “retained yolk sac” in ratite chicks is a common clinical challenge faced by ratite veterinarians in the southeastern United States. An affected chick’s growth lags behind that of it normal siblings, and progressively severe dyspnea is a common clinical sign. Clinical relief is usually rendered by vitellectomy - surgical removal of the “retained yolk.”

At the Schubot Center Avian Diagnostic Lab we have observed that so-called retained yolks occur, with few exceptions, in chicks that were, at hatching, “wet chicks”, i.e. suffering from *hydrops feti* (generalized edema of the fetus/hatchling). In the relatively humid southeastern United States this is usually associated with inadequate water vapor loss from the egg during incubation- the result of uncontrolled excessive humidity in the incubator. The normal egg should lose approximately 15 to 18% of its

lay-weight before hatching. High ambient humidity prevents the incubator from being run at a sufficiently low humidity to permit appropriate water vapor loss from the egg. Dehumidification and air conditioning of the room in which the incubator operates obviates the problem.

The incidence of hydropic chicks increases as the percentage of egg weight lost during incubation decreases. It should be noted that hydropic hatchlings and "retained yolks" are relatively uncommon to rare in the more arid regions of the American southwest where low ambient humidity permits more accurate control of the incubator humidity. I'm more than mildly curious as to whether or not a similar dichotomy obtains among ratite embryos artificially incubated in the arid regions and those in the more humid regions of Australia.

Some surviving "wet chicks" grow normally and survive; others grow slowly and finally exhibit the distended abdomen and dyspnea usually observed in chicks with "retained yolk". At surgery or necropsy the yolk sac is not only retained (not involuted to a normal Meckle's diverticulum) but massively distended and filled not with normal yolk, but with a thin to watery, yellow-green fluid. Affected chicks usually die of suffocation resulting from compression of the caudal air sacs to potential spaces. The chick's tidal volume has essentially been reduced to zero.
This pathologist hypothesizes that the pathogenesis of so-called "retained yolk" is the result not of abnormal retention of the yolk and its sac *per se*. Rather, it results from abnormalities in the control of active and/or passive movement and transport of water, electrolytes, and osmotically active molecules across the yolk sac membrane peculiar to chicks that suffered hydrops as embryos and hatchlings.

The above may ultimately be shown to be merely another example of fallacious *post hoc, ergo propter hoc*reasoning, but circumstantial evidence suggests a reasonable basis for the stated hypothesis.

Small intestine:

The small intestine of granivorous species, those consuming a relatively concentrated, high energy diet, is generally considerably shorter than that of omnivorous and carnivorous species. True villi - cylindrical, finger-like projections of the mucosal epithelium that greatly increase the absorptive surface of the intestinal lining - are the exception rather than the rule in most taxonomic groups of birds. Instead, many birds have paddle-shaped projections, or accordion-pleated longitudinal folds that nestle parallel to their neighbors. The relatively short intestine of seed-eaters combined with a relatively short passage time of digesta combine to amplify the clinical significance of intestinal diseases.

Diarrheal Diseases:

Most birds presented to the veterinarian with the complaint of diarrhea are, in fact,

producing “loose” droppings as a result of polyuria. True diarrhea - the loss of abnormally fluid feces from the intestinal tract - does, however, occur and the pathogeneses of avian diarrheal diseases are essentially the same as those of mammals.

Exudative enteritis with associated diarrhea is usually the result of fibrinous, fibrino-necrotic, or hemorrhagic-necrotic enteritis of bacterial infection by *Salmonella sp.* or *Clostridium sp*. or of certain enteric protozoal infections (coccidiosis, histomoniasis).

Hypersecretory diarrhea is most often the result of the action of toxins of enteropathogenic *E. coli* upon the intestinal crypt secretory epithelium. At necropsy such birds show no gross or histological evidence of enteritis or of any degenerative changes of the mucosal epithelium.

Malabsorptive diarrhea is usually the result of extensive granulomatous enteritis caused by *Mycobacterium avium / intracellulare* infections. Intestinal infection by *Giardia sp.* and *Cryptosporidium sp.* also impair the function of absorptive epithelium and result in diarrhea with no gross or microscopic evidence of enteritis.

