CHAPTER
14

Evaluating and Treating the Gastrointestinal System

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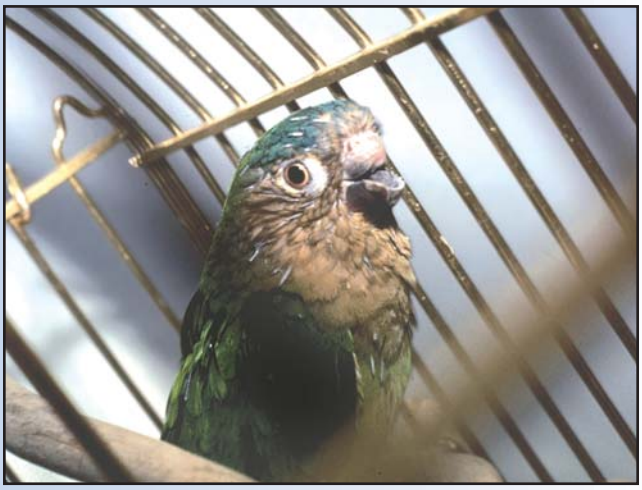


Greg J. Harrison

The avian gastrointestinal tract (GIT) has undergone a multitude of changes during evolution to become a unique anatomical and physiological structure when compared to other animal orders. On the one hand it has evolved to take advantage of the physical and chemical characteristics of a wide variety of food types.[1] On the other hand, it has had to do so within the limitations of the requirements for flight.[2] To this end, birds have evolved a lightweight beak and muscular ventriculus, which replaces the heavy bone, muscular and dental structure characteristic of reptiles and mammals. The ventriculus and small intestine are the heaviest structures within the gastrointestinal tract and are located near the bird's centre of gravity within the abdomen. The overall length of the GIT is also less than that of a comparable mammal, another weight-saving flight adaptation. Interestingly, these characteristics are still shared with the flightless species such as ratites and penguins. In addition, the actual digestive process needs to be rapid to support the high metabolic rate typical of flighted birds.[3]

Gastrointestinal adaptations to the wide range of ecological niches that birds occupy mean that birds can take advantage of a huge variety of foodstuffs. The GIT hence shows the greatest degree of diversity of all the organ systems between different avian taxa. However, the pressures of convergent evolution have also meant that many distantly related species have developed a similar gastrointestinal anatomy to take advantage of particular food niches.[3,4] Examples of these will be presented in the discussion of each section of the GIT.

The avian GIT also has the capacity to accommodate changes which occur during the life cycle of a bird and also which occur due to seasonal environmental conditions and hence differing available foodstuffs over the course of a year or years.

Anatomy and Physiology of the Digestive Tract

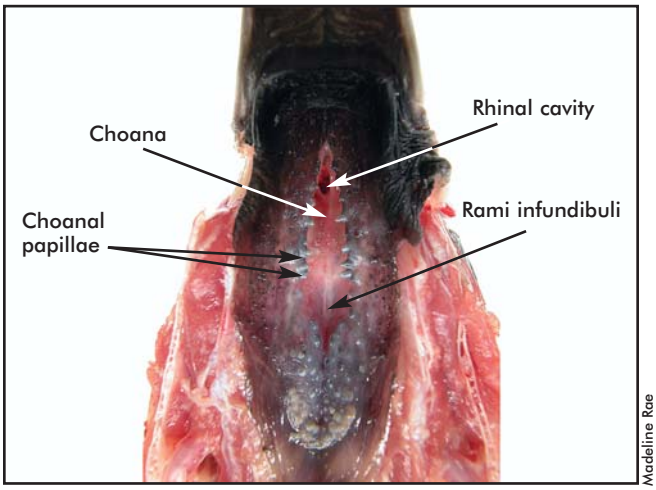
The avian gastrointestinal tract is a double-ended open tube (as is also seen in mammals) that begins at the beak and finishes at the vent. In sequential order it is composed of a mouth, esophagus, crop, proventriculus, ventriculus (gizzard), intestine, ceca, rectum and cloaca. Some of these structures may be vestigial or even lost during the evolution of some species. The progress of food through the tract follows a specific digestive sequence including premoistening and softening, acidifying, grinding, hydrolyzing, emulsifying and propulsion of the end products.[1] This propulsion is not always in a unidirectional pattern as will be outlined later.

BEAK, MOUTH, TONGUE AND PHARYNX

The beak or bill is the avian substitute for teeth and lips and forms the entrance to the oral cavity. It is used for grasping and processing foods, as well as for climbing and various behavioral functions such as biting, preening and displaying. It consists of the mandibular bones, the premaxilla and maxilla and their horny covering, the rhamphotheca. The upper bill covering is known as the rhinotheca, which covers the premaxillary bones and partly covers the maxillary bones. It is usually composed of hard keratin, although in waterfowl only the tip is hard, and in shorebirds the entire bill is relatively soft.[2] The keratin layer covering the lower bill or mandible is known as the gnathotheca. Both keratin layers are continually lost by wear and replaced by new growth. Beak shape is influenced by the location and rate of wear and hence regrowth, which is in large part determined by diet. This may subtly change over time as food types change. For example, the anterior of the outer edges of the beak, the tomia, may be sharp in some species to assist in cutting seed coats.[1] Other anatomical characteristics further facilitate the feeding process. The lower beak is loosely attached to the skull, allowing for a large gape. The size of the gape determines the maximum size of food particles that can be swallowed. This is particularly important in fruit-eating species such as toucans. Psittacines typically have a very powerful beak. The rhinotheca is broad with a curved rostral tip, giving the typical hooked appearance. The gnathotheca has a blunt chisel-shaped rostrum that pushes against a prominent ridge found on the undersurface of the rhinotheca. Psittacines also have developed a prokinetic maxilla that allows them to move their mandible and maxilla independently. This allows an increased gape of the beak, an improved ability to position food items in the beak, as well as providing flexion and shock absorption associated with seed and nut cracking and also with some behaviors such as pecking.[5] The strength generated by these structures is exemplified by the Hyacinth macaw's ability to crack palm nuts in order to extract the kernel. Pigeons, on the other hand, have a typical seedeater's bill being mildly conical.[6] It is also not as keratinized as in psittacines. Raptors tend to also have curved, hook-like bills but lack the prokinetic maxilla.[6] Their bill is adapted to tearing and shredding meat. "Darwin's Finches" on the Galapagos Islands best exemplify the variability in beak shape brought about by the need to adapt to changing environments. Here, the species are all similarly colored, but are separated on the basis of bill shape and feeding habits. Each has evolved to its own ecological niche, avoiding interspecific competition.[7] The avian bill is often endowed with sensitive nerve endings, particularly in species that use the bill to probe for food. Examples include waders, diving ducks and woodpeckers.[3]

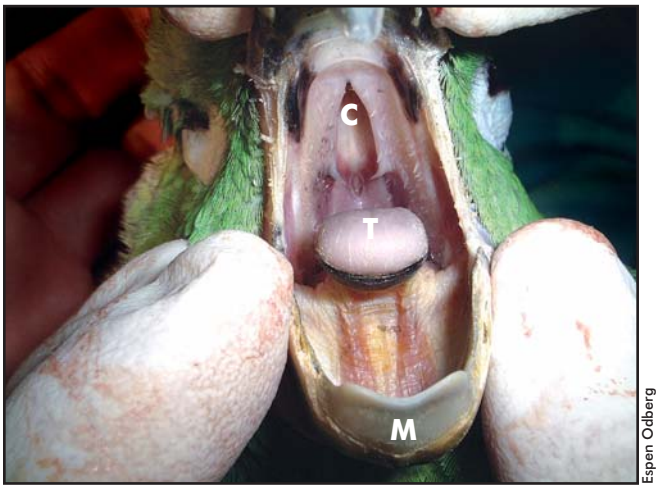
Unlike mammals, birds do not have a soft palate or a pharyngeal isthmus, nor do they have a sharp demarcation between the mouth and pharynx.[2] Instead, they have a combined oropharynx (Figs 14.1a,b). A longitudinal fissure, the choana, which connects the oral and nasal cavities, splits the palate. The choana is variably developed. In pigeons it is narrow and lacks papillae. In falcons it forms a narrow "V"- shape with few papillae. In psittacines, it forms a wide "V" and is bordered by caudally-pointed sensory papillae.[6] The choanal slit closes during swallowing. The infundibular cleft is located at the caudal edge of the choana and is the caudal opening of the left and right pharyngotympanic (Eustachian) tubes (*Rami infundibuli* Fig 14.1a) from the middle ear.[6] The palate forms a roof over the anterior part of the oral cavity. In finches, canaries, budgerigars and cockatiels it contains two ridges that assist in the removal of husks from seeds before ingestion.[1]

The tongue (Fig 14.1b) originates from the floor of the oropharynx and is mobilized by the hyoid apparatus and its multiple articulating bones and musculature. It functions to collect, manipulate and swallow food. Again, great species diversity exists in tongue development. The tongues of passerines and pigeons tend to be smooth, short and simple. Psittacines are unique amongst birds in having additional striated muscles in the anterior regions of their tongues that are independ-



Madeline Rae

Fig 14.1a | Ventral dorsal view of the palate area of the orapharnyx.



Espen Odberg

Fig 14.1b | Frontal view of orapharnyx of an Amazon parrot. T=tongue, M=mandible, C=choanal slit. Note the reduction of papillae.

ent of the hyoid apparatus and permit added flexibility and manipulative capabilities.[1] The typical psittacine tongue is thick and muscular and its maneuverability allows for the extraction of seeds and nuts from their husks, cones or pods. Lories and lorikeets have relatively long tongues that end in fine papillae that aid in the harvesting of pollens and the collection of nectar from flowers by capillary action.[8,9] In birds of prey the tongue is rasp-like, with a roughened tip and many small caudally pointed papillae near the base.[6,10] In ducks and other waterfowl which strain food particles, the rostral part of the tongue is scoop-like and has a double row of overlapping bristles on its lateral borders. These bristles work with the beak lamellae to filter particles.[2] It is interesting to note that birds have poor taste sensitivity compared to humans. For example, parrots have approximately 350 taste receptors compared with 9000 in humans[11], and chickens have up to 300 taste buds.[12] These are mostly located on the palate near salivary glands and on the posterior tongue. However, the beak, tongue and oral cavity have many touch receptors that make the mouth an important sensory area.[1]

The laryngeal mound lies immediately behind the tongue in most species and contains the glottis, the opening to the trachea. In most species the glottis lies directly under the caudal portion of the choana or just caudal to the choana in raptors. The laryngeal mound contains rows of caudally directed papillae that assist in the propulsion of food towards the esophagus during swallowing.[2] Birds lack an epiglottis.

Salivary Glands

There is great species variability in the number and distribution of salivary glands.[2] Granivorous species such as some parrots, pigeons, chickens and finches have a large number of glands to assist in swallowing the dry feeds they ingest. These glands are located in the roof, cheeks and floor of the oropharynx. Raptors have less developed salivary glands and piscivorous (fish-eating) species have poorly developed glands, or lack them all together. This is presumed to be related to the lubricated nature of the food they ingest. The content of the saliva produced also varies between species. The salivary glands of house sparrows secrete significant amounts of amylase whereas those of chickens and turkeys secrete little amylase.[13]

Esophagus and Crop

The esophagus is a thin-walled distensible tube that delivers food from the oropharynx to the proventriculus. It allows birds to swallow their food items whole. In birds the esophagus is divided into a cervical and a thoracic region. In the budgerigar, it lies dorsal to the trachea in the anterior regions of the neck and then runs along the right side.[14] The esophagus' distensibility is facilitated by a number of longitudinal folds. These folds are large and extensive in owls (Strigiformes) and species that swallow whole prey items, or those that store large amounts of food material such as gulls (*Larus* spp.). By contrast, parrots exhibit minimal esophageal fold development and possess a relatively narrow esophagus.[6] Mucus-secreting glands are present in the esophageal mucosa of most birds, particularly in the thoracic esophagus. These glands are actually absent from the cervical esophagus of budgerigars.

The avian esophageal wall consists of a mucosa, submucosa, a muscular tunic and a serosal layer. It generally contains only smooth muscle cells with a circular muscle layer predominating.[10] Peristaltic contractions of inner circular and outer longitudinal muscles propel food posteriorly through the esophagus.[1]

The crop or ingluvies is an expansion of the cervical

esophagus that functions as a food storage organ.[1,2,6] It mostly lies on the right side of the neck and when distended may also lie on the left side and will rest on the furcula. The crop has varying degrees of development in different species. In its simplest form, it is merely a spindle-shaped enlargement of the cervical esophagus. This arrangement is seen in ducks (*Anas* spp.) and owls.[6] Parrots have well-developed crops that lie at the caudal cervical esophagus. A prominent right pouch and a small left pouch typifies parrots. Pigeon crops have a more complicated structure. Both right and left lateral pouches or diverticulae are well-developed. The lateral pouches produce a holocrine secretion from the crop epithelium — "crop milk" — which is fed to the squabs during the breeding season.[15] It is produced in response to prolactin. It contains 12.4% protein, 8.6% lipids, 1.37% ash and 74% water.[2] Therefore it is mainly a protein and fatty acid source for these chicks, and is devoid of carbohydrate and calcium. Both males and females produce crop milk. Sheets of striated muscle that attach to the crop adventitia support the large crop. Parrot and pigeon crops possess a functional sphincter at the junction of the crop and the thoracic esophagus. This helps to form and regulate the boluses of food being propelled to the proventriculus.[6] It should be noted that birds lack the true upper and lower esophageal sphincters found in mammals.[1] Some granivorous species such as the European goldfinch (*Carduelis carduelis*) lack a true crop but have a very expandable esophageal pouch that can store food items.[1] Gulls, penguins and ostriches lack a crop but have a very distensible esophagus.[2]

The crop's storage function allows birds to ingest and store feed in the evening before roosting, thus providing for overnight energy needs. It also allows birds to rapidly ingest food items in a short period of time, and then take refuge in safe cover where the meal can be digested at a more leisurely rate. The crop also acts to soften ingested food by holding swallowed water and by contributing mucus to the saliva. Enzymes within the food or microbes present in the crop may further contribute to digestion.[16,17] Any glucose released in the crop can be absorbed by the crop mucosa, but this is of minimal importance.[2]

The crop is particularly well-developed in chicks to store food fed by the parents. The parents of altricial chicks premoisten and soften food in their crops and esophagus before regurgitating it to their chicks. The crop also provides an important immunological function in pigeons feeding squabs.[15]

THE AVIAN STOMACH: PROVENTRICULUS AND VENTRICULUS

The avian stomach consists of 2 distinct structures, the first part being the proventriculus or glandular stomach and the second structure is called the ventriculus (gizzard) or muscular stomach.

The relative size and shape of these structures is based on diet and is hence quite variable. In carnivorous and piscivorous species both structures are very distensible and may be difficult to differentiate grossly. This is due to the soft nature of their diet. In birds that eat hard food items, the proventriculus is relatively thin-walled and glandular. The ventriculus is muscular, thick-walled and powerful. The intermediate zone connects the two.[2,6] This gastric arrangement is typical of granivores, omnivores, insectivores and herbivores and hence most of the commonly found species in captivity.

