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SPECIAL INTEREST ARTICLE

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Pediatric tracheomalacia and the perioperative anesthetic management of thoracoscopic posterior tracheopexy

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Summary

Tracheomalacia is a broad term used to describe an abnormally compliant trachea that can lead to exaggerated collapse and obstruction with expiration. We describe the perioperative management of a complex pediatric patient undergoing a posterior tracheopexy which is a relatively new surgical treatment, with a novel surgical approach—thoracoscopy. This procedure has competing surgical and anesthetic needs and presents unique challenges to the physicians involved in caring for these patients. We also review the current literature on pediatric tracheomalacia and examine the newest treatment options to highlight the potential anesthetic challenges and pitfalls associated with management.

KEYWORDS

infant, newborn, 1-lung ventilation, tracheal diseases, tracheobronchomalacia, tracheomalacia/diagnosis, tracheomalacia/surgery

1 | INTRODUCTION

Tracheomalacia is a broad term used to describe an abnormally compliant trachea that can lead to exaggerated collapse and obstruction with expiration.[1-3] This usually results from impaired cartilage integrity or from external impingement caused by adjacent intrathoracic structures.[1-3] The etiology, degree of tracheal narrowing, and severity of clinical symptoms dictates the treatment.[4] A novel approach, thoracoscopic posterior tracheopexy, involves stenting the trachea by attaching the posterior trachea to the anterior longitudinal spinal ligament under guidance of fiberoptic bronchoscopy (FOB).[2,3,5] Although this minimally invasive technique is advantageous for patients with compromised respiratory mechanics, the simultaneous need for operative FOB guidance and lung isolation makes for a challenging anesthetic. We describe the management of a complex pediatric patient undergoing a novel approach to a relatively new treatment—thoracoscopic posterior tracheopexy, review the literature on pediatric tracheomalacia, and examine the newest treatment options to highlight the potential challenges and pitfalls associated with caring for these patients.

2 | CASE REPORT

A 3-year-old, 13.5 kg girl with tracheomalacia was presented for thoracoscopic posterior tracheopexy. Her medical history was notable for a chromosomal deletion syndrome (46XX del 2q37.1), cervical subluxation at C3-C4, and foramen magnum stenosis with resulting

hydrocephalus requiring a ventriculo-peritoneal shunt. She also had a history of chronic lung disease and pulmonary hypertension (likely secondary to severe untreated obstructive and central sleep apnea) that required oxygen (1 L/min oxygen via nasal cannula) and nighttime bilevel positive airway pressure (BiPAP).

She was seen multiple times by our multidisciplinary aerodigestive team for assessment and surgical planning. This highly functioning, collaborative team consists of pediatric otolaryngologists, general surgeons, gastroenterologists, pulmonologists, anesthesiologists, speech pathologists, occupational therapists, registered dieticians, and social workers.[6] As part of her evaluation process, she underwent extensive testing that included both flexible and rigid bronchoscopy, upper intestinal endoscopy, polysomnography, and computerized tomography (CT). The CT imaging demonstrated significant compression (53%) of the AP diameter of the trachea at the level of the brachiocephalic artery at end exhalation (Figure 1A and B). Flexible bronchoscopy showed severe tracheomalacia in the distal portion of the trachea with 95% dynamic collapse of the posterior aspect of the trachea with coughing (Figure 2A and B). Based on these findings, a thoracoscopic posterior tracheopexy was deemed to be an appropriate intervention.

