# “Taz’ Traumatic Time”

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## Introduction

Tracheal collapse is a progressive, irreversible disease mainly occurring in middle aged, toy to small breed dogs, with Pomeranians, miniature and toy poodles, Yorkshire terriers, Chihuahuas and pugs most commonly being affected.[1-13] This condition causes narrowing and dorsoventral flattening of the tracheal lumen which leads to coughing and airway obstruction in dogs.[1-13] A lateral collapse can also occur but is uncommon.[4] The collapsing trachea usually involves the cervical region, but often both the cervical and thoracic areas of the trachea are involved, and the collapse may extend into the bronchi leading to bronchomalacia[4]. The trachea is composed of 35-45 cartilaginous C-shaped rings joined dorsally by the trachealis muscle, mucosa and connective tissue.[2,5] The tracheas' primary function is the conduction of gas; however, the trachea also has an important role in trapping and transporting impacted debris up the mucociliary apparatus which will be discussed further.[5]

## History & Presentation

Taz is an approximately 9-year-old male neutered Pomeranian mixed breed dog who presented to Mississippi State University College of Veterinary Medicine Internal Medicine Service on 6/2/2021 for a collapsing trachea. Taz was previously diagnosed with a grade III collapsing trachea in 2017 by bronchoscopy. At that time the bronchoalveolar lavage had no fungal or bacterial growth. Since then, Taz' chronic cough has continued, especially when stressed or exciting factors are involved, leading to disease progression. Taz was taken to his primary veterinarian on 05/24/2021 where he was administered Hydromorphone and Temaril-P. Taz's owner noticed some improvement on these medications but still believed that Taz' disease was continuing to progress. Taz was then referred to Mississippi State for evaluation of his tracheal collapse.

On presentation to MSU, Taz was bright, alert and responsive. He weighed 4.7 kilograms with a body condition score of 7/9. He had a heart rate of 124 beats per minute with strong and synchronous pulses, a respiration rate of panting and a temperature of 102.3ºF. Upon cardiopulmonary auscultation, no crackles, wheezes, murmurs or arrythmias were heard. He had significant upper airway noise along with a goose-honking cough that was easily elicited on tracheal palpation. His mucous membranes were pink and moist with a capillary refill time of less than 2 seconds. His eyes, ears and nose were clear and free of discharge. His abdomen was soft and non-painful on palpation. All palpable lymph nodes were small and symmetrical. Taz appeared mentally appropriate.

**Pathophysiology**

The cause of tracheal collapse is poorly understood but is likely multifactorial. The clinical disease initially results from weakening of the tracheal rings, which can be congenital or acquired. In collapsed tracheal cartilage, there are decreased amounts of glycosaminoglycans and glycoproteins which bind water, resulting in a decreased rigidity of the tracheal rings.[5,10,11] Diseased cartilage also contains less chondroitin sulfate and calcium which lead to the replacement of normal hyaline cartilage with collagen and fibrocartilage, subsequently weaking the tracheal rings leading to a collapsing trachea.[10] These structural changes within the cartilage matrix, alongside the reduced water content, in time, lead to reduced functional rigidity, a form of chondromalacia, stretching of the dorsal membrane and finally collapse of the trachea.[4,5,10,11] In some dogs, these changes and defects of the cartilage extend into the mainstem bronchi and more distal cartilaginous bronchi resulting in bronchomalacia.[5]

Many dogs will remain asymptomatic until later in life when secondary factors are thought to trigger the clinical syndrome.[11] Airway irritants, respiratory tract infections, obesity,

pollutants, environmental allergens, chronic bronchitis, laryngeal paralysis, cardiac disease, recent endotracheal intubation, hyperadrenocorticism and cervical trauma may be associated with disease progression.[2,4,10,11] Once symptomatic, dynamic collapse of the airway perpetuates a cycle of chronic inflammatory change within the tracheal mucosa, which is worsened by the coughing this causes.[11] Ongoing tracheal mucosal inflammation has been associated with epithelial squamous metaplasia, leading to loss of normal ciliary clearance.[11] This mucosal change and hyperplasia of the subepithelial gland, which secrete increasingly viscid mucus, causes coughing to become the major tracheobronchial clearing mechanism.[11] The coughing then results in more inflammation creating a vicious, perpetuating cycle.