The proventriculus is confluent with the esophagus cranially but has its own distinctly different structure. It lacks ridges, except in carnivorous and piscivorous species, and is lined with a mucous membrane. Its epithelium contains two principal types of glands that make up most of the thickness of the proventricular wall.[2] The first of these, the tubular glands, secrete mucus. The second type, the gastric glands, secrete hydrochloric acid and pepsin. This provides an acidic environment for digestion. Typically, the fasted chicken has a pH of 2.6, whilst that of a pigeon is 2.1.[6] Nectarivorous parrots have gland-free spaces between the longitudinal rows of glands. This allows for distension of the glandular stomach that may be an adaptation to pollen digestion.[16,18]

The proventriculus contains two muscular layers, the innermost circular layer and the outer longitudinal layer. The outer longitudinal layer is poorly developed or absent in parrots, waterfowl and some passerines. In these birds the myenteric plexus is located immediately under the serosal layer rather than between the two muscle layers.[2]

The intermediate zone between the proventriculus and ventriculus is aglandular and lacks folds. In parrots and pigeons, it closes tightly during ventricular contractions to segregate the ventriculus from the proventriculus.[6]

The ventriculus or gizzard has evolved to mechanically break food down. Hence it is best developed in species that ingest hard foods such as granivores,[1,2,6] and also in insectivores that need to break down the hard exoskeletons of their prey.[18] It is also the location where hydrochloric acid and pepsin can further chemically break food particles down. It consists of two pairs of opposing muscles. The caudoventral and craniodorsal thin muscles line the caudal and cranial sac of the gizzard respectively. The cranioventral and caudodorsal thick muscles are responsible for the powerful grinding

contractions seen in the gizzard.[16] The asymmetrical arrangement of these four muscles provides mixing and grinding actions during contractions.[1] The ventriculus is lined by the koilin, a cuticle layer, which acts as a grinding surface and protects the underlying mucosa from the acid and pepsin produced by the proventriculus. The koilin is made up of a combination of proteinaceous rod-like projections produced by the deep tubular glands lining the gizzard, together with desquamated epithelial cells that form a matrix.[1,2,6] The hardened composite frequently has raised areas and distinct longitudinal and transverse grooves that aid in mechanical breakdown of foods. It is thickest in species with well-developed, muscular stomachs. It is continuously worn and replaced in many species, but in falcons it may occasionally be sloughed and shed. The koilin lining may be green, brown or yellow in color due to bile staining caused by ventricular reflux from the small intestine. This is a normal finding.[6]

The gizzard is separated from the small intestine by a small pyloric fold that regulates the passage of food into the small intestine by slowing the movement of large particles.[19] In lorikeets and honeyeaters (both nectarivorous species), the proventricular and pyloric openings of the gizzard lie in a median plane, which is thought to allow rapid passage of ingesta.[8]

It is interesting to note that the size of the gizzard can change with diet within the same species, being thicker and larger when dry seeds are eaten and softer and lighter in summer when fruits are eaten.[1]

The role of grit in avian digestion is an interesting one. Insoluble grit may lodge in the gizzard and add to the maceration of the food, particularly in species that do not dehusk the seed before swallowing it, eg, pigeons, and galliforms like quail. It is controversial whether birds deliberately seek insoluble grit to aid in digestion or whether its ingestion is incidental to eating digestible foods or soils containing minerals and trace elements.[6] Grit is absent from the stomachs of nectarivorous birds, which also have poorly developed gizzards.[18]

GASTRIC MOTILITY

Food passes through the proventriculus very quickly where it is coated with hydrochloric acid and pepsin, with little enzymatic digestion. Digestion is controlled by the vagus nerve and by the hormones gastrin, secretin, cholecystokinin and pancreatic polypeptides. The food is propelled into the ventriculus where most of the mechanical digestion occurs by a combination of coordinated muscle contractions and the action of grit. The exact process varies with species. In turkeys and parrots, for example, the muscles contract in a clockwise direction around the ventriculus.[6,20] The paired thin muscles contract first and the isthmus closes, segregating the ventriculus from the proventriculus. As these muscles reach maximum contraction, the pylorus opens allowing digesta to pass into the duodenum. The thin muscles then relax as the thick muscles contract. This coincides with the closing of the pylorus and the beginning of peristaltic contractions along the duodenum. The isthmus may also open to allow the passage of ingesta back into the proventriculus for the addition of fresh acid and pepsin, allowing additional time for the breakup of large lipid globules and the breakdown of proteins. In particular, lipid is retained in the anterior region of the tract and digested more slowly than are protein or carbohydrates.[21,22] This cycle of contraction occurs as a seamless movement that gives the appearance that the gizzard is flipping.[6] This coordinated complex of contractions is controlled intrinsically by the myenteric plexus.

Raptors have a more simple stomach arrangement that has to produce the pellets, indigestible contents of fur, bone, teeth, feathers, claws, as well as perform the normal digestive function. Neck extension and head pumping assist the peristaltic propulsion of food into the proventriculus. The stomach fills with digestive juices over the next hour and vigorous, high frequency waves of contractions occur in a clockwise direction from the isthmus to the pylorus. This is followed by a 7- to 9-hour period of chemical digestion where forceful proventricular contractions occur at low frequency. By the end of this period, digestion is complete. Next, a short phase of paired contractions removes any further liquid from the indigestible pellet. This is followed by a further 5- to 6-hour phase of pellet compaction after which it is expelled by retroperistalsis. The timing of pellet ejection varies with the species.

INTESTINES AND PANCREAS

The small intestine is the main site for enzymatic digestion and nutrient absorption in the avian gut. It is less differentiated between species than are the more proximal regions of the gastrointestinal tract. The duodenum arises from the pylorus and forms a loop that encircles the bulk of the pancreas. The pancreas is trilobed in most species with the third lobe or splenic pancreas sometimes not being directly attached to the other two lobes. In budgerigars, the three pancreatic lobes are each drained by a separate duct. Two of these ducts empty into the distal duodenal loop adjacent to the bile duct whilst the other duct empties into the opposite side of the duodenum.[14] In pigeons, all three pancreatic ducts empty into the distal duodenum.[6] The exocrine pancreas contains enzymes similar to those found in mammals such as amylase, lipases, trypsin and chymotrypsin,

carboxypeptidases A, B and C, deoxyribonucleases, ribonucleases and elastases.[1] It also produces bicarbonate that buffers the intestinal pH. It is also important to remember that the intestinal wall mucosa also produces amylase, maltase, sucrase, enterokinase, lipases and peptidases and so contributes to enzymatic digestion.[6] These enzymes are produced in response to duodenal distension, hydrochloric acid, vagal stimulation, cholecystokinin, secretin and vasoactive intestinal peptide.[2,23] Birds have not been shown to possess any intestinal lactases so they should not be fed significant quantities of lactose-containing foods.[24] Amylase levels are actually highest in the jejunum, but the jejunum and ileum cannot be readily differentiated from the duodenum in birds. In general, the jejunum is thought to begin just after the ascending duodenal loop begins to turn back on itself, where the jejunal branches of the cranial mesenteric artery begin. The ileum is thought to begin at the vitelline (Meckel's) diverticulum and end at the recto-cecal junction.[6] There is great variation in jejunal and ileal anatomy in different species.

Nectarivorous and insectivorous birds have shorter intestines than do similar sized granivorous or herbivorous species.[25] This is believed to be due to the highly digestible nature of their diet.

The intestinal epithelium contains villi, microvilli and crypts. The villi's increased surface area allows efficient absorption of nutrients and their rich capillary system enables transport of these nutrients to the portal blood system. A thick layer of mucus produced by goblet cells in the epithelium protects the intestinal epithelium from the digestive juices and from physical abrasion, particularly anteriorly near the gizzard. Two muscle layers surround the intestine, the inner circular and outer longitudinal layers that allow mixing and propulsion of the digesta through the intestinal tract.

The avian duodenum is unique in its ability to exhibit both normograde and retroperistalsis.[6] These retrograde peristaltic waves bring the digesta back towards and into the ventriculus, as is evidenced by the presence of bile staining in the ventricular koilin. These waves are powerful and visibly distinct from the normal peristaltic waves. They occur every 15 to 20 minutes in the turkey[20] and up to once a minute in parrots on a moderate fat diet.[6]

The liver also empties into the distal duodenum via the bile ducts. Its primary digestive function is the production of bile acids and salts that assist in the emulsification of fats, allowing their digestion by lipases. These acids and salts, together with cholesterol and phospholipids, are secreted into the bile canaliculi that drain into the bile duct. Gall bladders are present in raptors and waterfowl, but are absent in many psittacines and pigeons.[6,26]

The ceca are important in fermentation of vegetable matter and in water balance and are hence most developed in chickens, ratites and ducks.[27] They are absent or vestigial in parrots and small insectivorous passerines, appearing histologically as a nodule of lymphatic tissue at the small intestinal-rectal junction. In the domestic pigeon they are entirely lymphatic in structure and are called the cecal tonsils. They are, however, well developed in herbivorous or omnivorous passerines.[16]

The avian rectum or colon is found between the ileocecal junction and the cloacal coprodeum. Except in the ostrich, it is very short and has a smaller relative diameter than the mammalian large intestine and is structurally dissimilar, being similar to the small intestine except for having shorter villi that are richer in lymphoid follicles. The avian rectum exhibits marked retroperistalsis, carrying urine from the urodeum and coprodeum into the colon up to the ceca.[6] This allows for further water resorption in the colon and hence aids in water conservation. In the pigeon the rectum enters the coprodeum from the right side, whereas in the parrot it enters from the left side at a 60 to 90° angle.[6]

CLOACA

The avian cloaca is a three-chambered structure that is responsible for the terminal deposition of digestive, urinary and reproductive products. It is much wider than the rectum. The first, most proximal chamber, is the coprodeum into which the rectum empties. It is the largest chamber of the psittacine cloaca and has a flat, vascular, avillous mucosa, covered by columnar epithelium and an extensive branching vascular pattern.[6,28] It is separated from the second chamber, the urodeum by an encircling sphincter-like ridge, the coprodeal fold. This fold can completely close off the coprodeum from the other chambers of the cloaca, preventing contamination of eggs or semen during egg laying or ejaculation.

The urodeum is the smallest cloacal chamber in psittacines, columbiforms and falconiforms. It receives the ureters and also the oviduct in females and the ductus deferens in males. The ureters enter the urodeum on either side of the dorsal midline, and in pigeons and parrots these openings are simple. In females, the oviduct has a rosette-like opening on the left dorso-lateral wall. It is smaller and less prominent in juveniles and hence difficult to visualize in these birds.[28] A membranous tissue may occlude this opening in females that have not yet laid in species such as the ostrich.[29] In males, the ductus deferens enters the urodeum on symmetrical, raised papillae located on the left and right dorsolateral walls. It is separated distally from the proctodeum by the uroproctodeal fold. The urodeal mucosa is smoother and less vascular than that of the coprodeum.

The urodeum exhibits retroperistalsis, pushing urates and urine cranially into the coprodeum and rectum where water and solutes are further resorbed, thus maximizing water conservation.[6,21,30] This retroperistalsis explains why urates and feces are sometimes intertwined when passed from the cloaca.

The proctodeum is the final cloacal chamber and is slightly larger than the urodeum in most species. The uroproctodeal fold is more developed dorsally and gradually loses prominence ventrally. This chamber is the most frequent site of papillomas in psittacines. It also gives rise to the Bursa of Fabricius on the dorsal midline just caudal to the uroproctodeal fold. The bursa is most prominent in the juvenile bird where its lymphoid tissue is responsible for the production of B-lymphocytes. In mature birds the lymphoid tissue involutes but the bursa's opening and chamber frequently persist and can be viewed during cloacoscopy. The timing of bursal involution is usually between 2 and 6 months of age, but varies between species.[6]

The cloacal blood supply is via the pudendal artery and vein. Innervation is via the pudendal nerve that follows the ureters to the dorsal cloacal wall where the cloacal ganglia are found.[28] These are important surgical landmarks.

The final structure of the gastrointestinal tract is the vent, a transverse opening in the ventrocaudal body wall through which body wastes and reproductive products are expelled. It is demarcated by lips dorsally and ventrally and is surrounded by voluntary muscles that form a sphincter. This provides birds with some control over defecation. For example hens that are incubating may pass a large urofeces in the morning when nest changeover occurs. Some psittacines can also be toilet trained to defecate on command. The act of defecation involves the partial eversion of the vent lips, resulting in the formation of a circular orifice through which feces, urates and urine can be expelled.[1]

Cloacal "sucking" has been noted in psittacines in juveniles and breeding females, where material is brought in from the outside via the vent lips under negative pressure.[28,31] In chicks, this is thought to have an immune stimulation function by exposing the B cells in the bursa to external antigens. In breeding females it is thought to facilitate sperm transport and hence fertilization in species where the male lacks a phallus.[31] See Chapter 18, Evaluating and Treating the Reproductive System for further discussion of the vent.

Gastrointestinal Diseases

DISORDERS OF THE BEAK

Deformities

Deformities of the bill of young birds, both congenital and acquired, have been described. Congenital deformities have been mostly described in poultry and waterfowl. Some of these are part of a more generalized problem, such as Micromelic Syndrome of white Pekin ducklings, an autosomal recessive mutation which causes a short maxilla, reduced overall size, shortened limbs, cervical subcutaneous edema and abnormal feathering.[32] Others are specific to the beak such as variations in the shape and curvature causing malocclusion, often leading to the maxilla being caught inside the lower mandible (prognathism) (Fig 14.2).[33] "Scissor-beak" is a condition where the upper beak rhinotheca is bent to one side, resulting in the overgrowth of the gnathotheca in psittacine chicks (Fig 14.4a). The condition becomes progressively worse due to the continued forces applied during the bird's normal beak usage and as the chick grows.[34,35] It has also been noted in other avian species such as ostriches,[36] softbills and passerines[32] where either the mandible or maxilla may deviate. Multiple potential etiologies have been described including heredity, incubation problems, malnutrition, infectious sinusitis, viral diseases and trauma.[36,37,38*]

In young psittacines, incorrect hand feeding techniques may result in bruising of the rictus on one side of the beak, leading to uneven growth and scissor beak.[38,39] **Eds. Note: In most psittacine rearing facilities establishing a formulated diet program for adults and youngsters has eliminated these problems completely. Facial bones are not as malleable in properly fed parents or their offspring.* Treatment involves altering the forces that direct the rostral growth of the affected part of the beak. Surgical techniques to achieve this have been described.[38,40] In addition, revision of incubation and chick feeding practices and nutrition may be warranted.