Osmotic diarrhea has been suggested as a possible result of feeding pet birds dairy products because they lack intestinal mucosal lactase. Accumulation of the fermentation products of disaccharides is thought to promote osmotic fluid loss into the intestinal lumen. However, the gut-passage time of granivorous birds is so brief that the liklihood that any significant amount of fermentation could take place is minimal.

The fallacy of “hemorrhagic enteritis” and “melena”

Although hemorrhagic enteritis and melena can and do occur in birds, such events are relatively uncommon to rare. The frequent gross necropsy diagnosis of “intestinal hemorrhage” and “melena” are only rarely confirmed by histopathology. In the great majority of cases the observed “black, tar-like” intestinal and cloacal contents is, in fact, chyme and feces heavily stained with dark bile pigment. Such bile-staining is a common, expected consequence of a period of recent anorexia, which is, of course, evidenced by many ill birds. When the putative “black, bloody feces” is smeared out thinly on a piece of white paper or unglazed white ceramic its deep verdure becomes evident.

The fallacy of “Omigosh! *E. coli*’s in the aviary! Goddagedidout”

Members of the *Enterobacteriaceae* are components of the intestinal flora in less than 25% of captive parrots and finches. The majority of isolates, however, are *E. coli* and are gained from clinically normal birds. The mere isolation of *E. coli* from healthy birds should not elicit an immediate, knee-jerk call-to-arms requiring elimination of the organism. Let’s get real. Such birds are not necessarily suffering “enrofloxacin deficiency.”

Temper the tendency toward precipitate action - to inflict a pre-emptive strike, as it were. Unless there is definitive evidence of disease caused by *E. coli* in the population, the potential risks inherent in exuberant antibiotic therapy should be weighed against the putative benefits of eliminating what is likely a non-pathogen. Disruption of the established alimentary tract microflora may negatively impact the health of the individual bird or aviary population. For example, elimination of megabacteria, or disruption of the possible benefits of competitive exclusion of enteric pathogens by normal flora are potential risks.

Fabricial (Cloacal) Bursa :

The Fabricial bursa, which involutes prior to sexual maturity, is given little credit beyond its admittedly indispensable role as the source of the humoral component of the immune system - the B-lymphocytes - and it usually receives short diagnostic shrift in avian medicine. It is often assumed that the hatchling is relatively immune-incompetent until its secondary lymphoid organs are adequately colonized by B cells (and T cells from the thymus) and must rely on maternal, passive immunity provided by immunoglobulins absorbed from yolk and/or received via parental regurgitus.

The locus of the bursa appears to be more than mere happenstance. The vent and cloacal proctodeum accomplish a feat impossible were the bursa to have been located elsewhere. The vent and cloaca physically ingest fine particulate material from the hatchling's local environment. Carbon-black applied to the vent can be demonstrated within a few hours in the deepest recesses of the bursal lumen and in intimate approximation to the bursal lymphoid follicles. An admirable bit of anatomical engineering, it would seem, to promote early stimulation of immunocompetent cells at their primary site of production by potential infectious agents which might be more successful pathogens by some other route of exposure.

Unfortunately, this propensity for enhanced antigenic stimulation occasionally backfires when the local environment is contaminated with pathogens capable of establishing bursal infection. Fabricial Bursa infections caused by parrot polyomavirus, *Chlamydia psittaci*, and *Salmonella typhimurium* suggest that the vent/cloaca route should be considered, as well as the oral and respiratory tract routes, when evaluating nursery outbreaks of these diseases and proposing changes in the hygienic management of the aviary nursery. In the case of parrot and pigeon circovirus infections, the bursa is the most likely site of primary infection.

Although by no means an exhaustive coverage of all the unique, clinically significant features of the avian alimentary tract, it is hoped that the foregoing has provided some new perspectives from which to consider the avian alimentary tract in health and disease.

Recommended Reading on the Avian Alimentary Tract:

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