**Diagnostic Approach/Considerations**

History, signalment, physical exam and clinical signs may be strongly suggestive of tracheal collapse. However, a thorough diagnostic evaluation should be completed to rule out other causes of airway disease, and to help localize and determine the severity of the tracheal collapse. Primary differential diagnoses for tracheal collapse include but are not limited to, infectious tracheobronchitis, tracheal or laryngeal obstruction, chronic bronchitis or bronchiectasis, pneumonia and congestive heart failure.[5] The list of differentials for a collapsed trachea is extensive therefore a full workup including bloodwork, electrocardiography, diagnostic imaging, laryngeal and endoscopic evaluation including collection of microbiologic and cytologic samples should be performed to help rule out potential causes and evaluate for underlying or concurrent diseases.[10]

On clinical examination the animal's respiratory pattern may help localize the area of collapse, but should be confirmed with radiographs, fluoroscopy and/or bronchoscopy. For example, increased inspiratory effort is usually associated with extrathoracic tracheal collapse or

cervical disease because of the decreased pressure within the trachea.[10,11] Whereas collapse of the intrathoracic trachea and bronchomalacia is associated with increased expiratory effort or coughing because of increased intrathoracic pressure.[10,11]

Radiographs may be limited in confirming and determining the extent of tracheal collapse, correctly identifying the disease in only 59% to 84% of patients.[5,10] However, radiographs are critical to rule out conditions that may cause similar signs such as intrathoracic masses, pleural effusion, cardiovascular abnormalities and pulmonary disorders.[2] Radiographs underestimate the severity and degree of collapse in comparison to fluoroscopy.[6] The most useful radiographic image is a lateral radiograph of the entire trachea taken during maximum inspiratory and expiratory phases.[10] A significant feature of patients with tracheal collapse includes frequent association of hepatomegaly, however, the relationship of hepatomegaly to the clinical syndrome is unclear.[4]

Fluoroscopy may be necessary to determine the full extent of the disease. Fluoroscopy offers superior evaluation of airway collapse, allowing the dynamic, real-time evaluation of the trachea and mainstem bronchi during all phases of respiration as well as during episodes of coughing.[10,11] The sensitivity of fluoroscopy in detecting airway collapse is enhanced if the patient can be induced to cough during the evaluation by applying pressure to the trachea.[9] Fluoroscopy is more accurate in documenting the location of tracheal collapse compared to radiographs, with one study documenting collapse in an incorrect location in 44% of dogs and tracheal collapse not being detected at all in 8% of dogs via radiography.[7]

Endoscopic evaluation of the airway is considered “gold standard” for diagnosing tracheal collapse and is an important step in both the diagnosis and grading of tracheal collapse. It allows full evaluation of the structure and integrity of the trachea and bronchi.[10] It also allows

for a thorough laryngeal examination during induction and permits sample collection for microbiologic and cytologic evaluation which is especially important in patients with evidence of lower airway disease.[10] Collapse can be graded during bronchoscopy from I to IV, characterized by 25%, 50%, 75% and 100% collapse, respectively.[2,11] Disadvantages to bronchoscopy include limited availability, cost and need for general anesthesia.[2] Dogs with airway collapse can have a difficult recovery from anesthesia as a result of complications, such as increased respiratory effort, that lead to further airway irritation or collapse.[7] When a patient with tracheal collapse recovers from anesthesia there is always the potential for respiratory complications and respiratory distress.

**Treatment and Management Options**

Animals with tracheal collapse often present in respiratory distress and must be treated as a medical emergency with oxygen supplementation, and mild sedation to help relax the patient and improve ventilation.[10] Due to their unstable state, unnecessary diagnostics should be postponed and stress minimized until the patient is breathing comfortably.[10] Most dogs with tracheal collapse are medically managed and respond well to medical therapy, with surgical interventions reserved for those refractory to treatment or with severe clinical signs, such as cyanosis, exercise intolerance or dyspnea.[11] The goal of medical management is to break the vicious cycle in which inflammation triggers coughing which then continues to worsens the inflammation. Studies have shown that between 71-93% of dogs respond well to medical management for a period of more than 12 months, with 50% able to have medication gradually withdrawn.[11]

Medical management typically includes antitussive therapy, steroids, bronchodilators and antimicrobials if there is evidence of a concurrent secondary infection based on culture and

sensitivity. If antimicrobials are indicated, agents with efficacy against *Mycoplasma*, such as doxycycline should be considered pending culture results from bronchoalveolar lavage.[11] Antitussive therapy, such as hydrocodone, butorphanol, codeine, and diphenoxylate has been the mainstay of medical management for dogs with tracheal collapse. Antitussives, specifically, diphenoxylate, act to reduce the volume of mucus secreted into the lower respiratory tract and also acts as a muscarinic bronchodilator.[11] The dosages of the antitussives may need to be changed over time to find the most beneficial dose.