Mandibular compression has also been described in young macaws.[35] Prognathism, where the upper beak tucks into the lower beak, is another congenital beak deformity sometimes seen in chicks, particularly cockatoos.[35,41] The etiology of this condition is unknown. If attended to early, it can be corrected with physiotherapy by applying traction rostrally to the maxillary beak several times daily. If the maxillia is calcified, physiotherapy and beak trimming may help.[42] In more severe cases, dental acrylic prostheses (Fig 14.3) can be applied to the tip of the maxillary beak to force it to stretch out over the mandible[41] or KE wires and rubber bands or cable ties can be used in a modified Doyle technique (Figs 14.4a-f, 14.5a-f).[40]


Greg J. Harrison

Fig 14.2 | Baby umbrella cockatoo with mandibular prognathism.


Greg J. Harrison

Fig 14.3 | Acrylic applied to beak tip to allow new growth pressure to overcome the prognathic condition. A metal transverse pin made from a hypodermic needle is often implanted to help the acrylic maintain adhesion to the beak.

Crusty scab-like lesions at the commisures of the beak have been associated with biotin and pantothenic acid deficiencies in gallinaceous birds and ostriches.[36,43] Vitamin D and calcium deficiencies have resulted in soft beaks due to insufficient mineralization in many species.[33,44] Malnutrition and hypovitaminosis A were associated with significant beak deformities in hand-reared African grey parrot (*Psittacus erithacus*) chicks (see Chapter 5, Calcium Metabolism).[44] These were seen as significant grooved ridges and indentations of the rhinotheca and gnathotheca. Birds with rhinothecal overgrowth characterized by intralaminal hemorrhages have often been diagnosed with previous or current liver disease and malnutrition.[44] Assessing liver function, correcting the diet and trimming the beak as required are all useful management tools.

Traumatic Lesions

Traumatic lesions to beaks are amongst the most commonly seen problems. These frequently occur as a result of intra- or interspecific aggression, parent birds that mutilate chicks, accidents or predator attack. Iatrogenic causes have also been described from incorrect handling, use of mouth specula or incorrect beak trimming.[39] It should be remembered that the avian beak lacks a subcutis, and hence the thin dermal fibrovascular stroma is directly apposed to the periosteum. Therefore pressure necrosis of parts of the rhamphotheca and gnathotheca can lead to permanent beak defects.[46] Immediate treatment for trauma cases involves stopping hemorrhage, counteracting shock via fluid therapy, providing analgesia and preventing infection. Nutritional support then needs to be provided.[39,47] The decision as to how to best manage the injuries needs to be made early, to decide if indeed the bird is salvageable or if the owner is prepared to accept the care and cost involved with long-term management. Loss of the distal third of the bill has potential for regeneration, at least in psittacines,[48] but not in other species such as ostriches (*Struthio camelus*) in which beak growth stops in adulthood.[37] Trimming and reshaping using a mild grinding tool, such as an electric motor drill, can treat minor distal fractures. Rhamphothecal fractures can be stabilized with tissue glues such as cyanoacrylate (Figs 14.6a-i).[49,50] More serious fractures may require surgery with pins, wires, sutures, plating or acrylic remodeling techniques, depending on species, patient size, nature and location of the injury or fracture (Fig 14.7).[37,47,51,52] It should be noted that fractures and avulsions of the upper rhamphotheca are the most challenging due to the kinetic nature of the maxilla (in psittacines), the forces exerted and the presence of small bones.[47] Damage to the germinative layer of the rhamphotheca or gnathotheca or of the underlying bone means that the affected area will not regenerate keratin (Fig 14.8).[50,52] If the associated beak structure has been avulsed and if the damage is great enough, the entire mandible or maxilla may be lost and not regenerate. Acrylic prosthetics have been used as a temporary means of restoring beak function and appearance until new keratin growth occurs.[52] In permanent injuries, these prosthetic beaks need to be remodeled, replaced or reapplied on a regular basis, as they invariably work loose. In cranes, this is every 3 to 6 months.[53] Natural prosthetic devices have been successfully used in toucans utilizing the beaks from dead birds of the same or similar species. The surgeon should instruct the owner that these prostheses are also, like the acrylic or metal repairs, only temporary.[47]

Palatine bone luxation has been described in blue-and-gold macaws (*Ara ararauna*) following trauma in which


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Fig 14.4a | Scissor beak in an umbrella cockatoo.

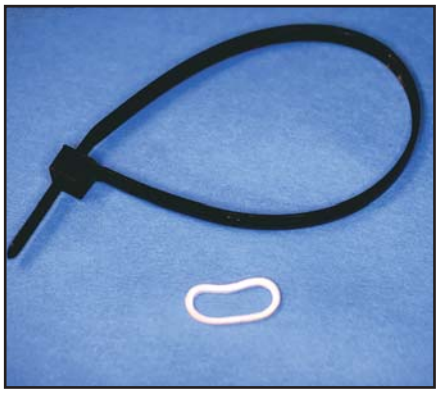
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Fig 14.4b | Bands used for beak orthodonture. A electrician's black cable tie and an orthodonture rubber band are shown.

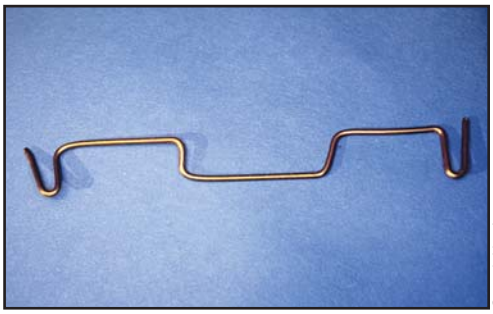
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Fig 14.4c | The final shape of the transverse sinal pin for a beak traction technique.


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Fig 14.4d | Band applied to beak with enough pressure on the transverse sinal pins to correct the deviation.

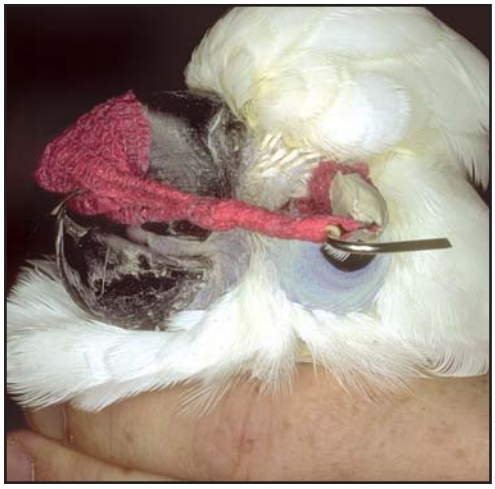
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Fig 14.4e| Bandage material is placed over the rubber bands to avoid removal or becoming snagged.


Greg J. Harrison

Fig 14.4f | Several weeks after correction and removal of appliance. Traction was applied for 2 weeks.

the palatine bones became hooked onto the interorbital septum.[40,54] The luxation was reduced under general anesthesia by using digital pressure on the maxilla both directly and via an intramedullary pin placed through the infraorbital sinuses.[54]

Infectious Causes of Beak Malformation

Various infectious disease processes can involve the beak. Psittacine circovirus disease (Psittacine Beak and Feather Disease) infects numerous psittacine species, both wild and captive, where it can cause beak lesions as part of the chronic presentation of the disease, particularly in young cockatoos.[55] Affected beaks typically become abnormally elongated and may develop transverse or longitudinal fractures.[56,57] In some cases only the tips may be fractured. There may be necrosis of the palate and ulcers of the mouth. As the disease progresses, the beak may fracture and avulse, exposing the underlying bone, which can be very painful. Secondary bacterial and fungal infections may complicate the infection and cause life-threatening disease. Diagnosis is by PCR or HA (hemagglutination assay)[55,56] for presence of the virus or HI (hemagglutination-inhibition) for antibody levels.[56] Histopathology of affected tissues is also useful. Severely affected birds are usually euthanized, as there is no specific treatment for this disease except for supportive care which includes maximizing hygiene, providing soft foods, treatment of any secondary infections and immunostimulation.

Poxvirus infections are seen on the unfeathered regions of many avian species, but less commonly in psittacines.[57] This *Avipoxvirus* classically causes raised lesions which may or may not become necrotic and then secondarily infected. These may be found on the beak or at the beak/skin margin and also in the oropharynx. The beak and mouth may become painful and disfigured, and the bird may show reluctance to eat. Transmission requires direct contact with open wounds or inoculation via an insect vector. In one case, the basal layers of the beak epidermis were infected causing sloughing of the keratinized layers.[57] Although species-specific strains are transmitted by mosquitoes, cross-species infections may occur,

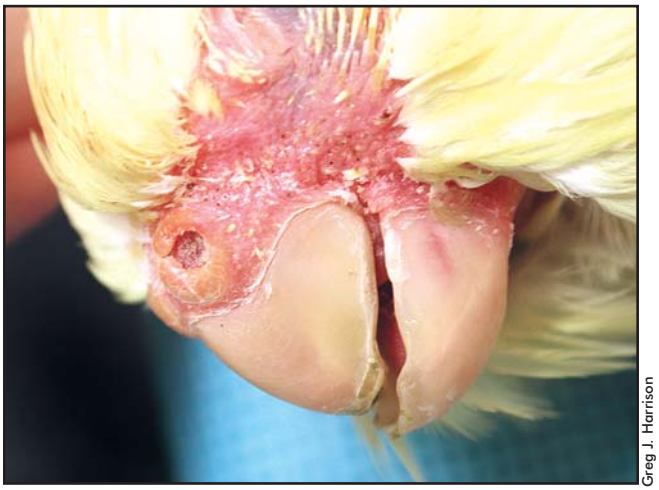
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Fig 14.5a | Young cockatiel that was having its beak ground as a first step in the therapy for prognathism.


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Fig 14.5b | A hypodermic needle is used to pre-drill a hole in the frontal bones. Then a stainless steel pin is placed transversely through the frontal sinus.

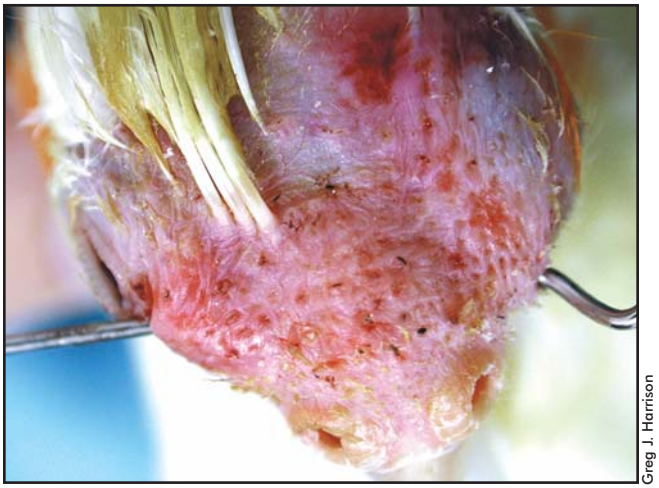
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Fig 14.5c | The first hook is bent into one end.

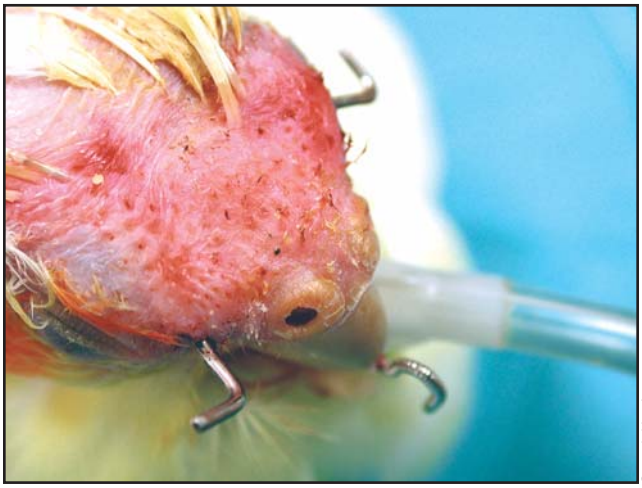
Greg J. Harrison

Fig 14.5d | A second bend is made in the transverse pin and a second S-shaped pin is formed and inserted in the distal rhinotheca.

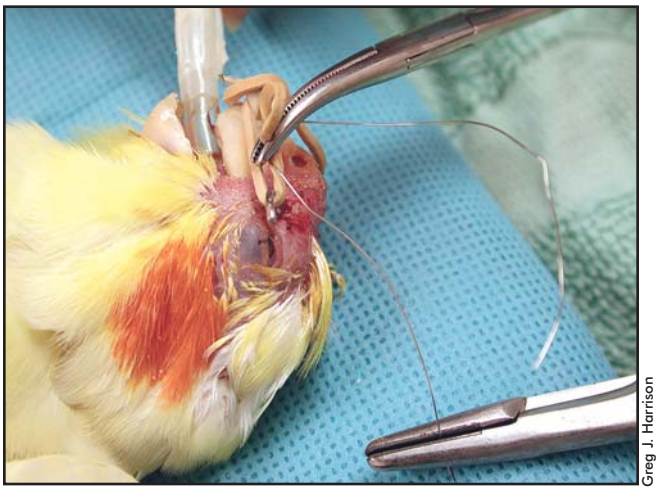
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Fig 14.5e | An orthodonture rubber band is placed around the left dorsal transverse sinus pin's hook and the ventral S-pin. A hemostat is placed around the tensed rubber band and stainless steel suture is placed to keep the traction on the rubber band once the hemostat is removed. The unused portion of the band is cut off.

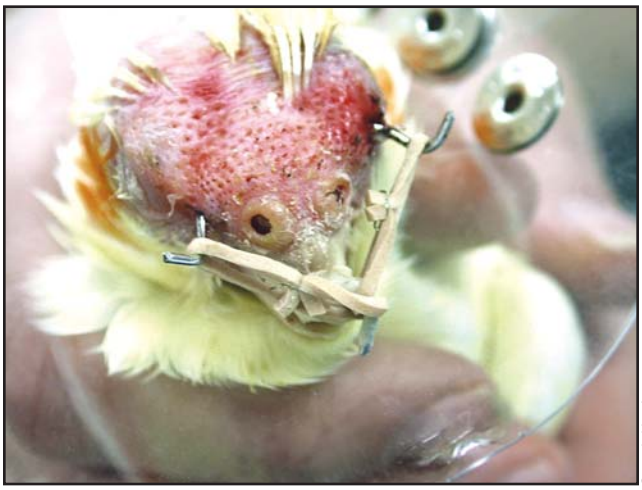
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Fig 14.5f | The finished traction device is in place. The extra length of the rubber bands have been cut off just above the stainless steel suture retention knot on the rubber bands. A plastic protective collar has been placed on the bird.


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Fig 14.6a | A sun conure has been bitten by a larger bird. The walls of the rhinotheca are fractured and compressed into the maxillary sinus diverticulum of the infraorbital sinus.

