Journal of Avian Medicine and Surgery 21(2):150–157, 2007

Tracheal Resection and Anastomosis in a Mallard Duck (*Anas platyrhynchos*) with Traumatic Segmental Tracheal Collapse

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Abstract: A male mallard duck (*Anas platyrhynchos*) presented for examination for acute respiratory distress and lethargy. The duck had experienced recurrent episodes of respiratory distress since being attacked by a raccoon the previous year, resulting in neck lacerations. Diagnostic tests, including a complete blood count, plasma biochemical analysis, radiography, and tracheoscopy, revealed a collapsed trachea. Surgical correction of the collapsed tracheal segment resulted in resection of 9% of the total tracheal length and subsequent anastomosis. Tracheoscopy performed 2 and 3 months after surgery revealed a healthy mucosa, minimal reduction of the tracheal lumen in the area of anastomosis, and minimal suture granuloma formation.

Key words: tracheal stenosis, tracheal collapse, tracheoscopy, tracheal resection, avian, mallard duck, *Anas platyrhynchos*

Clinical Report

A 2-year-old, 1.6-kg male mallard duck (*Anas platyrhynchos*) exhibiting clinical signs associated with acute respiratory distress presented to the Veterinary Teaching Hospital at the Louisiana State University (LSU) School of Veterinary Medicine. The owner and the duck had been displaced by Hurricane Katrina, and the duck had been placed in a temporary animal shelter on the LSU campus. The animal caretakers from the shelter reported that the duck started to appear lethargic 5 days before the episode of respiratory distress. The only other historical information was that the duck had been attacked by a raccoon the previous year and had sustained 4 lacerations around the neck and injuries to the right eye. Since the attack, the duck had experienced intermittent episodes of respiratory distress, which had resolved without complications.

On presentation, the duck was in severe respiratory distress and was placed in an oxygen cage for monitoring. After the duck's respiratory rate and effort had decreased, a physical examination was performed. The respiratory rate was 120 breaths per minute, and the bird had inspiratory stridor. A healed corneal injury was observed in the right eye, and a deformity was palpated in the cranial third of the trachea. Based on the history and physical findings, the differential diagnoses included tracheal stricture, fungal granuloma involving the trachea, syringeal, or ostium, tracheal or syringeal foreign body, airsacculitis, pneumonia, and toxin inhalation.

Anesthesia was induced with 5% isoflurane delivered by face mask. The duck was intubated with a 3.5-mm uncuffed endotracheal tube and manually ventilated with 2% isoflurane for radiographs. Right lateral and ventrodorsal whole body survey radiographs revealed loss of detail and increased soft-tissue opacity of 12 tracheal rings at the level of the fifth and sixth cervical vertebrae (Figs 1 and 2). No other abnormalities were observed. A blood sample was collected from the medial metatarsal vein and submitted for a complete blood cell count and plasma biochemical analysis. The duck was placed in

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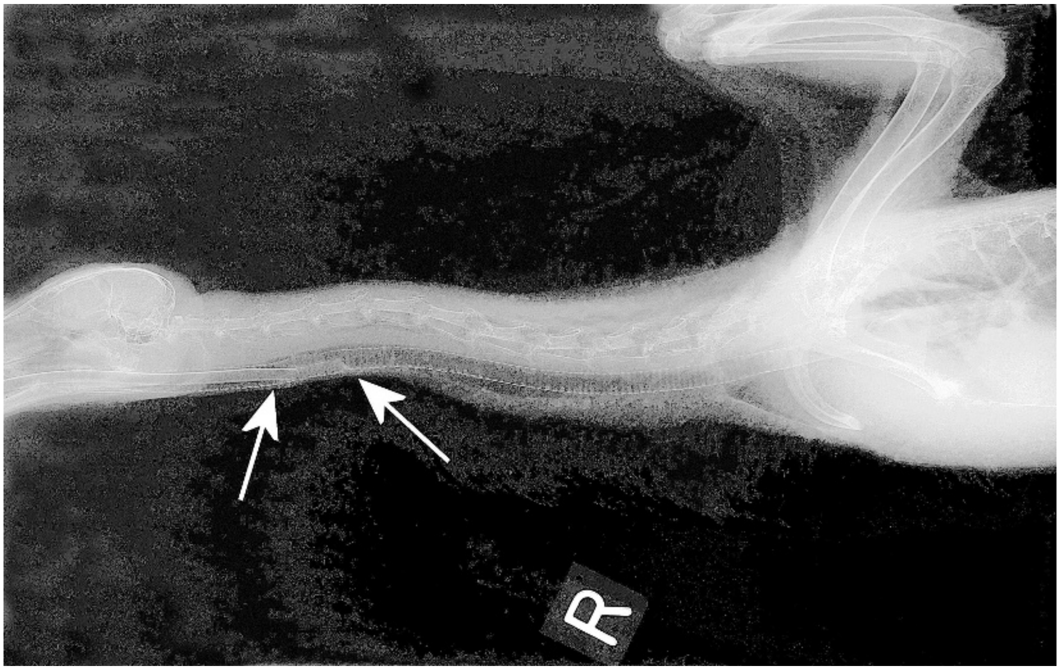