The use of short-term steroids can be beneficial through the reduction of airway inflammation. The lowest dose possible to control clinical signs should be used due to adverse effects such as increasing the risk of bacterial infection, increasing respiratory rate and may make weight loss difficult.[11] If antitussives alone are not enough to control the clinical signs, a bronchodilator such as theophylline or aminophylline may be added to the treatment regimen, however, this is controversial. The rationale for their use is based on the dilatory effects of the pulmonary airways, which decrease intrathoracic pressure and the tendency for tracheal narrowing during expiration, but they do not have a direct effect on the diameter of the trachea[10] They may also be beneficial by improving mucociliary clearance and reducing diagram fatigue, however, the benefit of bronchodilators in dogs with tracheal collapse has not yet fully been evaluated.[11] For this reason, if the patient does not demonstrate any improvement during the administration, the bronchodilators should be withdrawn. $\beta_2$-adrenergic agonists such as albuterol or terbutaline can also be used as a bronchodilator but mainly in emergency situations.

A dietary weight loss regimen involving a high fiber, low fat diet is essential in obese patients with tracheal collapse. Obesity exacerbates clinical signs due to effects on the cardiopulmonary system causing decreased lung expansion and increased work of breathing.[5,10]

Weight loss alone not only causes a dramatic improvement in clinical signs but has been curative in terms of dealing with the symptoms of this disease.[4] Other adjunctive strategies should be used to help manage patients with tracheal collapse. Exercise restriction is important, especially in hot or humid weather. The use of a body harness instead of a collar will eliminate external pressure applied to the trachea and is recommended to decrease coughing and tracheal irritation. Removal of other respiratory irritants from the patient's environment, such as cigarette smoke is also recommended.

Surgical interventions are reserved for cases refractory to medical management or with severe clinical signs.[11] The aim of surgical intervention is to improve the tracheal anatomy to allow increased airflow without disrupting the mucociliary system.[11] It should be noted that no surgical intervention will cure tracheal collapse, therefore, continued medication is often needed to control coughing. Patient selection is key to obtaining good outcomes and only dogs with a severe tracheal collapse, grade II or higher should be considered for surgical management.[11] Various surgical techniques have been reported including tracheal ring chondrotomy and plication of the dorsal tracheal membrane, which has largely fallen out of favor due to tracheal narrowing, the placement of extraluminal ring prostheses and intraluminal stent placement.[11]

Extraluminal support of the trachea with a ring protheses allows restoration of the tracheal diameter during respiration and coughing and does not interfere with mucociliary function.[11] Due to high morbidity documented in association with the placement of extraluminal prosthesis around the intrathoracic trachea, the technique is usually limited to the support of the extrathoracic portion of the trachea.[11] Complications include coughing, dyspnea, laryngeal paralysis, tracheal necrosis and a tracheostomy tube may be required post-operatively.

Placement of intraluminal tracheal stents is a minimally invasive procedure compared to the placement of extraluminal ring prostheses, with rapid postoperative improvement in most cases.[11] Indications include patients with thoracic inlet and mainstem bronchial collapse or have collapse of most of the trachea.[10] Proper stent measurement is required, and guidelines should be followed to prevent obstruction of the main stem bronchi leading to mucus entrapment and complications such as infection. Studies have documented improvement in 75-90% of dogs treated with intraluminal stents. However, stent placement is not a curative procedure, it is a salvage procedure and long-term medical management may still be necessary. Complications include coughing, stent migration, positive tracheal culture, pneumonia, acute pulmonary edema, granulation tissue formation, and stent collapse or fracture.[10]

**Expected Outcome and Prognosis**

In most dogs, clinical signs can be controlled with continuous multimodal medical management. This is the mainstay of therapy with a 71-93% success rate with medical management alone and can generally alleviate clinical signs for months to years. A recent study showed that clinical signs of dogs that were medically managed showed improvement during the short term but tended to regress and worsen over time.[1] The study also showed that the median survival with medical management only was 3.7 years as compared to 5.2 years with a stent placement.[1] For more severe cases, the median survival time was only 12 days for medically managed dogs and 1,338 days for dogs that underwent stent placement.[1]