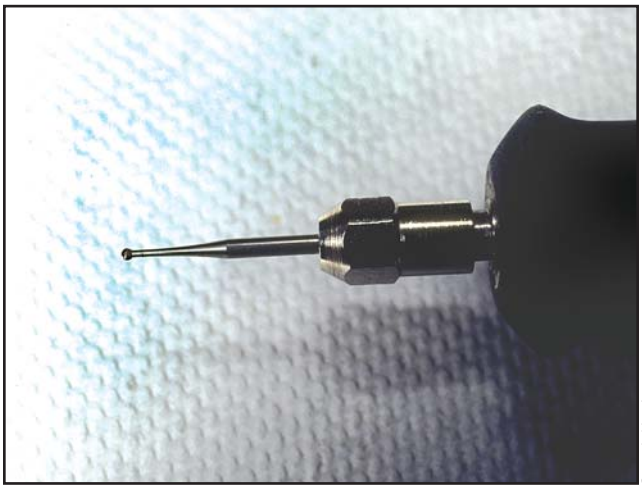
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Fig 14.6b | An electric motor drill has a small shank shim replacing the larger variety. This allows the use of a small dental burr to hone out the damaged rhinotheca and bone seen in 14.6a.

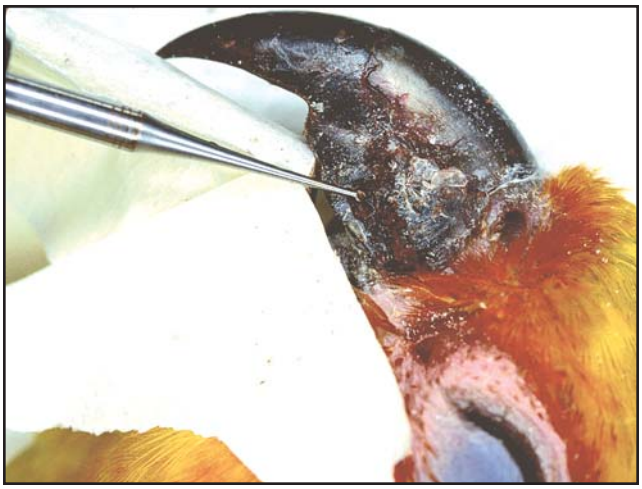
Greg J. Harrison

Fig 14.6c | A dental burr is used to hone out the damaged tissue around the edges of the depressed slab of rhinotheca and bone.

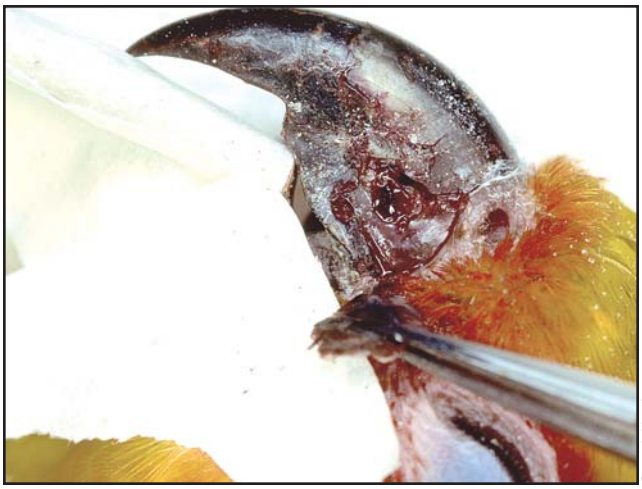
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Fig 14.6d | Microsurgical forceps grasp the slab and bone and remove it from the site to avoid a sequestration.


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Fig 14.6e | The rhinal cavity mucosa was not penetrated so an absorbable layer of calcium hydroxide[a] is applied as a bed for the regrowth of the bone, periosteum and rhinotheca. This layer is dried. The warm air from the electric motor can hasten this step.


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Fig 14.6f | A thin layer of cyanoacrylic solvent[b] is layered over the calcium layer.

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Fig 14.6g | Powdered dental acrylic resin[c] is sprinkled over the solvent layer, and the powder liquifies.

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Fig 14.6h | The process has been repeated on both sides of the conure's damaged maxilla. In a couple of weeks the acrylic will dehisce and the underlying tissue continue to heal with no further attention. No antibiotics or antifungals were used pre- or post-operatively.

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Fig 14.6i | The kit used to perform the procedure, 14.6 a-h.

Greg J. Harrison

Fig 14.7 | A fractured mandibular symphysis has been repaired using a pair of S-shaped hooks like those used in Fig. 14.5d,e. One is placed on each side of the fracture site and the impaction bands applied. A layer of acrylic helps hold the pins and protects the fracture site.

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Fig 14.8 | This gray-cheeked conure had a proximal traumatic maxillary amputation, involving the germinal area, and will likely not regrow. The conure is shown several months post-injury.

usually with less pathogenic consequences.[58] Diagnosis is by viral culture or histopathological demonstration of proliferated epithelial cells with intracytoplasmic inclusions or Bollinger bodies. No specific antiviral treatment exists but lesions can be topically debrided and treated with antimicrobials for secondary infections as deemed necessary. Systemic antibiotics and fluid therapy along with supplemental vitamin A may aid recovery.

Disinfection with lipid solvents (eg, quaternary ammonium compounds, sodium hypochlorite) and exclusion of potential insect vectors will help to stop further spread of the infection. In outbreak situations, euthanasia of severely affected birds may be carried out. Vaccines are also available for some strains.[59]

Avian polyomavirus (APV) has been seen to cause tubu-

lar elongation of the lower mandible in Gouldian finch (*Erythrura gouldiae*) juveniles that have survived outbreaks of the disease.[60] These birds also exhibited delayed fledging and did not grow well. Other passerine infections with this virus have been associated with outbreaks of sudden death.[61] It has been postulated but not proven that recovered passerines may in fact become persistently infected and may shed the virus intermittently. Vertical transmission of the virus through the egg has also been postulated.[61]

Parvovirus infection in ducklings reportedly caused stunted beaks with protruding tongues in survivors.[62]

Primary bacterial and fungal infections of the beak are usually associated with trauma. They may cause necrosis, inflammation, hemorrhage, hyperkeratosis and the accumulation of necrotic debris with or without malodor. Cytology, culture and sensitivity are required to diagnose these infections.

Cryptococcosis has been associated with proliferative masses causing disruption of the nares, rhamphotheca and deeper beak and sinus structures in several psittacine species.[63,65] In some cases these lesions may be mistaken for neoplasms. They are characterized by gelatinous exudates which, when stained, contain large oval budding yeasts (4 to 7 μm) surrounded by a capsule 2 to 4 times the diameter of the cell.[64,65] Gram's stain, India ink, and Wright's stain have all been used to diagnose this infection cytologically. It can also be easily cultured on Sabouraud- dextrose agar. There is usually little surrounding inflammation, restricted to mild numbers of epithelioid macrophages, multinucleated giant cells and heterophils. *Cryptococcus neoformans* var. *neoformans* has a worldwide distribution, grows poorly at temperatures over 40° C and hence rarely causes problems in birds. It is commonly found in pigeon droppings. *C. neoformans* var. *gattii* is restricted to river red gums (*Eucalyptus camaldulensis*) and forest red gums (*E. tereticornis*) and grows poorly above 37° C and is most commonly identified in avian infections.[63] Treatments such as fluconazole orally at 8 mg/kg/day for at least two months, ketoconazole at 2 mg/kg BID per os gradually increased to 25 mg/kg bid per os and surgery to debulk the proliferative masses have all been suggested.[63-65] However, recurrence of lesions weeks to months after treatment is common, with early detection and aggressive therapy most likely to yield favorable results. Cryptococcosis is a potential zoonotic infection so public health issues need to be considered before treatment is instituted.

Fungal infections causing beak necrosis in Gouldian finches (*Erythrura gouldiae*) have also been described.[60] Affected birds had rhamphothecas that were characterized by a flaky white or yellow appearance. Fungal hyphae were detected in the beak matrix but no species identification was presented.

Bob Doneley

Fig 14.9 | Knemidocoptes mites and gross over-growth of the beak.

Knemidocoptes spp. mites can cause proliferation and inflammation of the psittacine beak (Fig 14.9) and are commonly seen in budgerigars, particularly young or immunosuppressed birds. Close inspection reveals the characteristic honeycomb patterning resulting from the mites tunneling into the skin. In chronic lesions, the germinal layer of the rhinothecal and gnathothecal epithelium can be so disrupted that permanent beak deformities result. Diagnosis is by way of skin scrapings. Treatment is simple with ivermectin/moxidectin, topical ectoparaciticides or even paraffin oil over the lesion that suffocates the mites being successful. Spiruroid (*Oxyspirura* spp.) infections in cranes have also been linked to beak deformities, as has trichomoniasis in cockatiels.[44]

Mycotoxins from *Fusarium* spp. in moldy food have resulted in beak deformities in poultry.[32]

Neoplasia

A number of neoplasms involving the beak have been described. Fibrosarcomas are considered the most common neoplasms of the beak, whilst squamous cell carcinomas and malignant melanomas are also seen. They cause distortion of the beak and surrounding tissue. Cytology of fine needle aspirates or histopathology on biopsy specimens provide a diagnosis, give information as to the likelihood of success with surgical debulking or chemotherapy and provide a prognosis for the patient.[57,66]

DISEASES OF THE OROPHARYNX AND ITS STRUCTURES

Diseases of the oropharynx are characterized by anorexia, dysphagia, halitosis, gaping, rubbing of the

beak or more generalized signs of ill thrift such as lethargy and disheveled plumage. Direct visual examination of the oral cavity under illumination will reveal most lesions, especially if magnification is used. This can be done with the patient awake or under general anesthesia. Further magnification in difficult to examine places can be achieved via endoscopy. Offending lesions can be swabbed or biopsied and the material obtained can be stained on slides, cultured or sent for cytological or histological examination.

Infectious Causes

Various viral infections have been found to infect the avian oropharynx. As mentioned previously, poxvirus can cause proliferative caseous lesions in the mouth and esophagus. Pigeon herpesvirus (PHV-1) can cause mucosal ulceration and diphtheritic membrane formation in the oropharynx, cere or beak commissure as part of the overall infection.[44,67] It affects young birds and the immunosuppressed most severely and should be suspected in flocks that suffer repeated bouts of trichomoniasis that are difficult to control. Spread is via fecal and pharyngeal secretions, and latent carriers are important reservoirs of infection.[67] Diagnosis is presumptively based on the presence of basophilic and eosinophilic intranuclear inclusion bodies seen on histology or cytology of affected tissue, particularly epithelial cells. Virus isolation and neutralizing antibody techniques are also available.

Abscesses and micro abscesses, plaques and granulomas are consistent with a number of diseases including viral, bacterial, yeast and parasitic infections, hypovitaminosis A and even chemical burns. Bacterial infections in the mouth can be caused by a variety of bacteria. Some of the more frequently isolated pathogens include *Staphylococcus* spp., *E. coli*, *Klebsiella* spp., *Pseudomonas aeruginosa* and other gram-negative bacteria.[37,44,68] The lesions may be localized or cause a generalized stomatitis and are usually secondary to oropharyngeal trauma, other infectious diseases or other causes of immunosuppression. Treatment should include systemic antibiotics based on culture and sensitivity results, local debridement, supportive care, identification and, where possible, correction of underlying immunosuppressive factors.

Mycobacterial granulomas may sometimes be seen in the mouth, though they are more commonly associated with lesions in the intestinal tract and liver and other intracoelomic organs.[69] Fine needle aspirates of lesions and acid-fast staining may reveal the presence of the mycobacterial organisms within macrophages. Where possible, cultures to speciate the type of mycobacteria should be carried out, although recently PCR testing that gives more rapid results has become available.[70] Hematology is characterized by a very high leukocytosis, often with a monocytosis. *Mycobacterium genavense* has recently been recognized as the causative agent in many avian infections that were previously attributed to *M. avium* subsp *avium*.[70,71] *M. tuberculosis* has only infrequently been responsible for disease in birds, even though it is the primary cause of tuberculosis in people.[72] Mycobacteriosis is a chronic debilitating disease with the potential for zoonotic spread, particularly in the immunosuppressed person.[72] Therefore the decision to treat or to euthanize is an important consideration. Multiple drug therapy is essential if treatment is to be attempted due to the high level of resistance to any single antimicrobial. Several treatment modalities based on human trials have been suggested.[73,74] Currently the combination of clarithromycin, ethambutol and rifabutin is the treatment of choice in humans and has been used with enrofloxacin in psittacines with success (see Chapter 28, Implication of Mycobacteria in Clinical Disorders).[74]

Candidiasis is a very common cause of stomatitis in birds, particularly in young, immunosuppressed birds, those on antibiotics and in lorikeets because of the high sugar content of some nectar mixes. The causative agent is usually *Candida albicans*, although other species may be involved. It is opportunistic and can be a primary or secondary pathogen. It causes white oral plaques with a caseous exudate. It is easily cultured and, when smears of lesions are made, the characteristic budding spores may be seen. Gram's stain, Diff-Quik and new methylene blue stains may help visualization. Histopathology is required to confirm that the yeast is causing the infection, however the presence of large numbers of budding yeasts or the presence of hyphal forms is suggestive. Treatment may be topical and/or systemic. Mild infections may respond to oral nystatin at 300,000 IU/Kg orally twice daily and/or topical chlorhexidine or oral miconazole formulations. More severe infections may require systemic antifungal therapy such as ketoconazole (10 to 30 mg/kg orally twice daily), fluconazole (20 mg/kg orally every 48 hours),[75] flucytosine at 250 mg/kg orally twice daily for 14 to 17 days or itraconazole (10 mg/kg orally twice daily for 21 days).[76]

Parasites are another cause of oropharyngeal pathology. *Capillaria* spp. are the most common nematode in the upper gastrointestinal tract. They may cause oral inflammatory masses, diphtheritic oral lesions or hemorrhagic inflammation of the commisure of the beak.[75] They are more commonly found in the small intestine.[77] They parasitize most species of birds including psittacines, passerines, columbiforms, gallinaceous birds and raptors.[77,78] Affected birds exhibit head flicking, dysphagia, weight loss and diarrhea. The adult parasites are very thin and can be difficult to see, but may be found in smears of lesions, as may their characteristic bi-opercu-

lated ova. Ova may also be detected upon fecal floatation, but they are intermittent shedders of ova and produce less than most ascarids. Their life cycle can be direct or indirect using earthworms as intermediate hosts.[77] They can be quite resistant to anthelmintics so high doses may need to be instituted. Examples include benzimidazoles (fenbendazole 100 mg/kg once or 25 mg/kg daily for 5 days, oxfendazole 10 mg/kg,) levamisole (40 mg/kg, beware of narrow safety margin); moxidectin 200 μg/kg (used up to 800 μg/kg by this author).[77,78] Ivermectin at standard doses (200 μg/kg) has been ineffective.[78] Benzimidazole or levamisole treatments should be repeated in 14 days. Environmental hygiene to prevent reinfection and removing potential intermediate hosts are all important control measures.

Spiruroids have been diagnosed in raptors, corvids and other species, and may cause raised granulomatous reactions in the mouth and crop. The worms, or their thick-walled embryonated eggs, may be found in oral, crop or fecal samples. Treatments include oral dosing with moxidectin[77] and/or manual removal of adults. *Contracaecum* spp. have been associated with severe oral infections in young piscivorous birds, particularly pelicans.[44] In birds of prey, *Synhimanthus falconis* has been reported in the oropharynx[44] and *Serratospiculum amaculatum*, a parasite of the air sacs, can cause diphtheritic lesions of the oropharynx, which need to be differentiated from those caused by trichomoniasis.[77,79,80] Their eggs can be found in oral mucus or in feces.

