Clinically Significant Unique Features of the Avian Respiratory Tract (ART)

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The avian respiratory tract (ART) differs from that of mammals in numerous anatomical and functional ways. The unique features of the ART are of crucial significance to the practitioner of avian clinical medicine and surgery. Some of the more significant features (in this writer's opinion) of clinical import are discussed below.

Paranasal sinuses:

The paranasal sinuses of birds, unlike those of mammals, are extra-osseous; they are limited by the orbit, ocular globe, zygoma, levator muscles of the mandible, and facial skin. The sinuses are lined by simple low cuboidal to pseudostratified columnar epithelium. The epithelium reflects upon the adventitial surface of several nerves and associated blood vessels which traverse the sinus cavity.

Sinusitis, often accompanied by some degree of rhinitis and caused by a variety of bacterial, viral, and fungal agents, is frequently observed by the avian veterinarian. Serous to mucinous nasal discharge may be the initial or only presenting sign. Accumulation of exudates within the sinuses may ultimately cause swelling of the infraorbital, preorbital, or supraorbital skin, either independently or in different combinations. Note that prominent infraorbital swelling is often mistaken for conjunctivitis.

Transillumination of the sinuses to visualize the extent to which the sinus is filled with exudate is easily accomplished in a darkened room by placing, with aid of an appropriate oral speculum, a bright illuminator within the mouth or oropharynx and viewing the light transmitted thru the sinus region. Transillumination examination of the sinuses is often more revealing than are radiographs.

Etiological diagnosis of infectious sinusitis requires culture of samples collected aseptically by aspiration or sinus lavage. Attempts to culture the choanal cleft or

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nasal discharge routinely yield spurious results and rarely reveal the agent acting within the sinus.

Percutaneous aspiration of sinus contents requires care that the globe not be lacerated or punctured by the aspiration needle. The site of needle introduction entailing the least hazard is the palpable depression in the skin immediately caudal to the beak commissure and inferior to the rostral portion of the zygoma. A needle inserted at this site can be directed caudally or caudo-ventrally into the infraorbital portion of the sinus, partially withdrawn, and re-directed dorsally into the preorbital region of the sinus with the least risk of being accidentally displaced or misdirected by unanticipated movement of the maxilla or mandible. Such is a greater hazard if the usually larger - and thus more tempting - suprazygomatic space is used as the entry site.

"Lock Jaw"

"Lock jaw" has become an increasingly common sequel to bacterial sinusitis since it was first recognized in 1981. In the United States it is encountered almost exclusively in cockatiels. Hand-fed nestlings and weanlings are at greatest risk, although occasional cases have been reported in adults. Inflammation extends thru the medial mucosal epithelium of the infraorbital region of the sinus and into the immediately subjacent mandibular levator muscles. The inflamed muscles become progressively less extensible and ultimately contracted with fibrous scar tissue as the myositis progresses from acute to chronic.

Necropsy of many affected hand-fed babies reveals that the sinuses suffered accidental instillation of feeding formula into the choana, and thence to the sinuses. Such has not been observed in parent-fed nestlings which suggests that, in some cases, misdirection of formula may be a consequence, rather than an inciting cause of the sinusitis.

Bacterial cultures from affected sinuses have yielded a variety of bacterial infections, predominantly by Gram-negative organisms; *Bordetella sp*.was incriminated as the etiologic agent in at least one aviary in Florida.

Hypovitaminosis-A

A common causes of sinus swelling is squamous metaplasia of the sinus epithelium and accumulation of a compact mass of keratin debris resulting from hypovitaminosis-A. Secondary bacterial infection of sinuses so affected is, surprisingly, uncommon to rare. Needle aspiration of the keratin is usually impossible without the aid of concurrent instillation of sterile saline solution. Cytology of the aspirate reveals numerous epithelial squames. Absent secondary bacterial infection, the prognosis for full recovery is excellent with dietary vitamin A supplementation. The suggestion that a bird deficient in vitamin A is unable to absorb it from the diet

due to intestinal epithelial lesions has no basis in fact; the intestinal epithelium is unaffected, and it readily absorbs β-carotenes - which it converts to functional forms of vitamin A - as well as pre-formed vitamin A itself.

Larynx, Trachea and Syrinx:

The avian glottis is a longitudinal slit in the dorsum of the larynx and lacks an epiglottis. The laryngeal response to a solid foreign body at its threshold rapid and competent; accidental or inadvertent tracheal intubation with even a small diameter gavage tube is thus unlikely, if not nigh on to impossible. Acute glottal obstruction by an inhaled foreign body is an exceeding rare event. This pathologist has observed three cases in the last 31 years; a piece of muscle meat in the glottis of a Cooper's hawk, an intact sunflower seed in an Amazon parrot, and a mass of compacted down feather in a peregrine falcon.