Figure 1. This right lateral radiograph of a 2-year-old male mallard duck with acute respiratory distress reveals loss of detail and increased soft-tissue opacity involving 12 tracheal rings at the level of the fifth and sixth cervical vertebrae (arrows).

lateral recumbency for air sac cannulation, and a 4.0-mm fenestrated endotracheal tube was inserted into the caudal thoracic air sac through a skin incision in the left flank immediately caudal to the last rib. The tube was sutured to the lateral abdominal wall, incorporating the last rib, with 2-0 polypropylene suture (Surgipro, Johnson & Johnson, Sommerville, NJ, USA). Dyspnea resolved immediately after air sac cannulation. The duck recovered uneventfully from anesthesia.

Results of the complete blood cell count and plasma biochemical analysis were within reference ranges, except for monocytosis (2.0×10^3 cells/µl; reference range $0.2–1.0 \times 10^3$ cells/µl).[1] Postoperative treatment consisted of enrofloxacin (15 mg/kg PO q12h; Baytril, Bayer Health Care LLC, Shawnee Mission, KS, USA), meloxicam (0.2 mg/kg PO q24h; Metacam, Boehringer Ingelheim Vetmedica, St. Joseph, MO, USA), butorphanol (1 mg/kg IM once; Torbugesic, Fort Dodge, Fort Dodge, IA, USA), and supplemental fluids (50 ml/kg SC q12h).

The duck was anesthetized the next day for tracheoscopy. Anesthesia was induced and maintained through the air sac cannula by the anesthetic protocol described above. Tracheoscopy revealed segmental narrowing of the tracheal lumen that had reduced the luminal diameter by 70%–80% (Fig 3). The tracheal mucosa was inflamed, and a large amount of tracheal secretion obscured visibility. Findings were compatible with tracheitis and stenotic tracheal deformity. The duck recovered uneventfully from anesthesia and was scheduled for tracheal resection and anastomosis.

The duck was premedicated with butorphanol (0.5 mg/kg IM, Fort Dodge) and glycopyrrolate (0.01 mg/kg IM). Anesthesia was induced and maintained with 2.5% isoflurane through the air sac cannula, and the duck was manually ventilated at a rate of 12 breaths/min. An intravenous catheter was placed in the medial metatarsal vein, and fluids were administered throughout the surgery (Normosol, 5 ml/kg/h). The duck was positioned in dorsal recumbency, feathers overlying the ventral neck were removed, and the site was prepared for aseptic surgery. A 6-cm incision was made along the ventral midline of the neck approximately 12 cm cranial to the thoracic inlet. Subcutaneous tissues were dissected to reveal the paired sternothyroideus muscles, the separation of which was prevented by adhesion at the site of the tracheal deformity. The recurrent nerves were also tightly adhered to the trachea (Fig 4). During attempts to break down the adhesions, the recurrent nerves were torn. A tracheal ring cranial and caudal to the deformed segment was bisected circumferentially with a No. 11 scalpel blade. A total of 12 tracheal rings were resected. The tracheal ends were approximated by preplacing 6