Trichomoniasis is commonly found in pigeons, budgerigars and raptors and is occasionally seen in other species such as cockatiels, Amazon parrots, conures, canaries and zebra finches.[44,79] The causative organism, usually *Trichomonas gallinae*, can exist as different strains with different pathogenicities. In pigeons and raptors, white or yellow caseated plaques may be seen in the oral cavity. These usually extend to the crop and esophagus and may go as far as the proventriculus. These plaques may need to be differentiated from other diseases such as candidiasis and poxvirus infection. Budgies usually show no oral lesions. Affected birds usually exhibit regurgitation, dysphagia, weight loss, listlessness, palpable mucous in the oropharynx and crop and, in severe cases, vomiting blood and death. In pigeons the disease may be generalized, infecting the liver, umbilicus and cloaca, especially in squabs. Diagnosis is via wet mount examination of oral lesions or crop fluid, revealing the motile flagellated organism under high power magnification. Warming samples increases protozoan activity. The life cycle is by direct oral contact between birds, and spread through common drinking water is also important. Raptors are thought to acquire infection through ingestion of infected pigeons. However, freezing carcasses has proven not to work (S. Hudelson, personal communication, 2004). Carrier states exist and are thought to be responsible for reinfecting flock mates. Such birds should be culled. Treatments suggested include ronidazole (6 to 10 mg/kg once daily for 7 to 14 days), dimetridazole (100 to 400 mg/L drinking water), metronidazole (20 to 50 mg/kg twice daily) and carnidazole (20 to 30 mg/kg once).[75,77,78,81] A dose of 50 mg of carnidazole has been reported to be most effective (S. Hudelson, personal communication, 2004). Note that dimetridazole has a low safety margin and should be avoided in hot weather, during breeding or when racing pigeons. Diphtheritic plaques may need to be removed by debridement. Antibiotics for secondary infections may also be required.[68] Regular monitoring of flocks for infection is recommended in pigeons and budgerigars.

Nutritional Causes

Hypovitaminosis A can lead to squamous metaplasia of the oropharyngeal epithelium, particularly glandular epithelium, leading to plaque and granuloma formation.[44,75,82] In psittacines, this typically involves the submandibular or lingual salivary glands. Sometimes affected birds exhibit a subcutaneous swelling caudal to the mandible. The choanal papillae are often shortened and stunted. If severely damaged, the choanal papillae fail to regenerate, so caution is warranted when using this sign for diagnosis. Affected birds typically have a history of being fed a predominately seed-based diet. Dietary correction via parenteral vitamin A supplementation or use of reputable formulated diets is needed. Some granulomas may be excised surgically. Secondary bacterial infections may be found and should be treated as required. In gallinaceous birds, lesions are confined to the mucous glands of the pharynx and their ducts. Keratinization of the glandular epithelium causes blockage of duct openings, hence secretions and necrotic debris accumulate. These appear as small white hyperkeratotic lesions (see Chapter 4, Nutritional Considerations).

Traumatic Causes

Traumatic injuries can occur in the oropharynx due to fighting or accidental trauma. Injuries of the psittacine tongue are common due to its frequent use as a probing and sensing organ. Tongues are very vascular, and in psittacines well muscled, so control of bleeding is a first priority. This may involve the use of electrocautery or suturing. The birds may easily remove sutures. Some authors have found the need to wire the beak closed to prevent suture removal.[75] In this case a pharyngostomy tube may need to be placed until the wound heals. Caustic injuries can be caused by the ingestion of certain

chemicals, for example silver nitrate cautery sticks, access to excessively hot foods or ingestion of trichotecenes, especially T2 toxin.[44] These often cause anorexia and dysphagia due to the pain experienced. Analgesia may be of added benefit in these cases, as well as supportive care (pharyngostomy feeding, fluids, antibiotics) until healing is complete. Foreign bodies including wire, wood and plastic have also been known to cause penetrating wounds.

Neoplastic Causes

Neoplastic diseases of the oral cavity have been documented and include epithelial and mesenchymal tumors.[57] The most common problem seen in new world psittacines is oral papillomatosis. Lesions range from mild mucosal roughening to overt verrucous masses. They can also be found in the crop, esophagus, proventriculus, and cloaca and have been associated with bile duct carcinomas. Severe lesions can ulcerate, hemorrhage or cause gastrointestinal or distal reproductive tract obstruction. Their exact cause is unknown. Although histologically the lesions appear similar to those of mammalian papillomaviruses, there is no immunohistochemical or DNA evidence to support the presence of an avian papilloma virus. Instead, herpesvirus is consistently being detected in these papillomatous lesions.[83] True parrot papillomavirus has been reported in only one African grey parrot.[84] Squamous cell carcinomas, fibrosarcomas and lymphosarcomas have also been reported.[85] They can be quite painful and cause inappetence. Diagnosis is based on biopsy and histological examination.

DISEASES OF THE ESOPHAGUS AND CROP

Many of the diseases found in the oropharynx are, not surprisingly, also found in the esophagus and crop. The esophagus can be injured as a result of tube-feeding (Figs 14.10a-c).

Infections

Viral infections such as poxvirus and herpesvirus have been reported. Proventricular dilatation disease (PDD), of suspected viral origin, may also affect the crop but will be more fully discussed later. Bacterial infections, both primary and secondary, are commonly seen with crop diseases. In mild infections, there may be bacterial growth and mucosal colonization with little inflammatory response. Severe infections, however, are characterized by hemorrhage, necrosis and sometimes fibrinopurulent exudates of the mucosal surface.[57]

Typically, crop motility is impaired with delayed crop emptying and regurgitation. Birds can quickly become

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Fig 14.10a | Tubefeeding a sick bird is a frequent event in an avian veterinary facility. The round ball tipped needles can make the job easier for one person.

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Fig 14.10b | The clear delicate esophagus can be palpated for the tube presence or visualized by wetting the right ventral-lateral cervical area. The ball is easily seen through the transparent esophagus and skin of the neck in small thin birds.

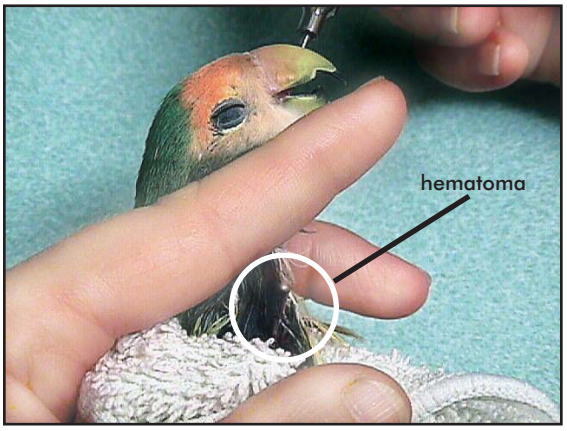

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Fig 14.10c | A similar feeding tube in a lovebird with a hemotoma in the crop area from trauma resulting from bruising that can occur from improper restraint and flailing when a bird is being tubed. Although this is very uncommon, it is even less likely to occur if a speculum and a soft catheter are used.

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Fig 14.11a | A crop burn, usually in a handfeeding baby that is fed scalding food heated by a microwave oven. The area is soft and friable and is best allowed to form a scab prior to any surgery.

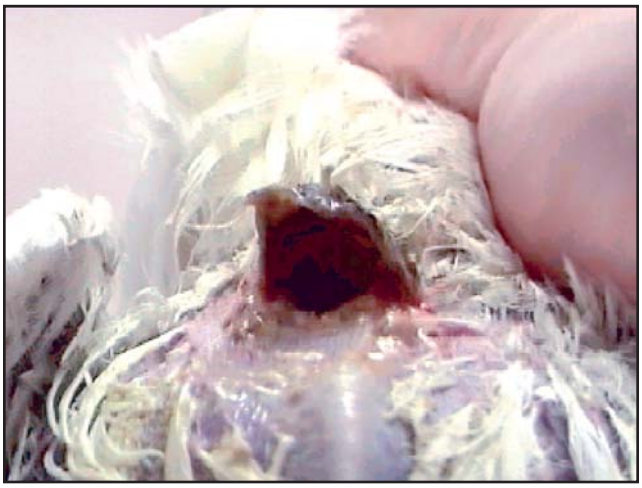

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Fig 14.11b | Several days after becoming a dark, hard scab the burnt area will open allowing food to fall from the crop. Topical acrylic glues can expand the time the scab is retained and often can be kept in place long enough for the damage to completely heal, avoiding surgery completely.

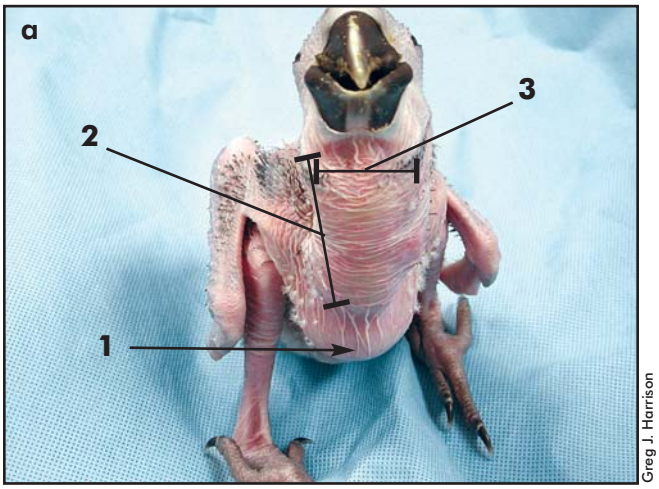
listless, toxemic, dehydrated and, if untreated, die. Yeast infections may also be involved, and the organisms are typical of *Candida* spp. infections. *Candida* spp. has also been described as a primary crop pathogen in some species, namely lovebirds (*Agapornis* spp.) and cockatiels.[86] As well as the clinical signs listed above, a palpably thickened crop wall may be found. Causative organisms can be diagnosed via examination of wet and stained smears obtained via crop washes and culture and sensitivity. Treatment involves the use of oral topical and/or systemic antifungals, crop washes, antiemetics and fluid therapy. Predisposing management factors need to be addressed, particularly in hand-reared birds.

A large number of endoparasites are known to infect the crop. The most familiar parasitic disease of the crop is trichomoniasis, which was discussed previously. A host of nematodes and trematodes can also be found in the crop and esophagus. As well as *Capillaria* spp. already mentioned, *Echinura uncinata*, *Gongylonema ingluvicola* (quail and gallinaceous birds) and *Dispharynx nasuata* have all been found to invade the crop esophageal mucosa.[44,77]

Noninfectious Diseases

Primary non-infectious diseases of this region include crop burns, foreign body penetration, lacerations and impactions, hypovitaminosis A and ingluvioliths. Excessively hot feeding formulas may lead to full thickness burns of the crop and skin in hand-reared chicks and occasionally in adults.[44,85] The burns usually occur in the anteroventral region of the crop. Initially the area may appear just dry, but subsequently reddening and edema may occur, followed by blistering and often necrosis (Figs 14.11a,b). This process may take several days. The dehisced wound, which is externally visible, allows food to fall out of the crop as the chick is fed. Treatment involves addressing any dehydration and infection problems. It may be best to manage this condition medically for the first 3 to 5 days to allow the burn to scab over and fully fistulate before surgery is attempted. Premature surgical closure usually results in wound dehiscence due to continued necrosis of the surrounding skin that may not have been noticeable at the time of initial surgery. Surgery involves anesthesia and debridement of the affected area, removing all dead and discolored tissue, and closure of the deficit in at least two layers, ensuring that the crop and skin are closed separately. Beware of reduced crop capacity immediately post-surgery, until healing and crop expansion can occur (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).[87] Other causes of crop trauma such as forceful use of feeding/medicating tubes, bite wounds or foreign bodies can be similarly treated. Caution — food traumatically placed subcutaneously or within the neck structures must be flushed and debrided immediately in the event feeding has caused a rent — let heal by secondary intention healing (Figs 14.12a-f).

Ingluvial foreign bodies include food items, grasses, wood, metal and plastic items usually accidentally ingested by birds. In hand-reared chicks, plastic feeding tubes can come loose and be accidentally swallowed during the vigorous head pumping which birds may do during feeding. Diagnosing these problems is via history, crop/esophageal palpation, radiography (both plain and contrast) or endoscopy. The offending items can be removed manually, endoscopically or surgically via an ingluviotomy. Impactions of the crop frequently occur as a result of a sudden change of food. In ducks and poultry



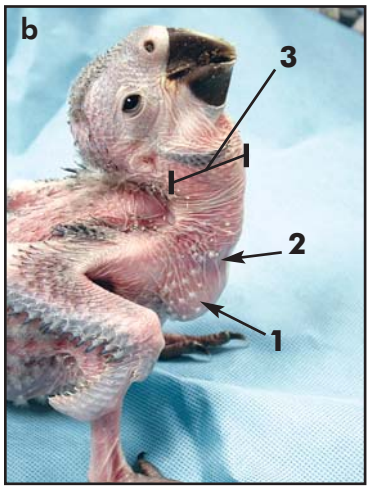


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Fig 14.12a,b | A baby macaw an hour after having hand-feeding formula traumatically introduced into the cervical neck tissues. This injury occurs from using a syringe for feeding. Macaws "pump" hard and if the head is not controlled the syringe tip can penetrate the pharyngeal tissues resulting in food being deposited into the cervical tissue. Edema and some hemorrhage also adds to the swelling. The food must be flushed out within hours, or septicemia may be rapid and overwhelming. 1. A normal crop with some food present. 2. Vertical swelling from food deposited into tissues. 3. Horizontal swelling of tissues.

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Fig 14.12c | An incision over the food swelling — avoiding the esophagus, crop and vascular structures of the cervical region.

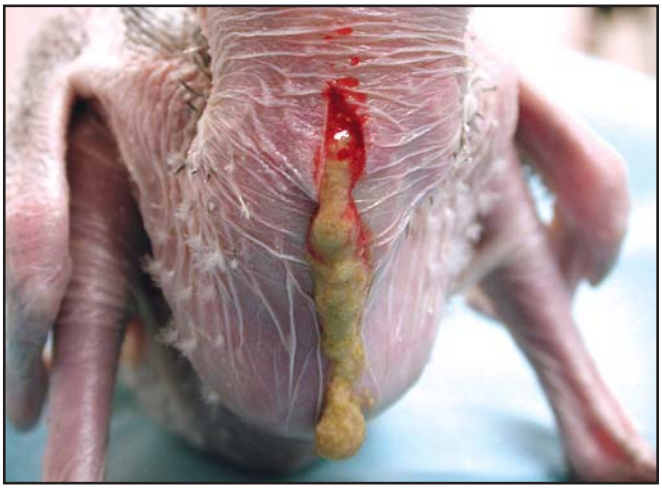

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Fig 14.12d | The incision has reached the pocketed food and it spills out.