3 | ANESTHETIC MANAGAMENT

The patient had an inhalational induction with sevoflurane, but was transitioned to a total intravenous anesthetic for maintenance of anesthesia to facilitate monitoring of somatosensory evoked potentials (SSEP) in the context of her cervical subluxation to minimize volatile anesthetic pollution of the operating room with an open airway, and to maintain a stable anesthetic depth during tracheoscopy. Intravenous medications used included propofol (initiated at 200 μg/kg/min and titrated to 100 μg/kg/min) and remifentanil (between 0.1—0.15 μg/kg/min). No neuromuscular blockade was used. The trachea was intubated with a cuffed 3.5 endotracheal tube, which was smaller than expected for her age and weight, but had been sized appropriately during a previous bronchoscopy, and downsized to provide adequate room to accommodate the extraluminal bronchial blocker without placing undue pressure on the tracheal mucosa. An extraluminal (outside the endotracheal tube) 5F Arndt endobronchial blocker (AEB) was placed into the right main stem bronchus for lung isolation and 1-lung ventilation. The blocker was inserted under direct laryngoscopy immediately followed by endotracheal intubation, and advanced using fiberoptic bronchoscopy. Apneic oxygenation via high flow nasal cannula helped maintain the patient's oxygen saturation within normal limits during intubation. An orogastric tube was also placed to aid in surgical identification of the esophagus and vagus nerve. In addition to standard ASA monitors, an arterial line and 2 peripheral intravenous lines were placed. No transcutaneous CO_2 monitoring was used.

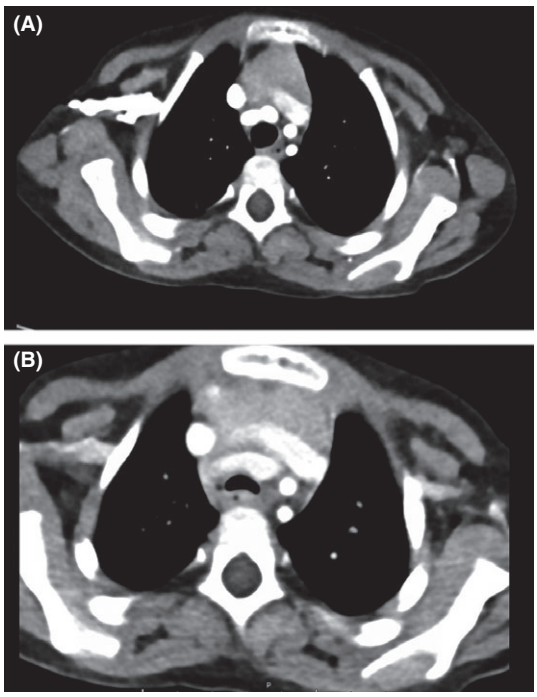

FIGURE 1 A, CT image at the level of the brachiocephalic artery during inspiration. B, CT image at the level of the brachiocephalic artery at end exhalation demonstrating approximately 53% collapse

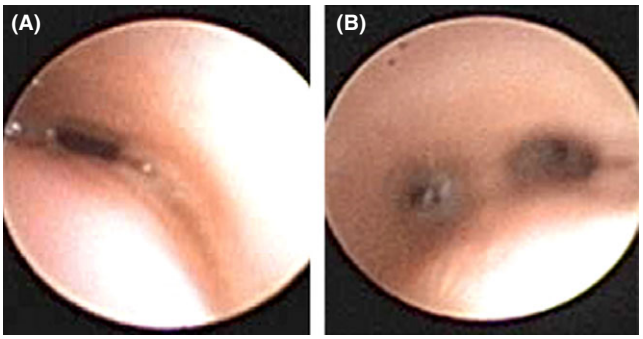

FIGURE 2 A, Preoperative flexible bronchoscopy demonstrating 95% dynamic collapse of the posterior trachealis with coughing, indicating severe tracheomalacia. B, Postoperative flexible bronchoscopy demonstrating correction of the malacic trachea