Fatal glottal obstruction as a result of progressively growing lesions on the glottal margins does occur in occasional cases of poxvirus infection and internal papillomatous disease.

The avian trachea is difficult to collapse owing to its complete and usually ossified tracheal rings. Primary tracheitis is rare in companion bird species. The single most common cause of acute to chronic endotracheitis is respiratory acariasis which is common in estrildid finches and fringillids infected by *Sternostomum tracheocolum*. Transtracheal illumination easily reveals the live mites moving about within the tracheal lumen.

The bird's organ of vocalization is the syrinx at the site of bifurcation of the trachea. Syringeal structure varies widely among avian species. It is a complex structure comprised of complete rings, incomplete rings, intrinsic and extrinsic muscles, and two or more flexible membranes. Tension upon the membranes (which affects the frequency of air vibration) is influenced by the intrinsic muscles and by the pressures within the immediately adjacent interclavicular air sac. The initial earliest clinical sign of syringeal dysfunction is loss of vocalization or change in the character of vocalizations.

The single common non-infectious cause of syringeal obstruction is hypovitaminosis-A with consequent endosyringeal hyperkeratosis. The single common infectious cause of obstructive endosyringitis is mycotic infection - usually *Aspergillus fumigatus*. In both instances, if diagnosed before obstruction is complete and fatal, endosyringeal debridment via endoscopy may relieve dyspnea and permit appropriate treatment of the primary disease; respiration is aided during surgery by positive-pressure ventilation of the caudal air sacs, and placement of an isobaric stent or air sac tube in the interclavicular airsac to permit unrestricted exhalation of respired air.

Rarely, endosyringitis may be secondary to inhalation of foreign material or of septic

exudate fragments from higher or lower in the ART.

Lungs:

The avian lungs are the sites of gaseous exchange between blood and air just as they are in mammals. Here the similarities to the mammalian lung end.

The bird's lung does not depend upon significant cyclic expansion and collapse to deliver oxygen-rich air to the gas-exchange sites. It has no blind sacs analogous to mammalian alveoli. Absence of blind sacs absolves the avian lung of risk of *pulmonary* atelectasis (lack of dilation of end spaces in the lung - A.K.A. alveoli). Because the lung need not expand and contract and thus need not slide its surface against the inner thoracic wall or mediastinal structures, a pleural cavity lined with slippery pleural mesothelium is neither needed nor present. Indeed, the dorsolateral surface of the lung is gently adhered to the body wall by sparse,loose connective tissue.

Oxygen-rich air is moved from the caudal air sacs into the lungs, passes unidirectionally thru increasingly fine air channels (tertiary bronchi), then via atria in their walls and into the air labyrinth (air "capillaries" are a misnomer) where gas exchange with blood in pulmonary capillaries occurs. Efficiency of gas exchange in birds considerably surpasses that in mammals due to countercurrent flow of oxygen-rich air against oxygen-depleted blood. The oxygen-depleted, CO_2-replete air passes thru progressively larger channels whence it ultimately enters the cranial air sacs prior to exhalation.

So…. "How's all this relate to clinical concerns re: avian respiratory diseases?" I hear you cry. Patience ! Lets talk about the air sacs and the respiratory cycle of birds!

Air Sac System:

The air sacs of birds are, from the point-of-view of ventilatory function, analogous to the alveoli of the mammal's lung. In mammals, air is moved by changes in alveolar volume, in birds, by changes in air sac volume. The air sacs are blind sacs, the volume of which is totally and passively dependent upon 1.) free entrance and exit of air and 2.) the cyclic adaxial and abaxial excursion of the sternum which changes the volume of the coelom in which the air sacs are fixed. When coelomic volume is expanded, the air sacs fill with air under atmospheric pressure; when the coelom is compressed, the air sacs are likewise, and their air is expressed against atmospheric pressure.

The air sacs may be compressed to the point of complete collapse by intracoelomic space-occupying lesions. For example, ascites, severe obesity with excessive depot

fat, or a distended, "retained" yolk sac may cause collapse of the caudal air sacs - *air sac atelectasis*. Elimination of air sac volume eliminates the possibility of lung ventilation. Birds so-affected evidence dyspnea and die of suffocation unless the cause of compression is removed. Paradoxically, Pneumo-coelom that results from surgical disruption of air sac membranes does not usually interfere with respiration because the air-filled coelom becomes a functional extention of the original air sac space.