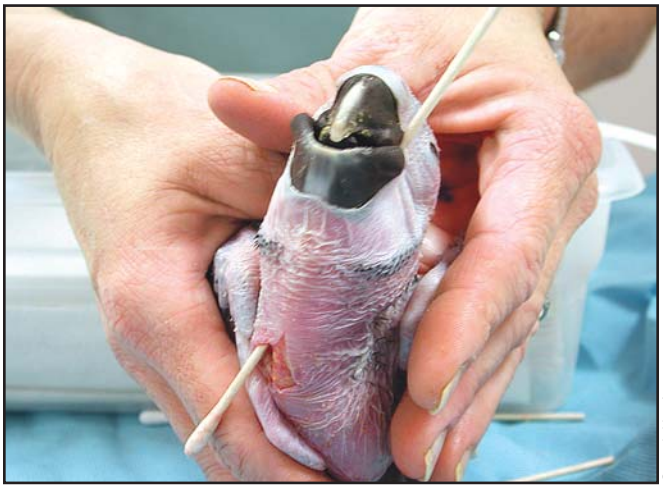

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Fig 14.12e | A cotton-tipped wood applicator enters the oral penetration wound and exits the neck incision. The cotton tip is grasped with a forcep and the forcep is pulled up and out through the mouth. A cut rubber band is grasped by the forcep and pulled out of the incision and tied in place to act as a seton to allow flushing for 2 days.

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Fig 14.12f | Rubber band seton tied in place. Flushing of the wound QID with Normosol®, and a tissue disinfectant is performed. Antibiotics and antifungals are administered in a hand feeding formula that is fed via a silicone tube to assure the food gets into the crop.

it has been associated with sudden access to lush grasses and sprouted grains.[44] In caged birds, *ad libitum* supply of grit has been associated with crop impactions and, on occasions, this has resulted from the overzealous feeding of grit to chicks.[88] In raptors it is associated with the sudden availability of roughage in a previously low-roughage diet. Diagnosis and treatment are as described for foreign bodies.

Ingluvioliths are various mineral concretions that occasionally develop in the crops of some birds, particularly budgerigars. Calculi consisting of urates surrounding seed husks, potassium phosphate, oxalate and cystine have been described. The exact cause of these is not known, but it is speculated that birds that have experienced periods of starvation may have been forced to eat seed husks and urates.[44] In some instances these calculi become large enough that they need to be removed. This can be achieved via endoscopy or ingluviotomy.

As well as the neoplasms mentioned in the oropharynx, the crop and esophagus could suffer from tumors of smooth muscle origin such as leiomyomas and leiomyosarcomas. Although asymptomatic when small, they can become very large, necrotic and hemorrhagic. They are characterized microscopically by interlacing bundles of fusiform cells with moderate amounts of cytoplasm.[57] Carcinomas of the submucosal glands also occur. They are often large, sometimes necrotic and hemorrhagic, and involve much of the esophageal or crop wall with invasion into surrounding tissue.[57]

Crop Stasis

Crop stasis or "sour crop" is a clinical sign of disease, and not a disease in itself. Clinical signs include regurgitation, delayed crop emptying, a sour odor, inappetence, dehydration, anorexia and listlessness.[87] "Sour crop" is usually complicated by bacterial and or fungal infection that may be primary, but is more often secondary. Crop stasis is most often seen in hand-reared chicks and results from poor management. Food fed at the wrong temperature or consistency, not allowing the crop to empty between feedings, poor hygiene, incorrect incubation temperatures and humidity and concurrent disease are all examples of possible causes of crop stasis in chicks. In adults, the condition can result from various crop infections, systemic or metabolic disease, heavy metal toxicity, foreign body ingestion or even PDD. Normal psittacine crop floras include few gram-positive bacteria and scant non-budding yeasts. Treatment of this condition involves identifying and treating the underlying disease, as well as crop flushes with mild antiseptic solutions, antimicrobial therapy as appropriate and supportive fluid therapy. See Chapter 7 Emergency and Critical Care.

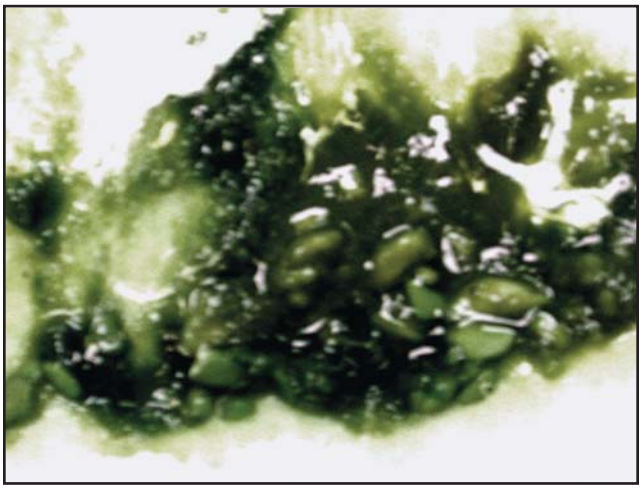

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Fig 14.13 | The passing of whole seeds is characteristic of gastrointestinal disease. Many causes such as lead, parasites, PDD and pancreatitis are possible.

DISEASES OF THE PROVENTRICULUS AND VENTRICULUS

Diseases of the proventriculus and ventriculus can have varying clinical signs ranging from regurgitation, weight loss, appetite changes (either anorexia or polyphagia), undigested seed in the feces (Fig 14.13) and lethargy. Most diseases of these organs produce similar clinical signs that make their identification more challenging.

Infectious

Perhaps the most common gastrointestinal disease is proventricular dilatation disease (PDD). The suspected cause is a virus. It has been diagnosed in over 50 species of psittacines, but also in several other avian species including Canada geese, canaries, weavers, toucans, spoonbills and honeycreepers.[89] It is characterized by a lymphoplasmacytic infiltration of peripheral and central nerve tissue. It commonly affects the myenteric plexuses supplying the gastrointestinal tract, resulting in atrophy of the smooth muscles of the crop, proventriculus, ventriculus or small intestine. This causes delayed gastrointestinal motility and organ dilatation. It can also affect the Purkinje cells of the heart, the adrenal medulla, the brain and the spinal cord. The lesions can be very segmental which may explain the variation in clinical signs seen.[90]

Gastrointestinal clinical signs include progressive weight loss, regurgitation, crop impaction, passage of undigested food and eventually death, usually within 12 months. An 80 to 120 nm enveloped virus has occasionally been isolated from infected birds and has been used experimentally to induce infection in some birds.[89] A virus is certainly suspected but not proven. The virus itself may not be the cause of the disease; however the inflammatory response may be. However, its exact identity

is unknown at the time of writing. Diagnosis is based on finding lymphoplasmacytic infiltrates in ganglia and associated nerves of the myenteric plexus of the gastrointestinal tract.[90,91] However, given the segmental nature of the disease, it is difficult to know which area of myenteric plexus to biopsy. Thus, a positive biopsy is diagnostic but a negative biopsy does not rule out the disease.[90] The crop is considered the safest area from which biopsies can be taken. Biopsy sections should contain blood vessels, as these are most likely to contain nerve tissue. The proventriculus has a thin wall and acid secreting glands which make it a risky choice for biopsy. Similarly, ventricular biopsies are not recommended, as there is real risk of damaging the myenteric plexus.

Large amounts of the suspect virus are shed by infected birds and transmission is proposed to occur via the fecal-oral route.[91] The virus is fragile in the environment and, hence, hygiene and management are vital in preventing spread of infection. The virus's environmental fragility may explain why epornitics occur more commonly in indoor aviaries rather than outdoor collections. Although many birds eventually die from this disease, some chronically infected parrots have been detected which have lived for years, intermittently shedding the virus. These may be reservoirs for reinfection in aviary situations. Although there is no cure for the disease, celecoxib, a Cox-2 NSAID, has been used to improve clinical signs by decreasing the inflammatory reaction around affected nerves.[92] The dose given was 10 mg/kg orally every 24 hours for 6 to 24 weeks. Improvement in clinical signs occurred in 7 to 14 days. Treatment was ceased once birds resumed normal body weight, condition and diet. The longest survivor reported was a blue and gold macaw (*Ara ararauna*) that finished therapy two years previously and remained in normal physical condition, eating a normal diet and had no radiographic signs of PDD and was negative on biopsy. However, no comment was made as to whether virus particles continued to be shed in its stools.

Macrorhabdus, previously known as avian gastric yeast (AGY) or megabacteria, have been reported to infect a large range of pet and wild birds (see Chapter 30, Implications of Macrorhabdus in Clinical Disorders).

Bacterial infections in the proventriculus and ventriculus can be primary but are usually secondary to other immunosuppressive or disease states. The organisms most often associated with disease are gram negative and include *E. coli*, *Klebsiella* spp., *Salmonella* spp. and *Enterobacter* spp.[85] These often affect the intestine, thus giving clinical signs including diarrhea, maldigestion/malabsorption, anorexia and weight loss. Diagnosis is based on culture, with fecal cultures often taken. Fecal Gram's stain may also show gram-negative overgrowth. *Candida* spp. infiltrating the proventricular and ventricular wall have also been documented, particularly in finches.[60,76] Treatments as outlined earlier, optimal nutrition, hygiene and correcting predisposing stressors are all important management tools.

A number of nematodes have been diagnosed in the avian proventriculus. *Echinura uncinata*, *Gongylonema* spp., *Cyrnea* spp., *Tetrameres* spp. and *Dyspharynx nasuata* have all been found. Ventricular parasites include *Amidostomum* spp., *Cheilospirura* spp., *Epomidiostomum* spp. and *Acuaria* spp.[44,77] Of these, *Acuaria* spp. appear to be the most commonly encountered. They are commonly found to infect finches, but galliforms are also susceptible. These "gizzard worms" are fine and hair-like and burrow just under the koilin lining of the ventriculus, impairing gizzard function and digestion of food. Affected birds usually exhibit ill thrift, may have undigested seed in the droppings and die. Secondary bacterial infections further complicate the infection. The life cycle is indirect, so removal of insects from the environment is important. Anthelmintics such as ivermectin, moxidectin, benzimidazoles and levamisole have all been used with varying degrees of success.[77]

Cryptosporidiosis is a protozoal disease usually seen to infect the intestine of immunosuppressed animals, including birds. In finches, however, it has a predilection for the proventriculus, where it causes necrosis and hyperplasia of glandular epithelial cells.[113] Finch isolates are different genetically from other species and may represent a unique species of *Cryptosporidium*.[114] Affected birds show decreasing body weight and yellowish droppings which may contain undigested seed. Azithromycin, roxithromycin, toltrazuril and paromomycin have met with some success as treatments.[113] Underlying immunosuppressive diseases or environmental stressors need to be identified and corrected.

Non-infectious

Proventricular or ventricular foreign bodies are more commonly encountered in ratites, galliforms and waterfowl, but are also seen in psittacines and other species. Psittacine chicks which ingest indigestible fabric fibers or bedding material such as ground corncob, kitty litter, crushed nut shells, shredded paper, styrofoam, grit, plastic, rubber or wood shavings may develop proventricular/ventricular impactions.[44,75] In older parrots, these same items plus other cage or household items may be ingested. In flightless birds, and in particular ostriches, exposure of birds to a new substrate may predispose

them to proventricular/ventricular impaction. Other illnesses may lead to a depressed appetite or pica.[115] Affected birds classically have poor appetites, pass scant feces, exhibit regurgitation especially if force-fed, and are depressed and lethargic. Diagnosis is via radiography, endoscopy, palpation (in larger species) or exploratory laparotomy. Where nails or other ferric compounds are suspected, this author has also seen metal detectors successfully used. Fiber will only show up on gastroscopy (Fig 14.15).

Treatment depends on the severity of the impaction, or the nature of the foreign body ingested. If the bird is bright, the impaction is not complete and the offending item is capable of being passed by the bird, then medical treatment may be adequate. This may include supportive fluid therapy and antibiotics, the administration of psyllium (beware in cockatiels) or paraffin liquid and force-feeding with easily assimilated high-energy soft foods. Metoclopramide may help stimulate small intestinal motility and thus assist in ventricular/proventricular emptying. Prevention of access to the offending items is also necessary. Attempts should be made to identify underlying disease states that have a bearing on the final outcome.

Some items may be able to be removed endoscopically. This can be done through the mouth or via an ingluviotomy. In the ostrich, a technique of proventricular flushing is described.[116] With the bird held firmly above the tarsus, it is turned upside down, its glottis closed and a hose connected to a steady stream of water is passed from the mouth to the proventriculus. Massaging the proventriculus helps to loosen the impaction. The loosened material often passes out the mouth with the hose *in situ*. This is repeated until clean water passes out the mouth. Needless to say, this procedure requires considerable manpower but is useful where general anesthesia is not an option and medical therapy has failed. In many cases, however, surgery is indicated to relieve the impaction or remove the foreign body. These techniques have been described elsewhere.[115,117]

Heavy metal toxicities can also lead to gastrointestinal, renal and CNS signs as part of their pathophysiology. Psittacines in particular fall victim to inadvertent acute lead and zinc intoxication due to their curious nature and penchant for chewing any object they may find. Items varying from galvanized cage wire to paint, curtain weights, stained glass windows, jewelry, coins, wine bottle foil, toys and mirror backing are possible sources for these heavy metals.[118] Waterfowl ingest lead shot whilst feeding, mistaking it for gravel.[119] Falcons ingest lead by eating prey that has been shot. The ingested heavy metal pieces are acted on by the acidic content of the proventriculus and macerated by the ventriculus leading to rapid absorption. The mucosal linings become very irritated, and in severe cases, the ventricular koilin may be damaged. Pancreatic damage may also be a result of zinc toxicosis. Affected birds may show variable clinical signs which include inappetence, decreased fecal volume, regurgitation and vomiting, ileus, green diarrhea, polyuria, polydypsia, CNS signs (particularly with lead poisoning) and feather picking.[118,120] Lead poisoning in waterfowl causes weight loss, limb and neck weakness, and bright green feces.[119] A tentative diagnosis is based on a suggestive history, clinical signs and the presence of radiodense particles within the gastrointestinal tract on survey radiographs. However, the client is often unaware of exposure to heavy metals, the clinical signs are non-specific and the heavy metal is not visible on radiographs. Definitive diagnosis is based on the presence of elevated blood lead or zinc levels.[90,118, 121,122] Elevated levels of amylase, CPK and uric acid may be found with zinc intoxication.[120] The acute cases respond well to chelation therapy with edetate calcium disodium (EDTA) at 30 to 50 mg/kg once to four times daily by intramuscular or intravenous injection, depending on the severity of signs and amount of heavy metal ingested.[90,120-122] Other chelating agents such as penicillamine given orally at 55 mg/kg twice daily can be used in conjunction with CaEDTA.[118] Succimer is the preferred oral lead chelator at 25 to 35 mg/kg orally twice daily.[81,118] Concurrent parenteral fluid therapy is essential for rehydration and assisting excretion of the metals. Where gastrointestinal function permits, gavaging of high energy/electrolyte fluids and lubricants such as mineral oil, peanut butter, psyllium, magnesium sulphate or sodium sulphate have all been suggested. These products have been found to be ineffective in waterfowl.[118] Antibiotics for secondary bacterial infections can be given and antiemetics and intestinal prokinetic agents (eg, metoclopramide) may also be helpful. If possible, the offending particles should be removed via endoscopy or ingluviotomy/gastroscopy. Particles that have a ferrous or iron base can also be removed by inserting a feeding catheter equipped with powerful neodymium-ferro-borium alloy magnets (Fig 14.14).[44] Pure lead, zinc, or many of their alloys cannot be removed with a magnet. It should be remembered that lead is not normally found in animals, as it is not involved in any normal biochemical pathway.[118,122] It also accumulates in the body over time. Zinc on the other hand is an essential trace metal and so needed in low levels. It is not stored in the body over time but is excreted.[118,121] Chronic zinc toxicities are thus due to continued or repeated exposure.