The patient was positioned in the left lateral decubitus position for a right thoracoscopy. Identification and mobilization of esophagus, thoracic duct, and aorta were necessary to approximate the trachea to the prevertebral fascia, while exercising extra caution to protect the left vagus and left recurrent laryngeal nerves.[7,8] As described in the technique used by Shieh et al,[2] endoluminal bronchoscopy (bronchoscope placed inside the endotracheal tube) was used to assist in suturing the posterior membranous trachea to the anterior spinal ligament (Figure 3A and B) using nonresorbable 4.0 prolene sutures. Continuous bronchoscopy was performed to ensure that these sutures did not enter the tracheal lumen during placement.[2] Ventilator settings were titrated to maintain normal values on arterial blood gases. During 1-lung ventilation, pressure control ventilation with 100% FiO_2 and higher positive end-expiratory pressure (5-8 cm H_2O) were necessary to maintain oxygen saturation levels >92%. Peak inspiratory pressures were between 17 and 18 cmH_2O throughout. The respiratory rate was titrated to maintain an endtidal

CO_2 of 35-40 mm Hg. We did not encounter any issues with oxygenation or ventilation during tracheoscopy; however, we were in constant communication with the surgeons about the need for intermittent ventilation and provided insufflation of oxygenation through the bronchoscope during its use. Theoretically, there is a risk of barotrauma with instillation of oxygen via the bronchoscope's working channel while in the tracheobronchial tree, which can be avoided by not insufflating while the scope is in the small, distal airways. Presumably, when working in the trachea during tracheopexy, there is enough retrograde leak of oxygen around the bronchoscope to minimize the risk of uncontrolled PEEP and subsequent barotrauma.

The patient was hemodynamically stable throughout surgical dissection, and SSEPs were intact at the end of the procedure. Acetaminophen, ketorolac, and hydromorphone were given at the end of the case for postoperative analgesia, and dexmedetomidine was infused at 1 μg/kg/h to facilitate extubation to her home BiPAP. Postoperatively, the patient recovered well. Her daytime work of breathing and chronic cough improved to the extent that she was able to wean to room air during the day, and only required BiPaP at night.

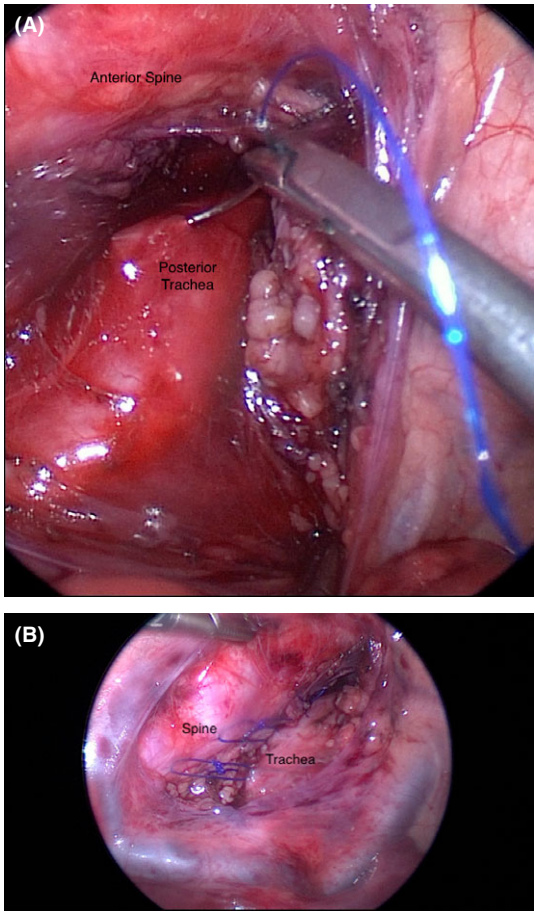

FIGURE 3 A, Thoracoscopic view of suture placement between the posterior membranous trachea and the anterior spinal ligament. B, Thoracoscopic view of final suture placement illustrating the attachment of the posterior membranous tracheal wall to the anterior spinal ligament

