TRACHEAL OBSTRUCTION IN PARROTS: AN APPROACH TO DIAGNOSIS AND TREATMENT

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INTRODUCTION

Tracheal obstruction, while not a common problem, can present as an acute, life-threatening condition. This presentation requires the clinician to have a sound knowledge of the clinical signs, the immediate steps necessary to keep the patient alive, the procedures needed to diagnose the cause of the problem, and how to resolve common causes of tracheal obstruction.

This paper presents a suggested approach to diagnosing and treating a parrot presented for dyspnoea associated with tracheal obstruction. To illustrate the success the author has had with this approach, three case reports are presented; a mid-tracheal obstruction with a millet seed; an Aspergillus granuloma in the syrinx; and a mid-tracheal stricture following the inhalation of plant material.

ANATOMY

The anatomy of the avian trachea is well adapted to allow variations in length and flexibility, properties that are desirable in birds because of the mobility of the neck. Overall, the typical avian trachea is longer and wider than that of comparably sized mammals (Clippinger and Bennett, 1998). In chickens, the length ranges from 170 to 180 mm in males and 155 to 165 mm in females (McLelland, 1965).

The number of tracheal rings varies between species and individuals, with McLelland (1965) reporting the number of rings in the domestic chicken ranged from 108 to 126 in ten birds. Avian tracheal rings are complete. Nearly every ring is shaped like a signet ring, with a broad part forming the left or right half of its circumference and a slightly narrower part forming the other half, both the rostral and caudal edges being wafer-thin (Figure 1).

The diameter and shape of the tracheal lumen changes progressively from the first to last rings. McLelland (1965) observed that in chickens the rostral quarter of the trachea is transversely oval in cross-section whilst, except for the last few millimetres, the rest was circular. In its terminal part the trachea becomes vertically oval and this shape continues into the syrinx. The diameter of the trachea decreases progressively from the first to the last rings, being 6-7 mm in diameter where it first became circular in cross-section and 2-3 mm at its caudal end. It is the author's observation that this finding corresponds well to that observed in parrots, although obviously with different measurements.

As with the majority of birds, parrots have a tracheobronchial syrinx; the last of the tracheal rings fuses into a syringeal box, which then joins to the first of the bronchial rings. It has a number of variably ossified cartilages and vibrating soft structures, and is the site of voice production.

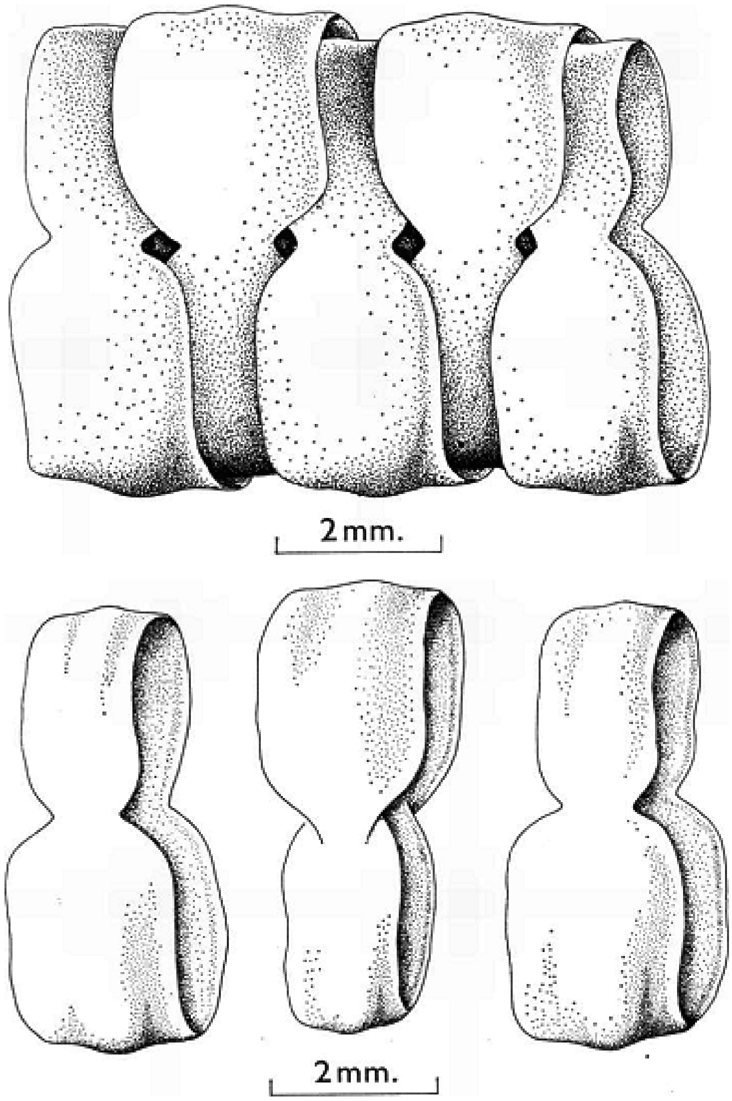

Figure 1. The structure of avian tracheal rings (from McLelland, 1965).