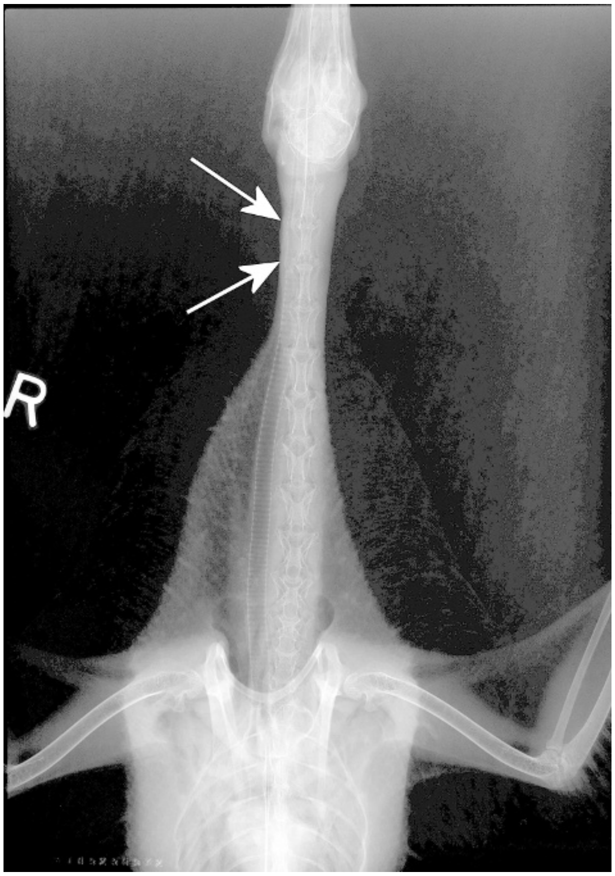

Figure 2. Ventrodorsal radiograph of the bird described in Figure 1 confirmed the findings (arrows) on right lateral view.

Figure 4. Intraoperative photograph showing the collapsed tracheal segment prior to resection and anastomosis.

simple interrupted sutures of 4-0 polydioxanone (PDS, Johnson & Johnson) on a tapered needle. The sutures were tightened individually to appose the tracheal ends. Three tension-relieving sutures (4-0 polydioxanone on a tapered needle) were placed on the ventral and lateral aspects of the trachea, each encircling a tracheal cartilage proximal and distal to the anastomosis site. All suture knots were extraluminal (Fig 5). The sternohyoid muscles were apposed over the ventral aspect of the trachea using 4-0 polydioxanone suture in a simple continuous pattern. The intradermal layer was closed in a subcuticular pattern using 4-0 polydioxanone on a tapered needle. One cruciate suture (4-0 polydioxanone on a cutting needle) was laced through the skin at the proximal aspect of the incision. The incision site was covered with a permeable wound dressing (Opsite, Smith and Nephew Medical Unlimited, Hull, England). The resected trachea was submitted for histopathologic examination. Recovery from anesthesia was unremarkable. During the 24 hours after surgery, the duck had episodes of mild coughing that resolved without complications. Treatment with metronidazole (50 mg/kg PO q24h) was initiated.

The air sac cannula was removed 2 days after surgery, and a sample collected from the tube

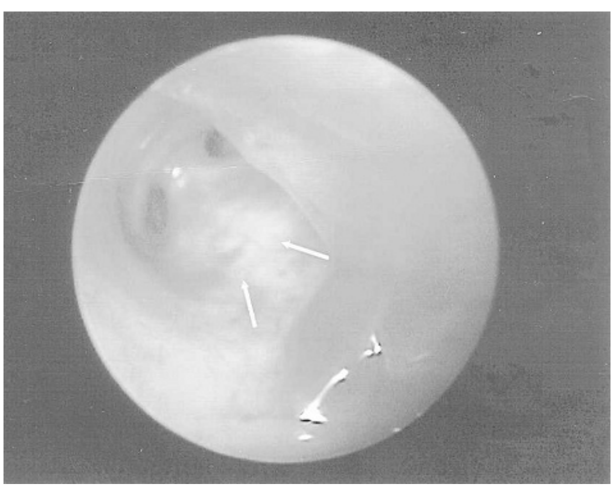

Figure 3. Tracheoscopy shows increased tracheal secretion, inflamed mucosa (arrows), and segmental narrowing of the tracheal lumen with a 70%–80% decrease in luminal diameter.

Figure 5. Intraoperative photograph showing the trachea after resection of the collapsed tracheal segment and anastomosis.

was submitted for aerobic culture and sensitivity. The entry site of the air sac cannula was allowed to heal by second intention.

The duck was discharged 3 days after surgery on treatment for 7 days with enrofloxacin (15 mg/kg PO q12h), meloxicam (0.2 mg/kg PO q24h), and metronidazole (50 mg/kg PO q24h). A recheck appointment was scheduled for 7 days later.

An alpha-hemolytic *Streptococcus* species, which was resistant to enrofloxacin, was isolated from the air sac cannula. Although attempts were made to change the antibiotic, the owner did not return to obtain medication.

