The Assessment of Major Airway Function in a Ventilator-Dependent Patient with Tracheomalacia


A 60-pack-year smoker presented with cough, dyspnea and orthopnea of three months’ duration. Spirometry revealed severe reduction in maximal expiratory flow; CT of the chest and bronchoscopy demonstrated expiratory collapse of a mid-tracheal segment, and a presumptive diagnosis of tracheomalacia was made. A right lateral thoracotomy was performed to resect the unstable segment and improve maximal expiratory flow. Diffuse major airway disease with absence of cartilaginous rings from the thoracic inlet to the mainstem bronchi was encountered. The trachea and mainstem bronchi were stented externally. A high resistance to airflow and absence of expiratory flow limitation were present, suggesting a fixed rather than variable intrathoracic obstruction of major airways. This case illustrates some potential pitfalls in preoperative assessment of patients with tracheomalacia. Recordings of airway pressure and flow during mechanical ventilation are useful in distinguishing between fixed and variable intrathoracic obstruction and may complement tests of airway anatomy.

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The physiologic hallmark of intrathoracic tracheomalacia is the collapse of major airways during expiration. This usually is caused by destruction of the cartilaginous support rings due to compression by extrinsic mass lesions or due to inflammatory diseases, such as relapsing polychondritis. In the following case report, we describe a surgical attempt to stiffen the trachea in a patient with relapsing polychondritis and show how the combined use of newer imaging techniques and physiologic measurements in the ICU aided in the postoperative management of this patient.

Case Report

A 56-year-old woman with a 60-pack-year smoking history was transferred to our institution with a diagnosis of tracheomalacia for consideration of surgical repair. She had been well until four months previously when she developed cough and fever but failed to respond to antibiotic therapy. Two months prior to admission, a chest x-ray film showed a right lower lobe infiltrate which cleared after intravenous antibiotic therapy. Computed tomography of the thorax, bronchoscopy and tracheal mucosal biopsy (revealing diffuse lymphocytic infiltration) suggested the diagnosis of tracheomalacia due to inflammatory disease.

On physical examination the patient appeared cachectic and in moderate respiratory distress. The blood pressure was 120/80 mm Hg, the pulse rate was 104 beats per minute, the respiratory rate was 24 breaths per minute and the temperature was 37.1°C. There was no inflammation or evidence of cartilaginous destruction of the ears or the nasal septum. The chest was hyperinflated, the expiratory phase prolonged and there were diffuse expiratory rhonchi. The cardiovascular examination was unremarkable.

The results of a pulmonary function test are summarized in Figure 1. There was a marked reduction in maximal inspiratory and expiratory flows consistent with dynamic collapse of intrathoracic and extrathoracic airways. The single breath diffusing capacity was normal, indicating the absence of severe emphysema. A multilevel CT examination of the chest revealed the presence of mid-tracheal narrowing (Fig 2). A bronchoscopic examination with the patient awake in the supine position confirmed the CT findings and revealed expiratory collapse of a segment of the mid-trachea. The distal trachea and mainstem bronchi remained patent throughout the respiratory cycle.

On the fourth hospital day, the patient underwent a surgical exploration through a lateral thoracotomy with the intent to resect the unstable tracheal segment. However, on palpation of the trachea, there was absence of cartilaginous support from the thoracic inlet to the carina and mainstem bronchi. In an attempt to stabilize the trachea and mainstem bronchi, a series of rings (5 to 6 mm in width) fashioned from 16-mm polytetrafluoroethylene vascular graft material (Gore-tex, W. L. Gore and Associates, Inc., Flagstaff, AZ) was sutured to the external surface of the airways. Following the surgical repair, intraoperative bronchoscopy with the chest still open showed near normal dimensions of the central airways, which remained patent during endoscopic suctioning.