AETIOLOGY OF TRACHEAL OBSTRUCTION

The complete rings of the avian trachea make a functional obstruction (e.g. a collapsing trachea as seen in dogs) unlikely. The same anatomical feature makes external compression of the trachea equally unlikely, although Yeats (2000) reported a case where a fractured coracoid compressed the trachea in an Indian Ringneck Parrot (*Psittacula krameri*). This leaves a physical obstruction of the trachea the most likely problem to be seen in practice. It is the author's experience that these physical obstructions can result from three aetiologies: an inhaled foreign body; a diphtheritic, granulomatous or neoplastic lesion within the trachea or syrinx; or a stricture following an insult to the tracheal mucosa.

Inhalation of millet seeds causing a complete, or near-complete, obstruction is frequently seen in cockatiels (*Nymphicus hollandicus*). Other inhaled foreign bodies have included hand rearing formula (author's observation) or insects (Cannon, 2006). If the foreign body is large enough to occlude the diameter of the trachea, acute collapse and death frequently results. If the object is smaller than the tracheal diameter, acute respiratory distress is more likely to be the presenting sign.

Diphtheritic and granulomatous lesions are often slower to develop, making clinical signs of dyspnoea usually evident before acute collapse occurs. Diphtheritic lesions, in the author's experience, are more common in cockatoos, mid-tracheal in location, and are associated with a bacterial infection often secondary to the lodgement of inhaled objects such as plant material (Doneley 2010). Granulomatous lesions are more often seen in the syrinx and are associated with aspergillosis (Doneley, 2008).

Strictures are usually associated with trauma, either due to external wounds (such as those resulting from bites) or internal trauma following endotracheal intubation or other mucosal insults**.** The development of a stricture is usually more insidious, with obvious clinical signs often only seen when the bird's respiration is exerted by stress, exercise or handling. Careful observation of the bird at rest may reveal subtle mouth breathing and tail bobbing.

CLINICAL SIGNS

The clinical signs seen in a bird with a tracheal obstruction depend on the length of the time the obstruction has been present, and the degree of obstruction that is present. Birds with an acute obstruction may present weak and collapsed with the wings partially extended, the neck extended, obvious mouth-breathing and sternal lift, and audible inspiratory noises (Case report 1).

Case Report 1. Young adult cockatiel, kept indoors as a pet and fed a seed only diet. It was presented for sudden onset of dyspnoea and weakness some 12 hours previously. On presentation it displayed mouth-breathing, increased sternal lift, tail-bobbing and an audible respiratory noise. It was in good body condition, with no obvious abdominal swelling, displayed minimal handling tolerance and was weak.

However, birds with slowly developing obstructions may show milder signs that become more obvious only when the bird is exerted by stress or handling. These birds compensate for the obstruction by keeping still and have a slightly increased respiratory effort. Vocalisation is decreased and there may be a change in voice. As the degree of obstruction increases, the clinical signs become more obvious (Case Reports 2 and 3).

Case Report 2. An 8 year-old male Grey Parrot (*Psittacus erithacus erithacus*) housed indoors with six other parrots, all in separate cages in the same room. Its daily diet consisted of a formulated diet, fruit, vegetables, and spouted seed. It was handled daily by the owners and was tame and easily managed. He was presented for an unusual respiratory noise noted for the first time that morning.

On examination the bird was in good body condition. At rest it displayed no evidence of respiratory disease, but while been examined it exhibited moist inspiratory rales and a mildly increased respiratory effort. On auscultation moist rales were readily discernible.

Case Report 3. An 11 year old female Red-Tailed Black Cockatoo (*Calyptorhynchus banksii banksii*) was presented for sudden onset of dyspnoea and weakness following a history of aggression towards the bird by its mate. On examination the bird was in good body condition but displayed clinical signs suggestive of a tracheal obstruction including audible respiratory noises.

DIAGNOSIS

While the clinical signs described above are suggestive of an obstruction, they are not always pathognomonic. Lower respiratory tract disease i.e. pulmonary or air sac disease, can appear very similar even to experienced clinicians.

Endoscopy usually offers the most rapid and accurate tool for diagnosing tracheal obstruction. The author's approach is to mask induce the patient with isoflurane, and then perform a left flank laparoscopy to evaluate the lungs and air sacs; an air sac catheter is then placed unless contraindicated (e.g. by the presence of severe airsacculitis or pulmonary disease) and anaesthesia is maintained via this route. With practice and preparation, this can be performed in 1-2 minutes. With an alternate airway secured, tracheoscopy can then be performed to evaluate the trachea and syrinx.

