Use of intraluminal nitinol stents in the treatment of tracheal collapse in a dog

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Tracheal collapse in dogs can affect the cervical and thoracic portions of the trachea and mainstem bronchi.

Surgical management of collapse of the thoracic portion of the trachea and mainstem bronchi has been difficult because of migration of intraluminal devices and the inability to apply extraluminal support.

Tracheal collapse in dogs may be treated successfully by placement of intraluminal nitinol stents.

An 8-year-old castrated male Yorkshire Terrier weighing 3.7 kg (8.1 lb) was evaluated at the University of Wisconsin Veterinary Medical Teaching Hospital (UW-VMTH) because of signs of respiratory distress. Respiratory disease was initially diagnosed in this dog at 18 months of age. Clinical signs since that time included increased respiratory noise and exercise intolerance. The dog had been treated intermittently with prednisone, aminophylline, and acepromazine. In the owners’ assessment, the disease was worsening progressively, and the dog was becoming less responsive to medical treatment. Four days prior to examination, there had been an increased level of activity and dust in the dog’s environment because of construction in the owners’ house. The dog became severely dyspneic, necessitating treatment with oxygen, butorphanol, dexamethasone, and aminophylline at an emergency clinic. Thoracic radiography revealed tracheal collapse at the thoracic inlet; the dog was referred to the UW-VMTH for further evaluation and treatment.

During the physical examination, the dog required treatment with oxygen but remained dyspneic with severe inspiratory and expiratory stridor. Bilateral lumbar-sacral alopecia was the only abnormality found on physical examination. A hemogram revealed no abnormalities. Results of serum biochemical analyses indicated mild hypernatremia (153 mmol/L; reference range, 139 to 146 mmol/L) and mild hypokalemia (3.3 mmol/L; reference range, 3.8 to 5.4 mmol/L); these abnormalities were attributed to dehydration and anorexia, respectively. Examination of inspiratory and expiratory thoracic radiographs revealed severe collapse of the trachea at the thoracic inlet (Fig 1).

A decision was made to treat the dog for tracheal collapse by implantation of an intraluminal nitinol stent. Prior to stent implantation, the dog remained in the critical care unit and received ampicillin-clavulan-

Figure 1—Lateral radiographic view of the thorax of a dog with severe inspiratory and expiratory stridor. Notice collapse of the trachea at the thoracic inlet.

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Thoracic radiography revealed that the stent had again migrated to the carina; the procedure was repeated to reposition it. To secure the stent, a 2-cm skin incision was made cranial to the thoracic inlet to expose the trachea; with endoscopic guidance, a 3-0 polypropylene suture was passed through the lateral aspect of the tracheal wall and mucosa and around the mesh wire of the stent. The dog recovered from anesthesia, and clinical signs improved for 18 hours.

Eighteen hours after placement of the stent, the dog had signs of respiratory distress. Thoracic radiographs revealed that the stent was displaced cranially by approximately 0.5 cm but was still in an acceptable position. The cause of respiratory distress was not apparent at this time, but medical treatments were continued under the assumption that irritation from placement of the stent was a contributing factor. Thirty-six hours after stent placement, the dog had signs of severe respiratory distress, and administration of sedatives and oxygen was no longer effective. Fluoroscopy performed at that time revealed that the stent had not migrated from its last known position; however, the portion of the trachea cranial to the stent had collapsed. In a second implantation procedure, a 4 × 1.5-cm, noncovered nitinol stent was secured in the trachea immediately cranial to the first. The dog recovered from anesthesia, and signs of respiratory distress were not noticeable until the dog became excited during the next morning. Eighteen hours after the second placement procedure, a separation of approximately 2.5 cm between stents was detected fluoroscopically, and the intervening portion of the trachea had collapsed. We could not remove the stents without damaging the tracheal mucosa and decided to implant a third stent to span the gap created by their separation. A polyurethane-covered nitinol stent, measuring 6 × 2 cm, was selected; this stent was placed inside the previous 2 stents (Fig 4), and because of its larger width, it was firmly anchored. The stent occupied a region of the trachea approximately 0.5 cm caudal to the larynx to 1 cm cranial to the carina. This stent was not sutured in place, because it overlapped the other 2 stents and there was no space in which it could migrate. This stent appeared to be effective; after recovery from anesthesia, the dog had diminished signs of respiratory distress.

Twelve hours after the third stent placement, the dog had a good appetite and was considerably less dyspnic. The dog coughed occasionally but did not require sedation or oxygen treatment. Thirty-six hours after placement of the final stent, the dog had a high temperature and expectoration of a small amount of blood. Radiographs were obtained; there was no evidence of stent migration. Treatment with enrofloxacin (6.1 mg/kg [2.8 mg/lb], PO, q 24 h) was initiated, and administration of amoxicillin-clavulanic acid and hydrocortone bitartrate was continued. Because the dog was much improved clinically despite the fever, it was discharged from the UW-VMTH later that day.