During the next several days, the patient failed repeated attempts to wean her from mechanical ventilation. Within minutes following cessation of mechanical support, the patient became tachypneic and diaphoretic, had lower rib cage retractions and complained of intense shortness of breath. Several causes of respiratory failure were entertained. We considered that the flow-limiting segment had simply shifted to more peripheral conducting airways, either because of widespread intrinsic airway disease or because severe emphysema had not been recognized preoperatively. Alternatively, there was concern that postoperative airway edema had reduced the lumen of the trachea and increased the resistance to gas flow. Measurements of respiratory system mechanics, dynamic CT imaging of the chest and bronchoscopy were performed to resolve
these questions.

Figure 3 shows a recording of volume, flow and airway opening pressure during mechanical ventilation with a volume-cycled ventilator with constant inspiratory flow (0.45 L/s) and stepwise deflation of the respiratory system from end inspiration to static equilibrium volume (ie, the volume at which airway occlusion pressure is atmospheric). Gas flow was measured at the oral end of the endotracheal tube (Filevich No. 2 pneumotachograph, Validyne MP45 pressure transducer) and volume was derived by integration of the flow signal. A pneumatic occlusion valve (Hans Rudolph 4200) was interposed between the airway and the ventilator tubing.

Note that the peak airway pressure was 54 cm H₂O while the static elastic recoil pressure of the respiratory system at end inspiration (end inflation hold pressure) was only 18 cm H₂O. This pressure difference of 36 cm H₂O is consistent with an extremely high resistance to inspiratory gas flow (46 cm H₂O/L/s). Also note the marked reduction in expiratory flow at all volumes, indicating an increase in expiratory resistance as well (25 cm H₂O/L/s). The additional resistance of the expiratory ventilator tubing reduced expiratory flow even further, suggesting that this patient was not flow limited (ie, not breathing on the maximal expiratory flow-volume loop).

Based on the absence of expiratory flow limitation and the high inspiratory resistance, we concluded that this patient had a fixed, rather than a variable, intrathoracic large airway obstruction. This conclusion was confirmed by dynamic ultrafast CT imaging (Picker Fastrack, model C-100XL, Imatron, South San Francisco) on the following day (Fig 4), which revealed massive postoperative edema of the tracheal wall and both mainstem bronchi. There was no change in the diameter of either the trachea or mainstem bronchi with respiration. The cross-sectional area of the tracheal lumen averaged 9 sq mm (~3 mm diameter) at all levels between the end of the endotracheal tube and the carina. The small tracheal lumen precluded a bronchoscopic examination beyond the tip of the endotracheal tube.

The patient was treated with prednisone, 60 mg daily, and underwent a tracheostomy. Serial bedside evaluations of pulmonary mechanics documented a dramatic improvement in expiratory flow during her prolonged postoperative course (Fig 5). Three months after surgery, the patient was able to breathe without mechanical ventilatory support for several hours each day.

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**Figure 1.** Pulmonary function testing results.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Result</th>
<th>Pred, %</th>
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<td>TLC, L</td>
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<tr>
<td>DCO₂sb⁻³</td>
<td>17.8</td>
<td>83</td>
</tr>
</tbody>
</table>

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**Figure 2.** CT coronal view reconstruction demonstrating midtracheal narrowing, maximal above the aortic arch level.

**Figure 3.** Simultaneous bedside recordings of airway opening pressure, flow and volume vs time during a mechanical inflation and subsequent stepwise occlusions during deflation. Note the very high peak airway pressure relative to end inflation hold pressure. The decrease in volume during airway occlusion is an artifact because of integrator drift.
which aortopexy alone had failed. In our patient, the trachea was reinforced with Gore-tex vascular graft material. This was clearly an experimental procedure because other approaches were not viable. It is not our intent to endorse this surgical procedure, yet this case does illustrate some important lessons for the management of patients with tracheomalacia.