Histopathologic findings included marked tracheal deformity, tracheal mucosal ulceration, and necrotic debris containing mucus in the tracheal lumen (Fig 6). The submucosa was diffusely infiltrated with heterophils and lymphocytes. The tracheal rings were largely replaced by mature bone containing bone marrow. This bone was markedly deformed, containing a steep angle resembling a fold on one side and a V formation protruding into the lumen in the center.

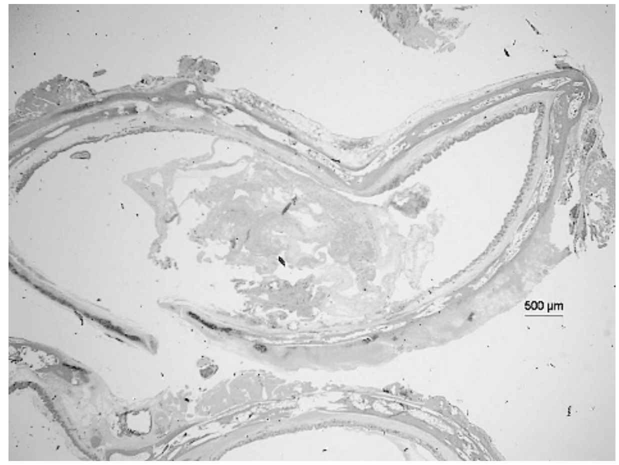

Figure 6. Pathologic findings of the tracheal segment removed from the duck included a markedly deformed trachea, mucosal ulceration, and necrotic debris containing mucus in the tracheal lumen.

The owner returned to have the duck re-evaluated 2 months after surgery. The bird had been doing well at home, but its vocalizations had changed. On physical examination, the duck weighed 1.75 kg. Suture material could be palpated in the area of the tracheal anastomosis. The bird was anesthetized the next day for tracheoscopy to assess healing and any sign of stricture. Anesthesia was induced with 5% isofluorane delivered by mask. Tracheoscopy revealed healthy mucosa and what was presumed to be mucus or suture material lying across the tracheal lumen. Despite attempts to confirm these findings, complications with anesthesia shortened the tracheoscopic exam. The duck went into respiratory arrest, which was resolved after a hemorrhagic mucus clot was expelled from the trachea with positive pressure ventilation. The duck exhibited a mild cough after recovery, which was attributed to tracheal trauma during the procedure. Seven days of treatment with trimethoprim-sulfa (20 mg/kg PO q12h) and meloxicam (0.2 mg/kg PO q24h) was initiated.

Three months after surgery, the bird presented for re-evaluation. It had been doing well, and the owner reported that coughing after the last tracheoscopy had resolved a few days later. On physical examination, the duck's weight had not changed since the last visit and no sutures were palpable at the tracheal resection and anastomosis site. Anesthesia was induced as above, and the left caudal thoracic airsac was cannulated for anesthetic gas delivery during tracheoscopy. Tracheoscopy revealed healthy tracheal mucosa with minimal lumen reduction at the level of the previous anastomosis. No suture material was visible within the tracheal lumen (Fig 7). The bird recovered from anesthesia without complications and was again treated with trimethoprim-sulfa and meloxicam at previous dosages. The air sac cannula was removed the next day, and the duck was discharged.

Discussion

Reported causes of tracheal luminal narrowing in birds include dynamic tracheal collapse,[2] membranic stricture,[3,4] inflammatory granuloma,[5,6] foreign body,[7,8] and fungal granuloma.[9] Neoplasia has been reported in reptiles as a cause of tracheal stenosis.[10] In this case, tracheal stenosis was believed to be secondary to previously fractured cartilages that healed in malalignment causing static tracheal collapse. This condition has not previously been reported in birds. The term "tracheal collapse" in birds differs from that used with dogs and should not be confused. Birds have complete tracheal rings, which are relatively more resistant to compressive collapse than the C-shaped tracheal cartilages of mammals.[11]