Case Report 1. Tracheoscopy revealed a millet seed wedged in the mid-tracheal region**.**

Case Report 2. Tracheoscopy revealed a large white, glistening granuloma occluding approximately 85% of the tracheal lumen at the level of the syrinx. Cytology showed branching fungi consistent with *Aspergillus* spp.

Case Report 3. Tracheoscopy revealed diphtheritic plaques and exudate, leaving an opening approximately 10% of the luminal diameter through which the bird was breathing. After careful debridement of these plaques, a tracheal stricture was visible.

If endoscopy is not available, or the syrinx cannot be visualised, contrast radiography may be necessary in some birds to obtain a definitive diagnosis. A small amount of barium (0.5 ml/kg bodyweight) is placed in the trachea using a catheter. Barium is inert, and radiographs taken immediately may show a small filling defect in the tracheal lumen or syrinx (Dennis et al, 1999).

TREATMENT

The treatment of tracheal obstruction revolves around two key principles: the obstruction must be removed, and an alternate airway must be maintained until this is done.

Avian anatomy favours the clinician dealing with a tracheal obstruction. Not only does the flexibility and structure of the trachea make it more forgiving of attempts of remove an obstruction, an alternate airway is easily established via an air sac catheter. With patient in right lateral recumbency and the left leg drawn forward, a skin incision is made where the last rib passes under the *flexor crura medialis* muscle. Blunt dissection with a small pair of curved haemostats then provides access, firstly for endoscopy and then for an endotracheal tube, into the left caudal thoracic air sac. This catheter is then sutured to the skin and can usually be maintained for 3-5 days. If it becomes obstructed before then, or a longer treatment period is required, the process can be repeated on the right side.

All three birds in these case reports had air sac catheters placed as part of their treatment.

The next step is to relieve the obstruction. The approach taken here will be determined by: the location of the obstruction; the type of obstruction; and the size of the obstruction.

Small, non-adherent obstructions in the mid- to caudal trachea have been removed in a variety of ways. These have included removal with grasping forceps, suction, tracheotomy, or the introduction of a catheter caudal to the obstruction and pushing/blowing it rostrally.

Case Report 1. A 24g I/V catheter was introduced into the trachea, caudal to the millet seed. The stylet was removed and the catheter advanced rostrally. Once it physically dislodged the seed, a 10ml syringe of air was attached and the seed 'blown' out of the trachea. The air catheter was left in place for two days, and the bird recovered uneventfully.

Similar obstructions located in the syrinx may be approached through a distal tracheotomy and removed surgically (Dennis, 1999). (Another technique, once recommended but now out of favour, was to use a small bone pin to push the obstruction through the syrinx into the supporting tissues, where it could be left to form a granuloma outside the airways.)

Diphtheritic or granulomatous lesions present a different problem. Although they can be endoscopically de-bulked, care must be taken to avoid iatrogenic trauma to the tracheal mucosa. In the short-term this can lead to bleeding worsening the degree of obstruction, and in the long term may contribute to stricture formation. Nebulisation with hypertonic saline and/or acetylcysteine can help to 'dissolve' these lesions. Great care, however, must be taken to ensure the patency of the air sac catheter, as the lesions may swell prior to dissipation and completely obstruct the airway. If the lesion is an aspergillus granuloma, injections of diluted amphotericin-B trans-tracheally may be of assistance as well, although it must be remembered that this drug can be irritant to mucosal surfaces (Doneley, 2008).

Case Report 2. Treatment given was piperacillin-tazobactam 100mg/kg IM BID; itraconazole 2.5mg/kg PO BID, nebulisation with Amphotericin B (Fungizone®, Bristol-Myers, Squib Australia) and acetylcysteine (Mucomyst®, Bristol-Myers, Squib Australia) TID and Amphotericin B 1mg/kg diluted in saline and injected intra-tracheally SID. The obstruction cleared after 9 days, and the bird was treated for 6 months with voriconazole. It remains asymptomatic 3 years later.

Tracheal strictures can be removed by resection and anastomosis (Doneley, 2010). The flexibility of the avian neck confers a similar flexibility on the avian trachea, which is relatively longer than its mammalian counterpart (Clippinger and Bennett, 1998). This inherent flexibility makes the resection of even long pieces of trachea feasible in birds. One report estimated that, in one bird, 9% of the tracheal length was removed without adverse effect (Sanchez-Migallon Guzman et al, 2007).

Case Report 3. The tracheal stricture was resected and the tracheal ends anastomosed. Recovery was uneventful and the stricture did not recur. The bird remains clinically normal two years later

Conclusion

Tracheal obstruction in parrots, while life-threatening, can be effectively dealt with by utilising avian respiratory anatomy to provide an alternate airway while the obstruction itself is assessed and dealt with. The type and extent of the obstruction will determine the clinician's treatment, but the inherent flexibility of the avian trachea allows a greater flexibility in treatment options than does its mammalian counterparts.

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