4 | PATHOPHYSIOLOGY OF TRACHEOMALACIA

Tracheomalacia commonly results from a reduction in longitudinal elastic fibers or impaired cartilage integrity, and leads to flexible cartilage rings and/or a pliable posterior membrane causing increased tracheal collapsibility. Because of the lack of tracheal stiffness, normal physiologic airway collapse that occurs during expiration in response to elevated intrathoracic pressures during spontaneous ventilation is exaggerated, and can cause clinically significant obstruction.[1,9] This response is most profoundly noticeable in a child that is crying or coughing because of forced exhalation, when intrathoracic pressure is substantially greater than intraluminal pressure.[1,9] In addition to intrinsic causes for collapse, the abnormally compliant trachea can also face external compression from adjacent intrathoracic structures—specifically, anterior aortic impingement or posterior esophageal compression.[1,10,11] Although the size and the position of the aorta are relatively fixed, subtle changes in diameter can have dramatic effects on the airway.[1] The esophagus, however, is a dynamic structure that fluctuates in size with feeding and swallowing, and it can also have obvious clinical effects as it expands and impinges on the trachea posteriorly.[1] In a spontaneously ventilating patient, tracheomalacia is considered severe when the anterior and posterior walls collapse to the point of complete coaptation.[1-3]

5 | CLASSIFICATION

Although 1 in 2100 children is thought to have some degree of congenital tracheomalacia, there is growing evidence to suggest that its prevalence is actually underestimated.[3,12] Often diagnosis is difficult in newborns and infants due to their already compliant airway; however, severe tracheomalacia is easier to recognize in this population when it coincides with specific syndromes such as tracheoesophageal malformations, aortopulmonary anomalies, and bronchopulmonary dysplasia because it is suspected and is screened for selectively.[12,13] Less significant airway malacia is usually self-limiting and resolves by the age of 2 without intervention, but for those cases where tracheomalacia persists, children will frequently present later in life with nonspecific respiratory symptoms (ie, frequent infections, cough, wheezing, stridor); and, thus, go misdiagnosed.[4]

5.1 | Congenital or primary tracheomalacia

Some suggest that the congenital or primary form of tracheomalacia associated with tracheoesophageal pathology is a result of a faulty or improper division of the foregut during the embryologic development of the trachea and esophagus.[1,4,13] The walls of the trachea and the esophagus that contain the fistula connection are abnormal in patients with tracheoesophageal fistulas. This inherent pathology of the walls may be the actual cause for the laxity in the membranous portion of the trachea. For congenital malacia not associated with these disease states, some hypothesize that improper foregut

division still occurs and causes inherent tracheal weakness, but it is not severe enough to result in significant pathology of these structures.[1,4,13] Frequently congenital tracheomalacia is associated with other syndromes. Up to 50% of patients with primary tracheomalacia have concomitant congenital cardiac disease or pulmonary pathology.[14,15] A large majority also have other associated airway anomalies like tracheoesophageal fistulas, subglottic stenosis, and vocal cord pathology.

5.2 | Acquired or secondary tracheomalacia

Acquired or secondary tracheomalacia is more common than congenital forms and results from a degeneration of cartilaginous tissue. External compression from aberrant or dilated vascular structures typically found in patients with complex congenital cardiac defects accounts for a large majority of these cases and often results in anterior compression (ie, tetralogy of Fallot with an absent pulmonary valve; aberrant aortic arch with anomalous branching vessels). Other space occupying lesions like mediastinal masses, abscesses, and cysts can also cause ischemic injury leading to subsequent necrosis of the tissue.[4] This tissue defect disrupts the structural integrity of the trachea and causes increased collapsibility.[4,16] Iatrogenic factors like prolonged endotracheal intubation or need for tracheostomy creates weakness in the tracheal walls by this same mechanism and are also notable culprits. Occasionally, surgical correction of congenital tracheomalacia (ie, tracheoesophageal fistula repair) results in tracheal compression and subsequent secondary malacia. For example, a patient with tracheoesophageal fistula that has been operated on may develop stenosis of the esophageal anastomosis, creating a dilated proximal esophageal pouch that causes tracheal compression. Finally, skeletal deformities make up a smaller subset of acquired forms of tracheomalacia.[1,4]