Tracheal epithelium responds immediately to irritation or disease by increasing mucus secretion. If the insult is discontinued, cells desquamate and goblet cell hyperplasia occurs to increase the protective mucous layer.[12] Superficial defects created when ciliated and goblet cells slough begin to heal as early as 2 hours after injury.[13] Intact ciliated columnar cells surrounding the defect begin to flatten, lose their cilia, and migrate over the wound. Mitosis begins approximately 48 hours after the injury in both ciliated columnar and basal epithelial cells. Within 72 hours, the epithelial layer will double to look like transitional epithelium. Organization and differentiation of cells begin after 96 hours; transitional epithelium is transformed into a layer of low cuboidal cells, which then differentiate into ciliated and goblet cells. With insufficient time for healing between insults in mammals, both ciliated and goblet cells are replaced by squamous cells.[12] Full-thickness tracheal wounds heal in a manner similar to superficial wounds if the mucosal edges are in apposition. However, defects in the mucosa or retraction of the mucosal edges leaves a gap that is filled with granulation tissue prior to epithelization.[14] This type of healing may lead to a reduction in tracheal luminal diameter. Mucostasis, which can lead to infection, can occur if epithelium is nonciliated or in the presence of stenosis.[15]

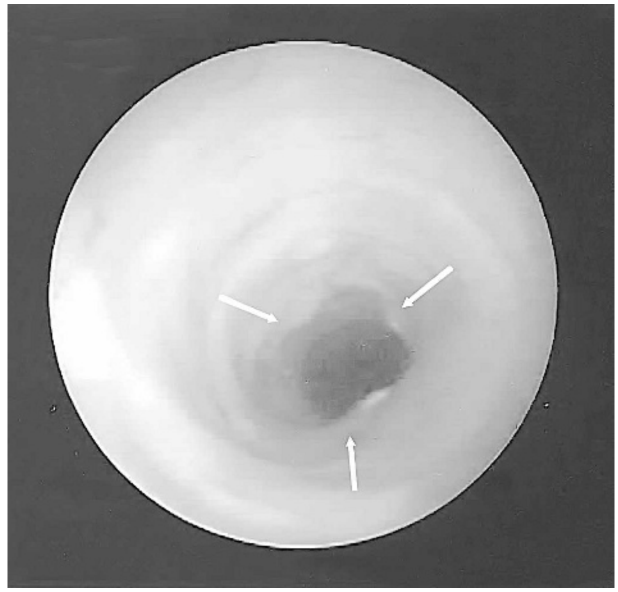

Figure 7. A tracheoscopy performed 3 months after surgery revealed a healthy mucosa, minimal reduction of the tracheal lumen in the area of anastomosis, and minimal suture granuloma formation.

Clinical signs associated with tracheal stenosis depend on the degree of airway narrowing. In active canine patients, up to 50% of the airway can be compromised without obvious clinical signs, and narrowing of 70% can occur subclinically in sedentary patients.[16] Clinical signs reported in cases of tracheal compromise include dyspnea, stridor, and mild to moderate increases in respiratory heart rate.[15] Affected individuals usually have a history of previous tracheal trauma, recent intubation, or tracheal surgery.[2,5,6,17,18] The duck in this report had a history of being attacked by a raccoon, and intermittent episodes of dyspnea accompanied by stridor had occurred during the year after the initial trauma. The clinical findings supported a preliminary diagnosis of upper airway obstruction; however, severe airsacculitis, pneumonia, or toxin inhalation could not be ruled out at the time of initial presentation.

Management of upper airway obstruction varies for the avian patient presenting in mild, moderate, or severe respiratory distress.[15] Underestimation of distress and/or overzealous examination and sampling for diagnostic testing can unduly stress the bird and adversely affect its condition. In mild cases of respiratory distress, the major goal is to keep the patient calm and avoid stress. Stabilizing these birds for 24 hours before pursuing surgical treatment is often recommended. In moderate cases, controlling the environment, keeping the patient calm, and administering supplemental oxygen may be sufficient; however, anti-inflammatory agents and tranquilizers may also be indicated. In severe cases, immediate establishment of an airway is required. In birds, air sac cannulation is described and indicated for upper airway obstruction.[19] The immediate resolution of clinical signs in this duck after air sac cannulation confirmed the diagnosis of upper airway obstruction.

