TRACHEAL COLLAPSE SYNDROME:  
WHAT’S NEW IN MANAGING THIS DIFFICULT DISEASE

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Intraluminal stenting is a palliative, minimally invasive therapy used for restoration of an obstructed or narrowed lumen. Interventional radiologists have employed these techniques in virtually every accessible lumen of the human body and, if recent history is any indication, it appears it is now the veterinarians’ chance to do the same in our animals. Following even a preliminary review of the human interventional radiology (IR) literature, it does not take a tremendous amount of imagination to conclude our patients can likely benefit from similar procedures as there is such abundant overlap of the disease processes. The respiratory system has the potential to be particularly well suited to these types of interventions as these patients are often severely affected and fragile, comorbidities are not uncommon, and the risk of associated surgical complications from traditional therapies can be relatively high.

Veterinary IR has evolved to encompass many different body systems, each with their own successes and failures. However, even with the advancements in intraluminal tracheal stenting devices and technical modifications, a number of these patients continue to be plagued by complications including intractable coughing, infection, tissue ingrowth, stent fracture, and others. Although these procedures are still not providing the ideal outcomes we might hope for at this time, veterinarians performing this procedure with even some regularity will generally agree that dramatic improvements can be achieved in certain cases that have failed aggressive medical management and surely would have not tolerated a surgical intervention. Clearly there is a role for tracheal stenting in our veterinary patients, and this may expand to bronchial stenting for similar collapse/compression of these structures in certain circumstances.

Stents, Technique, and Outcomes
Over the last decade, tracheal stents have become increasingly available to veterinarians, and their design and delivery systems have evolved accordingly. We have come a long way from the originally described metallic balloon-expandable stents to shape-memory self-expanding metal (and even tapered) stents. Unfortunately, currently manufactured metallic stents remain a long way away from mimicking the complexity of the living trachea. Our human medicine colleagues have described complications including “obstructive granulation tissue, stenosis at the ends of the stent, migration of the stent, mucus plugging, infection, and stent fracture”\(^1\); very similar to some of the major complications identified in our veterinary patients. However, we must interpret this information with an understanding of the different indications and circumstances in humans compared with our patients. Much of the tracheomalacia (TM) treated in humans is temporary so removable stents (silicone) are preferred. In addition, even for permanent indications, stent exchanges and “toilet bronchoscopy” procedures to suction, remove, and reculture infectious mucus accumulations are not uncommon in people with silicone stents; perhaps a limiting factor for routine use silicone stents in our patients due to cost and owner compliance.

Fortunately, it seems self-expanding metallic tracheal stents have better outcomes in dogs compared to those routinely reported in humans for benign diseases. Our problem has been determining which of the canine patients are mostly likely to respond favorably to this treatment, as the current outcomes are widely variable and difficult to predict. Perhaps the most important advancements in my own stenting experiences have been in recognizing the variability of the “tracheal collapse syndrome” (weakened dorsal membranes versus tracheal malformations\(^2\), for instance) and in refining our stent placement techniques. The technical advancements include improved medical management and recognition of comorbidities, appropriate fluoroscopic technique and tracheal measurements, proper stent diameter oversizing and preventing overlong stents, pre- and post-stent tracheoscopy, and vigilant client
communication with regular scheduled follow-up examinations.

Our approach to traditional flattened tracheal lumens (flaccid dorsal membranes) is different from the “W-shaped cartilage rings seen in tracheal malformation cases. More involved upper airway examinations identifying comorbidities such as overlong soft palates or epiglottic retroversion have led to concurrent airway surgeries performed more routinely in our patients. Performing careful patient positioning and both positive- and negative-pressure ventilation images with esophageal marker catheter placement confirms precise collapse location and minimizes error in radiographic magnification, one of the most common mistakes made during the stenting procedure. Pre- and post-stent tracheoscopy confirms the variety of collapse as well as appropriate stent-to-tracheal wall contact and positioning during the procedure so inappropriately positioned stents can be removed or repositioned while it is still possible.

Although tracheal stenting is one of the first IR techniques veterinarians attempt, it is a demanding technical procedure with little room for error, and advanced training is highly recommended to avoid some of the more common mistakes.