Ulceration of the proventriculus occurs occasionally in pet birds but more commonly in flightless birds secondary to foreign body ingestion or disease states. No common

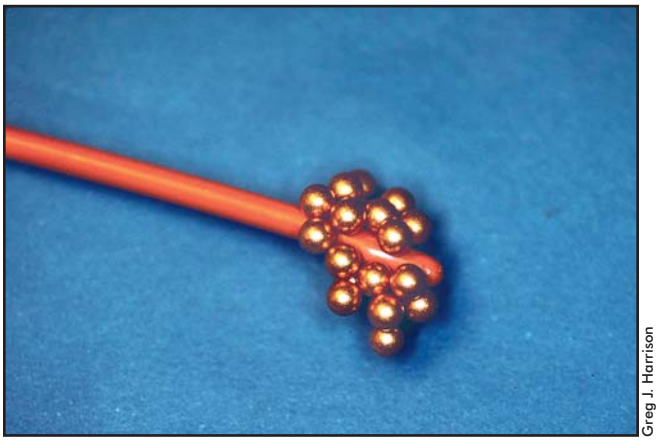

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Fig 14.14 | A set of magnets installed in the end of a rubber catheter that have attached to copper coated ferrous shot. This device can be used to remove ferrous metals, some of which can be galvanized and thus contain zinc and/or lead in toxic amounts.

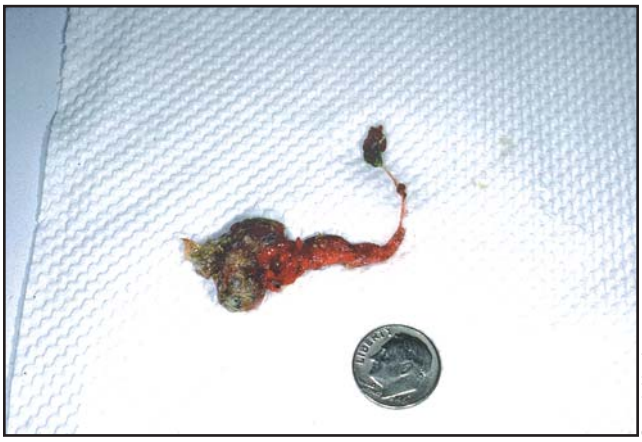

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Fig 14.15 | Nylon fiber retrieved from the proventriculus of a cockatiel on a seed diet that was allowed to pull apart a nylon carpet.

etiologic agent has been identified, but a link has been suggested between chronically stressful environments and the occurrence of proventricular ulcers.[90] Ulceration may also be secondary to zinc toxicity.[85,121] Clinical signs are non- specific but may include anorexia, regurgitation, gastrointestinal pain, lethargy and melena. Once an ulcer perforates, most birds will die from sepsis and shock within 6 to twelve hours.[90] Thus, early detection of an ulcer is imperative but can be difficult. Gastroscopy should be considered in any patient with persistent signs of gastrointestinal pain or melena. Some of the human rapid tests for melena are useful for detecting the presence of digested blood in the feces of seed eating birds.*

**Eds. Note: some debate continues on the specificity of such tests.*

There is a report of proventricular obstruction in an adult male eclectus parrot caused by a tubular diverticulum of the ventriculus.[123] This diverticulum consisted of dysplastic koilin and smooth muscle and caused complete obstruction of proventricular outflow. No inflammation, organisms or neoplastic changes were associated with the lesion. The cause was undetermined.

Neoplasms of the proventriculus and ventriculus are seen in a number of species, particularly in budgerigars and grey-cheeked (*Brotogeris pyrrhopterus*) and Amazon parrots. Proventricular carcinomas are most commonly found at the isthmus and are usually flat rather than nodular.[124] They are invasive, often extending through the muscular layers and may reach the ventricular wall and serosa.[57,125] However, they rarely metastasize.[125] Clinical signs may include anorexia, regurgitation, weight loss, maldigestion and melena. Papillomas may occur as discussed previously and smooth muscle tumors are uncommon.[57]

Surgery of the gastrointestinal tract is discussed in Chapter 35, Surgical Resolution of Soft Tissue Disorders.

DISEASES OF THE INTESTINES AND PANCREAS

Intestinal tract disorders usually manifest themselves clinically as changes in the color, bulk and nature of the feces produced. Thus, diarrhea, maldigestion, voluminous droppings and/or melena may be evident in affected birds. Anorexia, depression and weight loss often accompany these enteric signs.

Infectious

The majority of intestinal pathology is at least in part attributable to infectious agents. By far the most common cause of diarrhea in pet birds is due to bacterial infections, although these are seen less commonly in adult raptors.[57,78,90] Gram-negative bacteria are most commonly implicated. Enterobacteriacae are most frequently isolated including *E. coli*, *Salmonella* spp., *Klebsiella* sp., *Yersinia* sp., *Pseudomonas aeruginosa* and *Proteus* sp.[90] They can be both primary and secondary pathogens. The gross lesions induced in the affected intestine include redness, exudation and occasionally ulceration. Histologically, necrosis, fibrin deposition and predominately heterophilic infiltrates are noted,[57] although the bacteria may not always be present in all lesions.

Gram-positive bacteria have also been responsible for intestinal disease. *Enterococcus hirae* has caused enteritis and septicemia in 10 psittacine species.[126] *Campylobacter* spp. especially *C. jejuni* has been associated with yellowish diarrhea and enteritis in many avian species including psittacines,[85] passerines, waterfowl, galliformes[107] and ostriches.[127] Affected birds, which are often young, exhibit lethargy, anorexia, diarrhea and emaciation. Erythromycin and tetracyclines are the frontline

treatments for this bacterium.[107] Clostridial infections have also been diagnosed in many avian species and are known for their ability to produce potent toxins.[107] *Clostridium perfringens* can cause necrotic enteritis and foul-smelling feces in psittacines, ostriches,[127] and other species. *C. tertium* has been reported in a cockatoo with megacolon and chronic, foul-smelling diarrhea. This resulted in severe dilation of the colon characterized by a lymphoplasmacytic inflammatory reaction. The sporulated form of this bacterium has a "safety-pin" appearance that is quite visible under Gram's stain (see Chapter 4, Nutritional Considerations, Section II, Nutritional Disorders). Anaerobic culture yields a definitive diagnosis. Treatment involves the use of metronidazole (25 mg/kg orally twice daily)[75] or clindamycin (100 mg/kg orally once daily).[81,107]

Mycobacteriosis is typically a chronic wasting infection in birds, primarily affecting the gastrointestinal tract rather than the respiratory tract, as is the case with mammals. Most avian species are susceptible. Waterfowl, flightless birds, grey-cheeked parakeets, older Amazon and Pionus parrots, budgerigars, siskins, Gouldian finches, toucans and pigeons are particularly susceptible.[69,75] It is particularly a problem where birds are congregated. The intestine appears to be the primary site affected. The submucosa becomes infiltrated with large numbers of histiocytes that contain many acid-fast organisms. This affects the bowel's ability to digest and absorb ingesta. Other granulomatous lesions may be found in the liver and spleen, the bone marrow and the respiratory tract. In other cases, only skin lesions are noted. The course of this disease may take years.

Primary mycotic intestinal infections are rare, but secondary invasion by *Candida* spp. or *Zygomycetes* spp. are sometimes seen.[57]

Viral diseases can cause severe disease to the intestine. PDD can cause segmental damage to the intestinal smooth muscle, nerves and ganglia.

The clinical picture of paramyxoviruses (PMV) can include diarrhea and melena. The pathogenicity, types of lesions and clinical signs seen depend on the serotype and strain of the virus and the host's susceptibility. PMV-1, which causes Newcastle Disease, has caused gross hemorrhage (due to vasculitis of the intestinal wall) and necrosis of submucosal lymphoid tissue in the intestines of some birds.[57]

Adenoviruses also cause hemorrhagic enteritis in psittacines, American kestrels and turkeys, and a greenish diarrhea in pigeons and galliforms.[57,128] In affected psittacines, gross necrosis and hemorrhage are noted. Histologically, inflammation is variable, thrombosis of intestinal capillaries is evident, and the enterocytes contain large basophilic intranuclear inclusions.[57]

The intestine is the primary site of infection by various protozoal agents. Coccidia are some of the most widespread and well-known agents, consisting of several genera affecting a wide range of birds. Coccidia's pathogenicity may range from inapparent infections to severe hemorrhagic diarrhea and death. *Eimeria* is most common in pigeons and galliformes, whereas *Isospora* is primarily found in psittacines and passerines.[129] Both species have direct life cycles, with transmission occurring via ingestion of sporulated oocysts in fecal-contaminated food or water.[129] Disease is often precipitated by stress. Diagnosis is via detection of large numbers of oocysts in the fecal wet smears or floatation. Antiprotozoal treatments include toltrazuril (7 mg/kg orally every 24 hours), sulfa-based drugs or amprolium.[77,81] Cryptosporidiosis has been diagnosed in over 30 avian species and is considered an uncommon disease of the young and immunosuppressed (Figs 14.16a-c).[130] The only confirmed speciation in a non-galliformes was the detection of *Cryptosporidium meleagridis* in an Indian ring-neck parrot chick presented with diarrhea and delayed crop emptying.[131] Treatment has been covered previously. Microsporidia, primarily *Encephalitozoon hellem,* has been diagnosed in lovebirds, budgerigars, Amazon and eclectus parrots[132] and Gouldian finches.[133] Infection has been linked to concurrent disease, especially circovirus infections,[132] and other causes of immunosuppression.

Flagellated protozoa are also recognized as causes of enteritis. Giardiasis has been diagnosed in a variety of psittacines, poultry, waterfowl, finches and toucans.[77] Clinical signs vary from inapparent infections to weight loss, failure to thrive, diarrhea[75] or even feather picking in cockatiels in the USA.[134] Diagnosis is via direct fresh fecal examination for the presence of the pear-shaped trophozoites, trichrome fecal staining or ELISA testing. Treatments used successfully include ronidazole, metronidazole, dimetridazole and fenbendazole.[75,77,134] *Hexamita*/*Spironucleus* spp. occasionally cause enteric signs of variable pathogenicity in galliforms, pigeons and parrots.[77, 134] They can be recognized by their cigar shape and rapid motility and are more difficult to clear than are giardia.[134] *Cochlosoma* spp. cause lethargy and moist bulky droppings, as well as dehydration and death in young Gouldian finches being fostered under Bengalese finches. Adult birds are unaffected, although they may have bulkier stools. The Bengalese finches act as an asymptomatic carrier, as do a range of other finches, but not adult Gouldian finches.[135] The organism is characterized by its six anterior flagella and a helicoidal anterior ventral sucker; it is diagnosed on wet mounts of fresh fecal samples. Transmission is via the fecal-oral oral route, and the

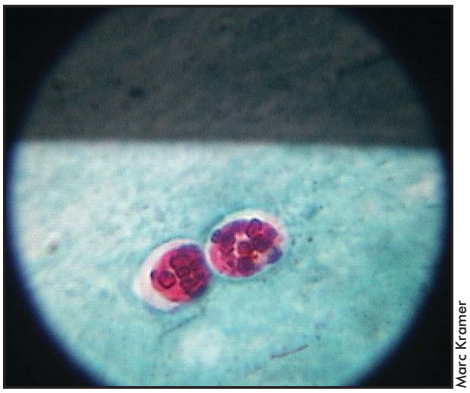

Marc Kramer

Fig 14.16a | Acid fast stain of a fresh stool specimen showing a form of cryptosporidium.

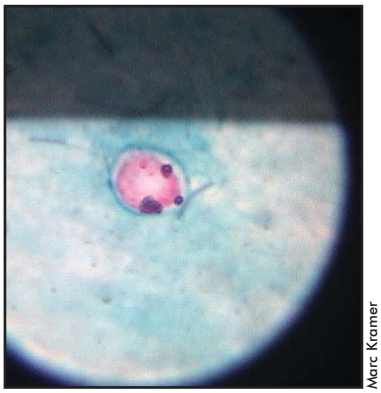

Marc Kramer

Fig 14.16b | Another form of cryptosporidium from the stool. Acid fast stain.

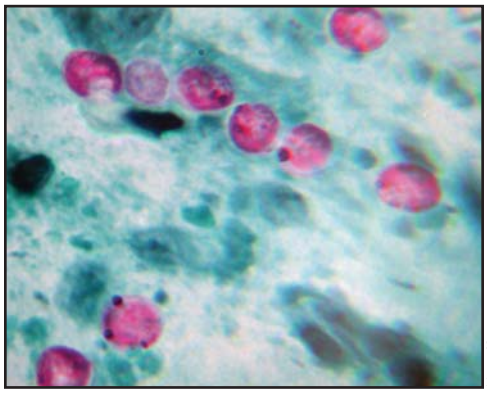

Marc Kramer

Fig 14.16c | A necropsy specimen had a proventricular scraping acid fast stained. The tissue forms of cryptosporidia are shown.

organism appears to be very sensitive to antiprotozoal drugs such as ronidazole and metronidazole.[135]

Nematode parasites are commonly diagnosed in Australian psittacines, although other psittacines have also been infected.[129,136] Pigeons, galliforms, waterfowl and toucans are just some of the other species susceptible. Nematodes are particularly a problem in wild-caught birds or those housed in planted aviaries that favor the parasite's life cycle. Ascarids and *Capillaria* spp. are most commonly diagnosed (Fig 14.17). They cause ill thrift, weight loss, diarrhea and death. Transmission is either direct by ingestion of embryonated eggs or indirect via ingestion of an intermediate host, depending on the parasite species. Diagnosis is via fecal floatation and identification of the offending eggs. Treatments include benzimidazoles, levamisole, ivermectin, moxidectin and pyrantel (4.5 to 25 mg/kg per os repeated in 14 days).[81,136] *Capillaria* spp. can be particularly difficult to eradicate, and high doses of anthelmintics may need to be given. Beware of toxicities associated with high doses. For example, Columbiformes appear susceptible to toxicosis after treatment with fenbendazole or albendazole at 50 to 100 mg/kg.[137] Secondary bacterial infections may also need to be addressed, and supportive care such as warmth, fluid therapy, nutritional support and intestinal lubricants and laxatives may all be helpful. Environmental control is paramount. Avoiding contact with contaminated feces and providing a dry environment to stop embryonation of eggs are all important. Decreasing environmental load and reinfection of an aviary by suitable housing and quarantining of new birds are all recommended.