The avian respiratory cycle:

Avian Respiration is a **two-cycle** phenomenon; it requires *two cycles of coelom expansion and compression* to move a single bolus of air completely thru the ART. The path of a single bolus of air from the moment it is inhaled into the trachea to the moment it is passed out of the trachea is as follows:.

On **expansion #1** (inhalation of the air bolus) air moves into the larynx, thru the trachea, thru the primary bronchus and mesobronchus (without entering the lung's gas exchange regions) and into the caudal air sacs. The air moves under atmospheric pressure into the caudal air sacs because they are under negative pressure.

On **compression #1** air is moved from caudal air sacs into the lung for gas exchange under positive pressure.

On **expansion #2** air is moved from the lungs into the cranial air sacs under negative pressure.

On **compression #2** (exhalation of the air bolus) air is moved by positive pressure from the cranial air sacs to the upper bronchus and trachea and into the great outdoors.

Obstructive respiratory disease

Chronic obstructive respiratory disease, sometimes accompanied by (presumeably) compensatory polycythemia, is usually the result of obstruction of air flow from the tertiary bronchi, thru the atria, and into the and air labyrinth.

Idiopathic hypertrophy of the smooth muscle of interatrial septa of macaws has been observed with increasing frequency by this pathologist since 1979. Progressively severe dyspnea, initially noticed as a prolonged post-exertional respiartory rate recovery interval, is the common presenting complaint. Some macaws so affected develop polycuthemia.

Severe, chronic *pneumonoconiosis* is characterized by massive accumulation of particle-laden macrophages in the walls of interatrial septa. Septal wall thickness eventually restricts air movement into the air labyrinth.

Inhalation and Distribution of Air-borne Particles

The basic principles of deliverance of particulate, airborne material to the various regions of the respiratory tract in mammals apply equally in birds. Large, heavier particles settle out first in the larger airways and sites where air is temporarily slow moving, eddying, or static. Progressively smaller particles remain longer in suspension and tend to settle out in the smaller air channels.

Thus, an aerosol of suspended large particles is likely to settle out in the caudal air sacs. Progressively smaller particles remain longer in suspension and settle out in progressively smaller and more tortuous channels of the tertiary bronchi and air labyrinth.

From the perspective of inhalation therapy, most of a coarse aerosol of inhaled medication will settle out in the caudal air sacs; a more finely atomized aerosol will reach the lung's air labyrinth, but is not likely to not likely to pass on to the cranial air sacs.

Note that the distribution of lung lesions of aerogenous infectious pneumonia that occurs in mammals - the classic "anterior and ventral distribution" is not observed in birds. The smallest and most tortuous portions of the tertiary bronchial system of most avian species are in the dorso-lateral lung regions. Although discrete infectious lesions in this region of lung may be visualized radiographically; they are not detectable by endoscopic examination of the ventro-medial lung surfaces and will be missed at necropsy unless the lungs are reflected from the body wall and their dorso-lateral regions inspected.

Inhalation of liquids:

Liquids such as regurgitated water, medications, radiographic contrast media, hand-feeding formula, regurgitated gavage formula, etc…, if of a sufficiently fluid consistency, are readily inhaled into the caudal air sacs (expansion #1) under atmospheric pressure. They may remain there if 1) their volume is small and 2) the bird is not recumbent. If the fluid volume is greater or the bird is recumbent, the offending liquid may hain access into the lungs under positive pressure during compression#1. Thus, careful case selection is necessary and caution exercised when performing air sac lavage for microbiological or cytological specimen collection.

So… how come we see cases of inhalation pneumonia in which the caudal air sacs are devoid of the stuff, but it's in the lung - or maybe in the cranial air sacs ? Well? How about that, Huh?

Although such cases seem to defy the principles of movement of air or thin liquids thru the ART, such seeming exceptions "prove the rule." In those instances the inhaled material is usually a relatively thick liquid or gruel, the viscosity of which

does not permit its ready flow down and through the mesobronchus and into the caudal sacs (as occurs with more watery fluids). Instead, it is retained in the mesobronchus. An active and forcible cough occurs and some of the material is forced into the lung thru channels normally utilized by air during compression #1. In cases in which the thick material is retained in the upper regions of the mesobronchus, some of it may be forced, retrograde, into the channels normally serving for evacuation of air from the cranial sacs

It is hoped that the foregoing has provided some novel perspectives from which to consider the avian respiratory tract in health and disease.