Nine days later, the dog was re-evaluated because of productive coughing. Thoracic radiography revealed that the stents remained in place and confirmed the diagnosis of pneumonia in the right cranial lung lobe. Administration of hydrocortone bitartrate was discontinued. The dog continued to receive amoxicillin-clavulanic acid and enrofloxacin; treatment with butorphanol (0.34 mg/kg [0.15 mg/lb], PO, q 6 to 12 h as needed) and guaifenesin (1 mL, PO, q 6 to 8 h as needed) was initiated. After 1 week, coughing and mucus production had decreased in frequency and severity.

Figure 2—Bronchoscopic view of the trachea of a dog after placement of an intraluminal stent at the area of collapse.

Figure 3—Lateral radiographic view of the thorax after placement of a stent in a dog with tracheal collapse.

Figure 4—Lateral radiographic view of the thorax after placement of 3 stents in a dog with tracheal collapse.
Four weeks after stent placement, the dog was able to exercise normally, appetite had returned to normal, and coughing occurred only in the morning after waking. Administration of antimicrobials was discontinued, and butorphanol and guaifenesin were given as needed during the following month.

Ten weeks after stent placement, the dog developed pyrexia and a productive cough. The referring veterinarian repeated treatment with amoxicillin-clavulanic acid and enrofloxacin at the same dosages as given previously. The dog responded well and appeared free of signs after 2 weeks of treatment. Ten months after the implantation procedures, the dog was receiving no medication, had no coughing when active, and had improved tolerance of exercise.

Tracheal collapse is a common problem in dogs and has also been reported in other species.1,2 Tracheal collapse is typically observed in middle-aged and older small-breed dogs, although it has been reported in large-breed dogs.3 It is a structural, obstructive airway disease with a dynamic component that can affect the intra- and extrathoracic trachea and mainstem bronchi.4

In the collapsed portion of the trachea, the cartilage- noes are deficient in glycoprotein and glycosaminoglycan components that contribute to structural rigidity and resilience; the defective rings may also be hypocellular.5 Collapse occurs most often within the cervical trachea and at the thoracic inlet, although it can occur at the carina and mainstem bronchi.6,8,9

Medical management of tracheal collapse in dogs is usually attempted prior to surgical intervention.10,11 When medical management is unsuccessful or clinical signs are severe, surgical correction of the collapse is attempted. Many surgical techniques have been described for tracheal collapse, including tracheal ring chondrotomy, plication of the dorsal tracheal membrane, tracheal resection and anastomosis, placement of intraluminal stents, and placement of extraluminal devices.12-19

Improvement of clinical signs associated with tracheal collapse has been achieved with each of these procedures, but complications may develop. Dorsal membrane plication is only useful if the tracheal cartilages are normal; the procedure may be unsuccessful if the dorsal membrane becomes stretched after surgery.13 Chondrotomy may predispose to lateral collapse of the trachea instead of dorsoventral collapse, which is commonly observed.14-16 Placement of intraluminal stents has been associated with stent migration, abscess formation, granuloma formation, tracheitis, chronic coughing, and expectoration of the stent.17-19 In general, intraluminal stents provide short-term relief, and success rates may differ slightly among types of stent.17-19 Extraluminal prosthetic devices comprising rings or spirals provide external support to collapsed portions of the trachea; placement of these devices has been associated with complications such as infection, disruption of the innervation or blood supply to the trachea causing laryngeal paralysis, necrosis of the trachea, and chronic coughing.20-22 Furthermore, application of extraluminal devices is restricted to the extrathoracic trachea and limited portions of the intrathoracic trachea.23-25 In contrast, intraluminal stents can feasibly be used to treat regions of collapse throughout the length of the trachea and mainstem bronchi.

Several intraluminal stents have been developed specifically for airway collapse in humans with tracheal or bronchial obstruction due to neoplasia or stenosis of the trachea or bronchus as a result of trauma, prolonged tracheal intubation, burns, or tumor ingrowth or resection.26-28 These implants have been used with success, although stent migration, erosion of the tracheal or bronchial wall, stent collapse, and increased secretion accumulation have been reported.29 Some of these complications arise because of the properties of the stent material. The material must be sufficiently pliable to conform to the trachea to prevent migration. However, if the strength of the material is not adequate, collapse will recur. In humans, there is no disease equivalent to tracheal collapse in dogs; nevertheless, stents that are presently available for use in humans should provide stability to the tracheal cartilages and increase luminal diameter in dogs also. A self-expanding stent made of nitinol has recently been used with minimal complications in humans.1 Nitinol is a flexible metal with properties that resemble those of the tracheal cartilages30; because of these physical similarities, the stent is more likely to conform to the trachea, and complications of wall erosion and stent migration are decreased. Results of studies27 to evaluate the use of the intraluminal nitinol stent for treatment of airway stenosis subsequent to tracheobronchial cancer in humans appear promising; there was no stent migration or erosion through the wall of the airway, no excessive secretion accumulation, and an immediate improvement in quality of life for all patients after placement of the stent. Nitinol stents are available in a variety of sizes for use in trachea and bronchi of different lumen diameter. For these reasons, an intraluminal nitinol stent may be a treatment option for tracheal or bronchial collapse in dogs.