Complications
Fortunately there are some complications we can control; Those include mistakes in case selection, stent choice and technical decisions, inappropriate monitoring, late detection of complications, and poorly executed medical management. Unfortunately there remain some complications we currently cannot easily control; These include granulation or inflammatory tissue ingrowth, infection, stent fatigue and fracture, and disease progression. As stent technology and designs improve in the future, some of these problems may become less of a concern, however in the meantime, appropriate client counseling, early detection, and aggressive medical management or intervention may reduce the consequences of these complications.

Tissue ingrowth into tracheal stents has been reported in both human and veterinary patients. In animals, any growth within the stent was initially considered “granulation tissue” due to human reports and appearance. More recently, we and others have appreciated tissue regression following high dose corticosteroid administration. We know granulation tissue does not regress with corticosteroids leading us to conclude this is often inflammatory tissue/granulomas rather than granulation tissue. This tissue typically develops at the cranial aspect of the stent but can develop elsewhere. Over the years we have recognized this tissue can respond to high dose steroids when administered early, however when treatment is delayed responses are more uncommon. Rather than granulation tissue “versus” inflammatory tissue developing independently, it appears that the longer inflammatory tissue is present without treatment the more this tissue will become organized and infiltrated with fibrous scar tissue; This what a number of our biopsies have suggested (currently unpublished data). This highlights the importance of regular monitoring and early detection of tissue ingrowth. Further we have recognized that tissue growth adjacent to or within the stent can often be unrecognized due to forelimb positioning in a lateral radiographic projection. For this reason we have instituted mandatory 3-view chest and neck radiographs for all tracheal stent cases with the forelimbs pulled cranially in one lateral and caudally in another. This position permits complete evaluation of the cervical and intrathoracic trachea without interference of the forelimb soft tissue and boney structures.

Infection has been considered a major component of the tracheal collapse syndrome but once a tracheal stent has been placed, it is clear that the trachea becomes susceptible to infections. This is exacerbated when there are persistent gaps between the stent and tracheal wall, permitting mucus accumulation, most commonly seen in the tracheal malformation cases we recently reported. Infections alone can be problematic, however the greater concern may perhaps be the increased risk of tissue ingrowth that occurs when tracheal stents become infected. This is well documented in humans with tracheal stents. It had been unclear whether the tissue ingrowth led to infection or the infection led to tissue ingrowth. More recent data suggests that both infection and stent micromotion independently contribute to the development of tissue ingrowth into the stent. Over the years we have been more aggressive about early intervention of cases with persistent clinical signs; early tracheoscopy and culture permit rapid, appropriate antibiotic therapy. Our impression has been diffuse stent infiltration with tissue is most often associated with infection and appropriate antibiotic therapy can often result in regression.
interrogate the problem can lead to inappropriate antibiotic selection, tissue progression and organization/fibrosis, and ultimately require additional stent placement during respiratory crises.

Stent fracture will continue to remain a concern when metallic stents are used. There is no perfect stent; the unforgiving environment and extreme forces encountered are more than we can currently expect a stent to sustain for the many years we are anticipating it to be in place. Super-elastic titanium alloys, special coatings, improved designs, and other technological advances will continue to improve the longevity of these stents. Currently, the most important aspect of tracheal stent fracture in our practice is early detection and restenting prior to patient decompensation. Good outcomes can still be achieved when stent fractures are identified and restented quickly.

Although previous literature cited stent migration and substantial stent shortening as common complications, our experience has not supported these findings (this information is being presented at this conference). When appropriately sized diameter stents are used, migration is almost negligible (as demonstrated by changing distance from stent to cricoid and/or carina) and our stent shortening rates are approximately 11% over the long-term compared to the approximately 30% previously reported. The stent type and diameter used and/or the placement technique likely explain these differences.