6 | SYMPTOMS

Diagnosis is often challenging since symptoms are usually not specific for tracheomalacia. Most children present with cough, stridor, and wheezing, which are frequently misdiagnosed as signs of reactive airway disease.[12] Some patients with tracheomalacia have recurrent respiratory tract infections secondary to impaired clearance of secretions given the morphological abnormalities of the trachea. This is most notable in patients with underlying lung disease like cystic fibrosis. One study showed that in patients with cystic fibrosis and known tracheomalacia, time to acquisition of *Pseudomonas aeruginosa* was significantly shorter than those without tracheomalacia (1.3 years earlier).[17]

More severe tracheomalacia can present with signs of significant airway compromise including retractions, cyanosis, and hyperextension of the airway. Small children can exhibit feeding difficulties because esophageal dilation caused by food boluses can narrow the pliable tracheal lumen to the point of collapse and cause brief periods of cyanosis. These "dying spells," also known as "reflex apnea" or "death attacks," are considered the most life-threatening symptoms associated with tracheomalacia.[1] Tracheomalacia has been implicated as a cause of acute life-threatening events (ALTEs).[1,4] The American Academy of Pediatrics has recently replaced the term "ALTE" with "BRUE," or brief resolved unexplained event.[18] Although similar in nature, BRUE describes "an event occurring in an infant <1 year of age when the observer reports a sudden, brief, and now resolved episode of more than one of the following: cyanosis or pallor; absent, decreased, or irregular breathing; marked change in tone; or altered level of responsiveness." This is slightly different than the definition of ALTE in that it includes an age limitation and removes "life-threatening" from the definition.[18]

7 | DIAGNOSIS

Generally, a detailed history and physical exam does not provide enough information for definitive diagnosis, although they are of utmost importance in determining the next steps. Although there are no standardized criteria to confirm the diagnosis of tracheomalacia, frequently both static and dynamic evaluation of the airway and surrounding structures is necessary. Many patients suspected of having tracheomalacia undergo a series of diagnostic procedures to evaluate and formulate a treatment plan. These include pulmonary function tests, radiographic imaging, and bronchoscopic evaluation.

Pulmonary function tests have limited utility in that they often require patient participation, which may be difficult to attain with young pediatric patients. If successful, a flow-volume loop will illustrate an expiratory flow restriction and a reduced mid-expiratory/mid-inspiratory ratio, both of which are nonspecific.[15] Effort-independent imaging is often more practical in diagnosing the extent of tracheomalacia. Advances in computerized tomography have made it an appealing option for reconstructing tracheal anatomy. Specifically, multidetector computed tomography reduces the amount of ionizing radiation a child is exposed to when compared to traditional single detector CT and is expeditious in producing images, which obviates the need for sedation or general anesthesia.[19-21]

Rigid and flexible bronchoscopy allow for direct visualization of the airway, evaluation of supraglottic structures, and examination the tracheobronchial tree throughout the respiratory cycle. Both, however, require general anesthesia that in and of itself can pose significant risk and can be practically challenging in the very young and the very sick.[19,20] An advantage of flexible bronchoscopy is less disruption and distortion of baseline airway architecture when compared to rigid bronchoscopy. Together, both rigid and flexible bronchoscopy provide the best global assessment of the airway.[22] Bronchoscopy can also help predict the likeliness that surgical intervention may be needed. In a more recent study, Okata et al[23] described a semiquantitative index for bronchoscopic assessment of tracheomalacia called the "oblateness index". This prognostic tool takes into account the relationship between the horizontal distance between the edges of airway cartilage (L) and the maximal height of the airway lumen (M) in a formula defined as = (L − M)/L.[23] The

oblateness index can range from 0-1 with the latter indicating complete collapse. They found that those patients who had "clinical symptoms of ALTE, oblateness index of ≥ 0.70, and multiple malacic lesions" were more likely to need surgical intervention.[23]