Tracheal stenosis is usually identified by radiography or tracheoscopy,[18,20] although routine radiography is considered relatively insensitive in delineating stenosis.[21,22] A major benefit of tracheoscopy is that a biopsy of the stenotic tissue can be done, and samples can also be submitted for bacterial or fungal culture. Tracheoscopy also provides an opportunity to subjectively measure the tracheal lumen and is considered optimal for assessing a stenotic area.[3,23] Repeated endoscopic examination during postsurgical re-evaluation may result in mucosal trauma and impaired healing at the anastomosis site.[14] In this duck, the type of tracheal stenosis could not be determined from survey radiographs, but tracheoscopy allowed direct visualization of the tracheal lumen and stenotic area. Contrast radiography is a useful means of identifying the presence and location of a tracheal obstruction but should be used with caution.[7] No ideal contrast medium for tracheobronchography exists, but barium sulfate has been successfully used and reported in at least 1 case.[7] Computed axial tomography and magnetic resonance imaging can also be use to add information about the stenotic area and assist with making therapeutic decisions.[21]

There are different surgical and medical options for managing tracheal stenosis. In humans, the most common surgical treatments involve tracheal resection and anastomosis, endotracheal balloon dilatation, stent implantation, radiosurgery, and laser ablation.[23–28] Although less common, tracheal prothesis has also been used.[29] Several different techniques have been used in birds to manage tracheal stenosis, including tracheal resection and anastomosis,[3,4,6] endoscopic laser ablation or break down,[5,6] and placement of intraluminal stents.[2] Permanent tracheostomy has also been described in birds for treating glottal or periglottal stenosis.[30] Although other treatments were considered for this bird (eg, endotracheal balloon dilatation, stent implantation, external support, permanent tracheostomy), none were considered optimal because of the nature of the tracheal stenosis. Because the stenosis was not caused by scar tissue and the affected cartilage rings were noncompliant, resection and anastomosis were the only feasible option in this case.

Tracheal resection and anastomosis can be accomplished by using various surgical techniques, but direct end-to-end anastomosis has been the most successful method for correcting tracheal stenosis.[31,32] One of the major complications reported is postsurgical tracheal stenosis, and efforts should be made to minimize anastomotic stenosis by near-perfect apposition of the anastomosis site,[15] using minimally reactive suture material,[33] applying appropriate suture technique,[22] and reducing tension at the anastomosis site.[34] The split-ring technique, in which the trachea is resected by incising circumferentially through cartilage rings, has been used successfully in veterinary medicine and was the technique used in this case.[14] The annular ligament technique, in which the trachea is resected by incising the annular ligaments between cartilage rings, has been performed successfully in birds,[3] but normal anatomy may not be sufficiently achieved when performing anastomosis with this technique.[15] Polydioxanone (absorbable monofilament suture material) was chosen for this procedure because it elicits minimal tissue reaction and is stronger, more flexible, and absorbable compared with polypropylene (nonabsorbable monofilament suture material).[22,33,35] Polydioxanone and polypropylene have both been successfully used for avian tracheal resection and anastomosis.[3,4,6] Placing simple interrupted sutures allows more precise apposition of tracheal segments compared with results obtained by using a simple continuous suture technique.[3,6] In this bird, the sutures penetrated the mucosa and knots were tied external to the lumen. Sutures penetrating the mucosa are quickly covered by epithelium and do not consistently result in luminal stenosis when minimally reactive suture material is used.[18] Tension-relieving sutures were required in this case because of the extent of the segment resected, which represented 9% of the total tracheal length. In dogs, resection of 25%–50% of the trachea in adults and 20%–25% in puppies is tolerated.[16] No such data are available for birds, and to our knowledge this case represents the largest segment of trachea successfully removed from a bird.

Other possible complications of tracheal resection and anastomosis include injury to the recurrent nerves resulting from surgical manipulation or radical resection. In dogs, damage to the recurrent laryngeal nerve can result in laryngeal paralysis with abnormal vocalization and aspiration pneumonia.[36] The recurrent nerve does not innervate the larynx in birds but instead innervates the esophagus, crop, tracheal, and syringeal muscles; thus, damage to the recurrent nerves will not cause laryngeal paralysis.[11] The larynx in birds is innervated by the glossopharyngeal nerve.[11] The recurrent nerves of this bird were torn when the adhered sternotracheal muscles were being dissected from the abnormal tracheal cartilages. The permanent change in vocalization may have been associated with damaged innervation to the syringeal muscles. Additional complications of tracheal resection and anastomosis are dehiscence and infection.[36]

In this bird, postoperative management included antibiotic and anti-inflammatory therapy, air sac cannulation, cage rest, and careful observation for respiratory distress after the air sac cannula was removed. Reassessment of birds undergoing tracheal anastomois for tracheal stenosis by endoscopy and radiography at 1, 2, and 6 months after surgery has been suggested.[36] The bird in this case was re-evaluated by tracheoscopy 2 and 3 months after the surgery, which revealed a healthy mucosa, minimal reduction of the tracheal lumen in the area of anastomosis, and minimal suture granuloma formation.

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