To the authors’ knowledge, a nitinol stent has not been used previously in the treatment of a dog with tracheal collapse. Our experience with the dog of this report suggests that several factors should be considered for successful application of these stents. After the placement of the first stent, it became evident that most of the trachea had collapsed; the implant probably caused the area of greatest stress to shift to a weaker portion of the trachea. Placement of a second stent might have been effective if migration had not occurred. Prevention of stent migration may be difficult to achieve; in the dog of this report, positional changes occurred despite the use of sutures to anchor the implants in position. A contributing factor to stent migration may be size selection. In dogs with tracheal collapse, a redundant dorsal membrane may allow excessive distention of the trachea upon deployment of the stent and prevent the stent from obtaining adequate purchase to the tracheal mucosa. In future applications, it may be prudent to select a stent that is 1.25 to 1.5 times the diameter of the trachea as estimated from a lateral thoracic radiograph or via computed tomography (CT). However, it is possible that exces-
sive distention of the trachea by placement of an excessively large stent may cause pressure necrosis, leading to erosion of the trachea, pneumomediastinum, and pneumothorax.

In the dog of this report, the first 2 stents used were uncovered, which may promote ingrowth of the tracheal mucosa. The third stent was polyurethane-covered; this type of stent is typically used in human tracheobronchial disease to provide a barrier for tumor ingrowth. Because a third uncovered stent was not available on short notice and the condition of the dog necessitated intervention, a covered stent was considered acceptable. However, implantation of an uncovered stent would have been preferable.

The third stent was almost equivalent to the length of the dog’s trachea and larger in diameter than the other stents. In retrospect, instead of 3 implants, it would have been preferable to place 1 long, uncovered stent with a slightly larger diameter. The series of events that led to the placement of 3 stents highlights an inherent difficulty in estimating the appropriate size and length of the stent. As recommended by the manufacturer, the stent used initially was slightly longer than the area of tracheal collapse, yet it appeared that forces were redirected to a nonstented section of trachea with its subsequent collapse and signs of continued respiratory distress. In this dog, we were able to place 3 stents that together spanned the length of the trachea and appeared to be welltolerated. In our opinion, a better strategy for stent selection may be to adjust length estimations to provide coverage of much of the ostensibly normal trachea.

Determining size of the diameter of the stent would be most accurate via CT. In human medicine, CT and bronchoscopy are used to determine diameter and length of the tracheal lesion. However, in the dog of this report, such an approach was problematic since there was grave concern regarding the ability of the dog to recover from anesthesia required for CT or bronchoscopy, and the size of stents required were not available for immediate placement. Determination of appropriate stent diameter by bronchoscopy requires specialized bronchoscopes and computer programs to calculate relevant tracheal dimensions. This equipment was not readily available to us; therefore, stent size was estimated by use of tracheal measurements obtained from inspiratory and expiratory thoracic radiographs. Measurements obtained from radiographic images may result in underestimation of the normal diameter of the collapsed trachea. Thus, an estimate of the diameter of the trachea should be made as accurately as possible; selection of a stent that has a diameter 3 to 5 mm larger than the estimated tracheal diameter to accommodate stretching of the dorsal tracheal membrane may be beneficial. Also, we recommend the use of a stent that is 2 to 3 cm longer than the length of collapsed trachea observed radiographically. Furthermore, preoperative fluoroscopy may be helpful in determination of the extent of airway collapse.

Pneumonia is a potential problem associated with stent placement. This may develop because of impaired clearance of respiratory secretions or secondary to inflammation induced by the stent. In the dog of this report, the radiographic appearance of the lungs after surgery was consistent with aspiration pneumonia that could have developed as a result of multiple anesthetic episodes. Tracheitis is to be expected until the stent has been incorporated into the tracheal wall mucosa. Placement of 3 stents or the use of a covered stent in this dog likely impeded the mucociliary function of the trachea and promoted tracheal irritation and perhaps development of pneumonia. This alone may have caused the dog to cough more than usual; however, the problem was resolved after administration of antimicrobials.

The results of stent placement in this dog have been favorable for 10 months. However, the long-term prognosis is improved if the stent becomes enveloped by the tracheal mucosa. Endoscopic evaluation of the stents to assess ingrowth of the tracheal mucosa would be of value; mucosal growth over the 3 stents would suggest that the mucociliary apparatus would be affected only temporarily, and the risk of pneumonia would not be continued throughout life. However, the owner was reluctant to pursue any further examinations because the dog was much improved; therefore, the extent of mucosal ingrowth is not known. Our experience suggests that intraluminal nitinol stents may provide long-term relief from dyspnea caused by tracheal collapse, although further placements are needed to determine the success rate of this procedure.

References