**Case Outcome**

Thoracic radiographs of Taz revealed dynamic airway collapse of the trachea at the thoracic inlet and intrathoracic trachea. Hepatomegaly was also appreciated. The pulmonary

lobar arteries and veins, pleural space, pulmonary parenchyma, osseous structures and remaining extracavitary soft tissues were normal ruling out other potential causes for Taz' clinical signs. To further evaluate the extent and severity of Taz' tracheal collapse, fluoroscopy was performed. Taz' fluoroscopy showed 90-100% narrowing of the trachea within the thoracic inlet and thorax, carina, and mainstem bronchi during forceful expiration. Due to the severity, failure of long-term medical management and location of Taz' tracheal collapse, Taz qualified as a good candidate for an intraluminal stent placement.

A bronchoscopy and tracheal stent measurement was recommended to be scheduled at the client's earliest convenience. Until then, Taz was discharged to be further medically managed with hydrocodone, maropitant and trazadone. Strict instructions were given to monitor Taz for any signs of respiratory distress or cyanosis. Instructions were also given to only walk Taz with a harness, no neck leads, limit exposure to any respiratory irritants, hot/humid weather, and stressful or exciting situations. A dietary weight loss program with Hill's Metabolic and Motility was also highly encouraged.

Taz returned to MSU Internal Medicine Service on 06/16/2021 for a bronchoscopy and intraluminal stent measurement. Taz was placed under general anesthesia and radiographs were obtained for tracheal stent measurement. Bronchoscopy was then started where a grade IV tracheal collapse was noted throughout the thoracic trachea. The left mainstem bronchi was also completely collapsed, classifying as a grade IV collapse. The right mainstem bronchi exhibited a dynamic grade III collapse. The lower bronchi also exhibited severe dynamic collapse with a moderate amount of mucus, erythema and edema diffusely. A bronchoalveolar lavage was performed in the right caudal lung lobe and a moderate yield was obtained.

Afterwards, Taz became hypoxic, with his SpO2 dropping into the mid-60s, which persisted despite albuterol and terbutaline. This hypoxic state indicated that Taz most likely would not wake up safely from anesthesia, therefore, it was elected to place an intratracheal stent with fluoroscopic guidance. The stent was placed with no complications and Taz recovered from anesthesia uneventfully. Taz was maintained in the oxygen cage overnight and the following day to be monitored for any signs of respiratory distress, which is normal a post-operative protocol. Because the stent initially irritates the airway, coughing is expected but must be controlled to prevent stent fracture or granulation tissue formation.[2] Taz was administered maropitant, terbutaline and butorphanol while in the oxygen cage. If coughing was observed, acepromazine was administered to help keep him calm, steady his breathing and help minimize the risk of stent fracture. Acepromazine was only administered twice throughout his entire time in the oxygen cage.

The morning of 06/18/2021, Taz was removed from the oxygen cage due to his stability and decreased expiratory push. He was started on oral medications including an increased hydrocodone dose, maropitant and trazadone. Acepromazine and butorphanol were available to be given on an as needed basis if significant coughing or respiratory distress was observed. Taz did well with no supplemental medications or oxygen. On 06/19/2021 Taz was discharge with the increased dosage of hydrocodone, maropitant and trazadone. He was also sent home with emergency acepromazine to be administered if an uncontrollable coughing episode occurred to hopefully protect the stent from fracturing or any other complications.

Taz returned to MSU Internal Medicine Service for his recheck appointment on 06/30/2021 to evaluate his stent placement. Mrs. Nunn reported that since the placement of the tracheal stent, Taz has been doing well at home. Taz has had 3 episodes of uncontrollable

coughing where acepromazine was administered which helped significantly. Thoracic radiographs were then obtained to evaluate the placement of Taz' tracheal stent. His intraluminal tracheal stent placement remains unchanged and Taz was sent home to continue the previously prescribed medications. Today, Taz is doing great at home. He is no longer on any medications and on occasion with have a single cough. Mrs. Nunn continues to minimize his exposure to hot/humid weather, respiratory irritants and stressful/exciting situations.

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