Considerations on Bronchomalacia and A Theory of “Bronchial Compression”

Bronchial stenting may be the natural progression of veterinary respiratory endoluminal therapy. The presence of left mainstem bronchomalacia and bronchial collapse has been well reported in the veterinary literature, commonly associated with left atrial enlargement. It has also been reported as an isolated, incidental finding and in dogs with tracheal collapse with or without concurrent heart disease. Interestingly, brachycephalic breeds have also been shown to have unilateral bronchial collapse (84% left-sided) as well – even without the presence of left atrial enlargement. This has been explained due to chronic abnormal airway pressures leading to progressive weakening and subsequent collapse of the left mainstem bronchus. The unilateral nature remains unclear but theories have included different airway pressures encountered along this longer left bronchial segment when compared with the relatively shorter right mainstem bronchus. This left mainstem bronchomalacia has even been cited as a possible cause for left cranial lung lobe torsions in Pug dogs due to the weakened cartilage integrity. The wide variety of dogs experiencing mostly left bronchial collapse, with or without heart disease, with or without upper airway collapse, and with or without clinical signs suggest a variety of possible explanations, likely important to understand more clearly if we hope to predict outcomes following invasive, or even noninvasive, interventions.

Comparatively, bronchomalacia in humans is typically bilateral and when unilateral, the left and right sides seem generally equally affected for the most part. Anatomical differences between humans, animals, and even different shaped animals (narrow, deep-chested versus barrel-chested breeds) inspired a more detailed investigation into the thoracic anatomy of different breeds. Some examples of this investigation are included in Figure 1 demonstrating the possible “compressive, sandwiching” nature of the left mainstem bronchus between the left atrium and aorta, particularly apparent in barrel-chested dogs (not uncommon conformations of brachycephalic breeds and some of the at-risk collapsing trachea breeds including barrel-chested Yorkshire terriers). A static compression of the bronchus could lead to cartilaginous weakness and further collapse over time, progressing from “static” to “dynamic” compression. This phenomenon could explain why some bronchoscopically-identified bronchial compression is “static” or “fixed” (as might be identified with compression between two structures) versus “dynamic” (as might be expected with malacia of the cartilage during dynamic airway pressures). In fact the bronchial collapse described in the brachycephalic breeds was described as “fixed” in 87.5% of the dogs. The location of the pulmonary arteries may also contribute to airway compression and could even help explain the narrow pedicle of the left cranial lung lobe in Pug dogs and possible predilections to bronchomalacia, regional atelectasis, torsion, or combination. The more typical compression of the left mainstem bronchus may even be another possible explanation for the more commonly located right-sided (46%) bronchial versus left-sided (29%) bronchial airway foreign bodies in dogs. Figure 1 demonstrates just some of the wide variability of cardiovascular and airway structures positioned in different breeds, as well as individual dogs within a breed. If “bronchial compression” is confirmed in certain breeds and/or thoracic conformations, this might help determine which patients may benefit from bronchial stenting; it may also help determine which patients might not.
This information is preliminarily under investigation at our institution but further characterization of anatomical differences between breeds, anatomical conformations of vascular and respiratory structures, and even gestational growth patterns leading to cartilage weakness and future collapse could provide useful information for our patients in the future. Much work needs to be done to determine which patients may benefit from bronchial stenting. Personally I feel there will be a subset of patients that will benefit and others that will not; How we will categorize these patients remains to be determined and will likely take the work of many researchers around the world for years to come.

**FIGURE 1:**

![Figure 1](image1.png)

Figure 1: Serial, static, axial thoracic CT images in 4 dogs without cardiac or pulmonary disease. Note the variable thoracic width (red line) and depth (blue line) conformations and corresponding cardiovascular structure locations (A-Aorta, RB-right bronchus, LB-left bronchus, LPA, left pulmonary artery). A. Deep-chested Boxer with considerable space between thoracic vertebral body, aorta, and left bronchus. B. Intermediate barrel-chest conformation Pug with left bronchus compressed between aorta and heart. Note the lack of available space between the thoracic vertebral body, extremely dorsally located aorta, and the left bronchus. The right bronchus is free of compression from any cardiovascular structures. C. Intermediate barrel-chest conformation Yorkshire terrier with left bronchial compression between aorta and heart. The right bronchus is free of compression from surrounding structures. D. Severely dorsoventral flattened thoracic conformation in a French Bulldog with a retrobulbar sarcoma and no respiratory signs having thoracic CT for metastasis screening. Note considerable left bronchus compression between aorta and the left-sided deviated heart with additional compression from the left pulmonary artery.

**REFERENCES**

1. FDA medical safety warning: “Metallic Tracheal Stents in Patients with Benign Airway Disorders” (2005); [http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/PublicHealthNotific ations/ucm062115.htm](http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/PublicHealthNotific ations/ucm062115.htm)