8 | TREATMENT

Although in most patients tracheomalacia will resolve or become asymptomatic by age 2, for those who have symptoms that persist beyond that, there are several treatment options. These range from conservative therapy to surgical intervention. For mild tracheomalacia, symptom control is the mainstay of treatment.[7] Hypertonic saline to loosen and thin secretions aids with mucus clearance, and inhaled steroids decrease airway inflammation.[1,4] For more moderate to severe tracheomalacia, continuous positive airway pressure (CPAP) may be effective in stenting open the trachea by providing intraluminal pressure to improve respiratory mechanics. Positive pressure can also be delivered by tracheostomy, which can relieve obstruction and also stent open the airway.[1] Although this may be an effective treatment option, CPAP is burdensome to both the patient and the family.

Surgical intervention is indicated for severe symptomatic tracheomalacia that has failed conservative therapies. Patients who have experienced ALTEs, recurrent pneumonias or who require intubation for airway control should be considered surgical candidates.[4] Several surgical procedures have been described to correct tracheomalacia. These range from resecting and reconstructing a small portion of defective trachea to fixating intrathoracic structures to reduce extrinsic compression. Although the effectiveness of tracheal reconstruction is supported and well established in the literature, newer procedures are still being evaluated.[24] Preliminary outcomes from tracheoaortopexy and posterior tracheopexy seem promising, and are traditionally performed as open procedures. Tracheoaortopexy alleviates anterior vascular compression of the trachea by elevating the aorta and suturing it to the posterior aspect of the sternum, while posterior tracheopexy addresses a malformed posterior trachea and membranous tracheal intrusion.[2-5,8-11] This is accomplished by stenting open the trachea through the attachment of the posterior tracheal membrane to the anterior longitudinal spinal ligament under direct bronchoscopic guidance via thoracotomy.[2,3]

More recently, posterior tracheopexy procedures have been successfully performed thoracoscopically, which is advantageous given the already compromised respiratory mechanics typically found in this patient population.[7,8] However, this approach is relatively new, and, therefore, the anesthetic management of these patients is still variable. Our technique is described above. One must note that this mode of correction has competing surgical and anesthetic needs that present unique challenges to the physicians caring for these patients. Because surgical correction requires both operative FOB guidance for internal placement of tracheal sutures and simultaneous lung isolation for adequate external visualization of the trachea, the anesthesiologist must be resourceful when choosing a ventilation strategy.

Many airway tools traditionally used for 1-lung ventilation are limited in this context. For example, endobronchial intubation of a main stem bronchus is precluded because the bronchoscope must be placed through the ETT to guide tracheal correction intratracheally. The use of a double lumen tube is also similarly restricted because it does not allow for adequate visualization of the trachea; and, even the smallest sized double lumen tube is still too large for use in this typically young population. Additionally, traditional placement of endobronchial blockers through endotracheal tubes also restricted because it can reduce cross-sectional ETT diameter enough to impair ventilation in smaller children when a bronchoscope is passed simultaneously. However, we have found that by placing a bronchial blocker extraluminally, 1-lung ventilation with adequate oxygenation can be successfully achieved, while still allowing for FOB guidance.

9 | CONCLUSIONS

Tracheomalacia can be associated with significant morbidity and mortality. New techniques for the treatment of severe cases are evolving. Thoracoscopic posterior tracheopexy is a relatively novel approach that requires special surgical and anesthetic considerations. Additional thought is also aimed at preparing these patients for surgery since those who require this kind of intervention are typically complex and present with their own unique set of challenges. Given the heterogeneity of this population, a highly functioning interdisciplinary team is needed to optimize diagnosis and treatment. Adequate preparation, organization, and ongoing communication among a diverse set of specialty teams is necessary for the successful care of these patients.

CONFLICT OF INTEREST

Emily DeBoer, MD—consultant for TripleEndoscopy Inc. Jeremy D. Prager, MD—consultant for TripleEndoscopy Inc. David Polaner—Correspondence Editor for Pediatric Anesthesia. All other authors have no conflict of interest.

ETHICAL APPROVAL

None required.

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