Cestode infections can cause problems in the avian intestinal tract. Since their life cycles are largely indirect and involve an intermediate host such as an insect, mollusk or arthropod, cestodes are more a problem of birds with access to the ground.[77,129] They are most common in insectivorous finches and parrots of wild stock, particularly cockatoos, African greys and eclectus parrots.[129] Infections may cause diarrhea, ill thrift and death, particularly in finches.[138] Diagnosis is via the presence of proglottids in the feces. These may rupture, releasing the eggs. Microscopically, the eggs contain the hexacanth larvae with six hooks on the oncosphere. In some birds, the cestode can be visible, protruding from the cloaca after defecation. However, proglottids may not always be shed or may not rupture, so infections can be missed. Praziquantel (10 to 30 mg/kg orally, repeated in 14 days)[77,81,138] appears to be the most effective cestocide. Avoidance of exposure to intermediate hosts is important in control.

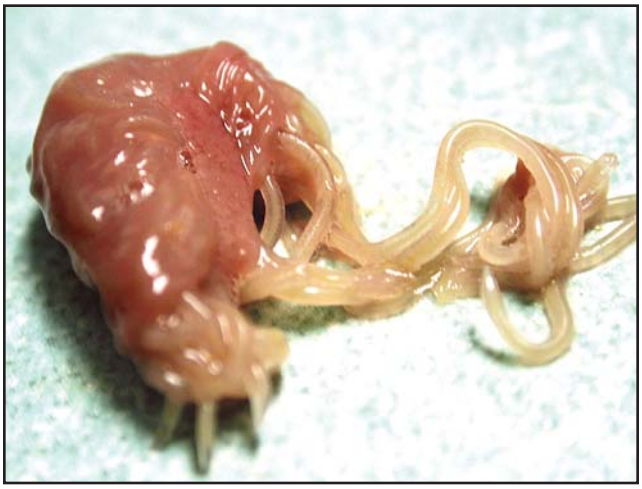

Gwen Flinchum

Fig 14.17 | A lethal intestinal nematode obstruction in this Quaker parakeet is an unusual finding in captive raised pet birds in the USA. To compound the issue, a fecal exam for parasites was negative.

Intussusception

Intussusception of the distal small intestine is less common in birds than in mammals and mostly occurs in gallinaceous birds secondary to enteritis.[44] As the proximal segment telescopes into the distal segment, blood flow is impaired and intestinal necrosis follows. Rectal intus-

Greg J. Harrison

Fig 14.18a | A malnourished female budgerigar with rhinal discharge over a hyperkeratotic cere.

Greg J. Harrison

Fig 14.18b | Same budgerigar in (a) showing swollen abdomen.

Greg J. Harrison

Fig 14.18c | The swelling was caused by massive fecal retention. A celomic mass, usually a tumor, is involved in such an obstruction. Changing the angle of the vent allowed 5-10 cc of feces to be removed, temporarily giving the bird and the owner time to decide on the final fate.

Greg J. Harrison

Fig 14.18d | An intestinal obstruction resulting from auto-obstruction in a parrot due to a necrotic intestinal lining slough. The cause was never determined. The necrotic section passed on day 3 of tube feeding and fluids, and the bird recovered.

susception may result in the rectum telescoping onto itself or into the coprodeum, where it may protrude from the vent lips. Both of these are medical and surgical emergencies, which may involve resection of the offending piece of bowel using magnification, and very fine sutures. (6-0 to 8-0).[90]

Ileus

Ileus or intestinal hypomotility/amotility can be caused by both physical obstructions (in the intestinal lumen, wall or as a result of external extra-intestinal compression) and by poor motor function. Examples of the former include foreign bodies, neoplasia, heavy parasite burdens, granulomas, strictures and various torsions and adhesions. Paralytic ileus can be caused by enteritides, PDD, peritonitis, lead toxicity and thrombosis of splanchnic vessels.[44] The impaired section of bowel dilates with intestinal fluid and gas. The bird becomes dehydrated. Ischemic necrosis of the intestinal wall leads to further fluid and protein loss. Gram-negative bacteria proliferate and produce endotoxins that can result in shock. Death can occur within 24 to 48 hours. Depending on how acutely the bird is affected, clinical signs can vary from vomiting, diarrhea, depression, listlessness, anorexia, decreased fecal output and emaciation.[44,90] In affected birds, abdominal palpation is resented.[37,115] Plain and contrast radiography may reveal dilated gas-filled bowel loops and identify the location of any obstructions. Supportive therapy with fluids, antibiotics, particularly for anaerobes, and analgesia are all recommended. Corrective surgery may then be performed to either remove or relieve any obstructions or to resect any debilitated sections of bowel.

Neoplasia

Primary intestinal neoplasms include carcinomas, papillomas, smooth muscle tumors and lymphosacoma.[57,85] Lymphosarcoma presents as diffuse or nodular thickening that may be mistaken for other conditions such as mycobacteriosis. Leiomyomas and leiomyosarcomas present as firm, red-brown masses within the intestinal wall and can only be distinguished from one another histologically (Figs 14.18a-d).[57]

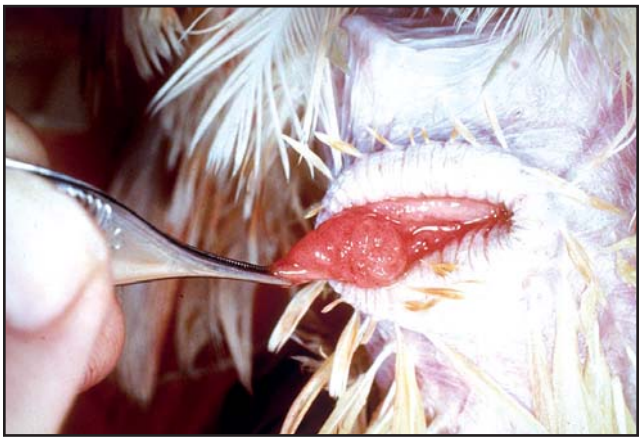
DISEASES OF THE CLOACA

Infectious

Speculums allow complete observation of the cloaca (Fig 14.19). Internal Papillomatous Disease (IPD) can be responsible for the irregular, cobblestone mucosal lesions seen in the psittacine proctodeum (Figs 14.20). IPD is one of the most common cloacal masses seen in

Greg J. Harrison

Fig 14.19 | A cloacal speculum can make observation of this complex structure much more understandable. The probe on the left side is in the vagina of a female cockatoo with PBFD. The fold containing the vaginal orifice separates the urodeum from the proctodeum. The bird is in dorsal-ventral position.



Espen Odberg

Fig 14.20 | Gastrointestinal disorders often involve papillomatosis. Any case of loose or smelly stool in a susceptible species should be investigated.

birds, particularly South American species. Moderate to severe lesions may lead to partial proctodeal obstruction.[31] Affected birds typically present with tenesmus, bloody droppings, malodorous feces, flatulence and staining of the vent and tail feathers with urofeces. Definitive diagnosis is via biopsy, but affected lesions will usually blanch when dilute acetic acid is applied to them. Various treatments have been suggested. These include sharp surgical excision, electrosurgery, silver nitrate cautery, cryosurgery, laser surgery and mucosal stripping.[139] All procedures carry the risk of causing iatrogenic traumatic cloacitis. Recurrences of the papillomas are common. Some lesions spontaneously regress but may recur. An empirical report of a commercial pepper diet allowing regression, as long as birds were fed the diet, has been reported (G. Harrison, personal communication, 2000) (see Chapter 32, Implications of Viruses in Clinical Disorders).

Bacterial cloacitis is rare in most birds but can create significant pathology when it occurs. It can occur as a result of localized trauma (such as cloacoliths, chronic cloacal prolapse), coexisting disease (such as internal papillomatous disease) or nutritional deficiencies. *Candida* spp. have most commonly been isolated from the proctodeum and vent lips but *Trichosporon begielli* has been found in one immunocompromised macaw.[31] These infections should be treated with appropriate antimicrobials both topically and systemically, and any underlying causes need to be corrected.

Cloacoliths are firm aggregations of urates that collect in the cloaca. They will at times also contain fecal material. They are often the result of iatrogenic intervention for other cloacal disease (eg, surgery or during forceful cloacal examination or sampling). The exact pathogenesis of cloacolith formation is unknown but is believed to involve impaired defecation with retention of urates that may cause dehydration. This may solidify and chemically alter the urate mass, causing it to form a solid structure. Gentle removal, application of topical cleaning, antibiotic and anti-inflammatory agents, systemic antibiotics and regular monitoring of the affected area may be required. In chronic cases, recovery can be slow and characterized by repeated recurrences.

Cloacal Prolapses

Cloacal prolapses are not uncommon in birds and can take one of several forms. Oviductal prolapse occurs in egg-laying females that strain excessively to lay due to uterine or egg-related factors. These often need to be surgically repaired, which may involve a hysterectomy if the oviduct damage is severe. Endoscopy may need to be performed to differentiate oviductal from rectal prolapses. Idiopathic coprodeal prolapse is seen in male cockatoos in particular and less commonly in other psittacines. The exact cause is unknown, but it is suspected that affected birds have never been fully weaned, are bonded to their human companions and interpret their owner's behavior such as petting as sexually stimulating. This is distinct from the overt masturbation exhibited by some cock birds in the presence of the owner. Various surgical techniques have been described, including cloacopexy of the ventral cloaca to the abdominal wall and ventplasty. Hormonal investigations and chemical and surgical neutering are all being evaluated.[31] Behavioral modification may be appropriate. A true intestinal prolapse can occur if a rent from the cloaca or rectum is opened into the abdomen (Fig 14.21).

Phallic Prolapses

Phallic prolapses have been described in waterfowl and

Greg J. Harrison

Fig 14.21 | A massive intestinal prolapse contains the congested pancreas. This can only occur from a fistula from the cloaca or rectum into the peritoneal cavity.

ostriches and result from excessive sexual stimulation, particularly in younger males, as well as other causes of phallic trauma and underlying systemic disease.[139] Cleaning and replacement of the phallus in the ostrich so that the tip is resting in the dorsal cloacal sulcus and sexual rest are the treatments of choice. Occasionally stay sutures across the vent may be required to keep the phallus in place. Treatment with antibiotics and anti-inflammatories may be necessary if the phallus has been traumatized.[139]

Neoplasia

Cloacal carcinomas are infiltrative tumors leading to thickening of the cloacal wall. Smooth muscle cloacal tumors are infrequently reported.[57]

Diseases of the Exocrine Pancreas

Although diseases affecting the pancreas will often impair both endocrine and exocrine function, only the exocrine effects will be discussed here.

Pancreatitis occurs when the digestive enzymes such as trypsin, protease and phospholipase are prematurely activated within the gland and begin to digest it. The resultant damage to the cell wall leads to the release and activation of these enzymes into the ducts and extracellularly. Free radicals are produced causing further damage. The initiating cause of this autodigestion can be difficult to pinpoint, but several factors have been recognized.[140] Viral infections such as Paramyxovirus type 3, herpesvirus, polyomavirus, adenovirus and avian influenza A can cause necrosis and variable inflammation. PMV-3 can also cause chronic pancreatitis leading to a firm and irregular pancreas. Histologically, a variably lymphoplasmocytic inflammation can be seen, with the formation of lymphoid follicles evident. *Neophema* spp. seem particularly susceptible to this form of the disease.[75] A number of bacterial agents have also been associated with pancreatitis. Non-infectious causes include obesity associated with fatty diets or high fat meals, zinc toxicosis and secondary damage from egg yolk peritonitis.[141] Birds on all seed diets seem particularly prone to pancreatitis. Psittacines, particularly quaker parakeets, may die suddenly from acute pancreatic necrosis.[57] The pancreas from affected birds appears pale and firm and may exhibit variable degrees of hemorrhage. There is often necrosis of adjacent fat. Histologically, the lesions include coagulation necrosis of pancreatic acini, intralobular hemorrhage and necrotic foci in mesenteric adipose tissue.[57] Zinc toxicity targets the pancreas, causing vacuolation and degeneration of acinar cells. Grossly, the pancreas may appear normal or exhibit mild parenchymal mottling.[57]

Diagnosis of pancreatitis can be difficult. Clinical signs are non-specific but may reflect gastrointestinal dysfunction and pain. Vomiting, diarrhea, anorexia, lethargy, ileus, weight loss, polyuria and polydypsia and abdominal distension are some of the signs noted.[140,141] Signs of abdominal pain include kicking, feather plucking (especially around the abdomen), falling off the perch, wide-based stance, sudden flight attempts, aggression and obsessive chewing. Measurement of blood amylase levels has been described, but absolute normal values are yet undetermined for most species. However, amylase levels above 1,100 IU/dl are considered elevated.[140,141] Increases of only 2 to 3 fold may be attributed to extra-pancreatic causes such as gastrointestinal disease (eg, small intestinal obstruction), renal disease or glucocorticoid administration. Thus, interpretation of blood levels needs to be done with care. Pancreatic biopsy is the method of choice for diagnosing pancreatic disease. This can be achieved via laparotomy or endoscopically through the right thoracic air sac. A histological examination may also shed light on likely effective treatments and prognosis.

Treatments for pancreatitis are based on mammalian strategies.[140,141] Fluid therapy to improve pancreatic perfusion is important. Converting birds onto low-fat pelleted diets is preferred to withholding food due to the high metabolic caloric requirements of most birds. Analgesia (eg, butorphanol, carprofen) to counteract abdominal pain, intestinal motility stimulants such as metoclopramide or cisapride to counteract intestinal ileus and parenteral antibiotic therapy are all of value in dealing with this disease. Any underlying causes should also be treated (eg, zinc toxicosis). Some workers have used omega-3 fatty acids for their lipid stabilizing and anti-inflammatory properties.[140,141] In life-threatening cases, plasma transfusions may help by replacing protease inhibitors and thus stopping further pancreatic damage.[141]

Pancreatic enzyme therapy may help stop pain by inhibiting the endogenous production of pancreatic enzymes; it is useful in treating pancreatic insufficiency,

which may follow a bout of acute pancreatitis.[141] Exocrine pancreatic insufficiency manifests in the production of pale voluminous feces. It can result from any chronic inflammatory process that may affect the pancreas, including those listed as causing acute pancreatitis.

Pancreatic neoplasms can be either benign or malignant. Birds suffering from IPD seem to have a high incidence of pancreatic adenocarcinomas[142] that may present as masses that have caused adhesions between viscera and peritonitis.[57]

Products mentioned in text

a. Hypo-Cal.® Calcium hydroxide. Ellman International, Inc., Hewlett, NY www.ellman.com.

b. Temp-Plus® Liquid. Ellman International, Inc., Hewlett, NY www.ellman.com.

c. Temp-Plus Resin.® Ellman International, Inc., Hewlett, NY www.ellman.com.

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