The demonstration of dynamic collapse of only one segment of the trachea led to the misleading assumption that the remainder of the airways were relatively normal. In retrospect, the pitfalls of this reasoning could have been anticipated by recalling the determinants of maximal expiratory flow. Maximal expiratory flow is inversely proportional to the square root of airway compliance. In the presence of normal lung elastic recoil (preventing flow limitation in peripheral airways), dynamic collapse must occur at the site of the most compliant (least stable) segment within the major airways. Excision or stenting of that segment may cause little improvement in flow if the compliance of remaining airway segments also is abnormal. Collapse will occur at the “weakest” link in the system irrespective of the average mechanical properties of the tracheobronchial tree, hence the intraoperative requirement of extensive tracheal stabilization in our patient. We would expect that repairing the weakest link would only have resulted in collapse of the next weakest segment.

A variable intrathoracic major airway obstruction is characterized by a decrease in maximum expiratory flow which is out of proportion to the reduction in maximum inspiratory flow. This is best appreciated from an inspection of the flow-volume relationships obtained during maximum voluntary inspiratory and expiratory maneuvers. Because the interpretation of flow-volume loops requires a certain threshold value of effort, this approach is not feasible for the diagnosis and classification of major airway lesions in intubated or mechanically ventilated patients. In this population, the physician must rely on the separate measurement of inspiratory and expiratory resistance to gas flow and seek to demonstrate expiratory flow limitation during passive deflation of the relaxed respiratory system. Fixed major airway lesions cause a proportional reduction in maximum inspiratory and expiratory flow. Dynamic airway collapse and flow limitation during passive expiration are not features of fixed obstructing lesions. This, in fact, was demonstrated in our patient.

Clarification of the airflow obstruction as being of fixed rather than of variable type had several important implications in the management of this patient. In the absence of dynamic airway collapse, neither further airway stenting nor resective surgery were rational management options. Knowledge of the severity of impairment led the attending physicians to postpone further ventilator weaning attempts and to institute a

**FIGURE 4.** Thin section CT view at aortic arch level demonstrating marked postoperative tracheal edema inside the supporting ring.

**FIGURE 5.** Serial bedside pressure-flow data demonstrating progressive improvement in airflow and reduced expiratory resistance accompanying the patient's gradual clinical improvement.

**DISCUSSION**

Most of the surgical experience in the treatment of tracheomalacia has been summarized in the pediatric literature. If the lesion is confined to a localized segment, it is usually resected. For more extensive lesions, a number of approaches have been proposed. Page and Klein have treated a patient with severe congenital tracheomalacia with prolonged endotracheal intubation. Vasko and Ahn have externally splinted the trachea in a child with segmental tracheomalacia. The segment was reinforced externally with a rib, which in turn was sutured to the anterior chest wall.

An alternative approach involves surgically displacing the ascending aorta closer to the sternum, thus causing secondary traction on the anterior tracheal wall (aortopexy). There is no clear evidence, however, that this procedure produces eventual tracheal stiffening or reversal of the underlying structural abnormality.

The use of a Silastic reinforced Marlex mesh as an external tracheal splint was reported by Filler et al. They found this technique to be successful in cases in
trial of corticosteroids. Furthermore, the lung mechanics studies served as baseline data to which subsequent tests could be compared (Fig 5).

While the assessment of respiratory system mechanics with the interrupter technique provides detailed, quantitative information, the distinction between fixed and variable intrathoracic obstructing lesions alone can be made without extra monitoring equipment using a few simple maneuvers at the bedside. First, in the absence of spontaneous respiratory efforts, the peak to end inflation hold pressure difference provides primarily a measure of the inspiratory resistance offered by the endotracheal tube and major airways. Values in excess of 20 cm H2O are unusual, even in patients with severe COPD. Second, the presence or absence of expiratory flow limitation easily can be assessed at the bedside by observing the peak airway pressure response to applied PEEP. Expiratory flow limitation means that the driving pressure for expiratory flow is in excess of that necessary to achieve maximal flow. In the presence of flow limitation, a small reduction in driving pressure through the application of extrinsic PEEP, therefore, does not reduce expiratory isovolume flow. As a consequence, end expiratory lung volume and peak airway pressure remain constant. In contrast, a reduction in isovolume flow, as is predictably observed with PEEP in patients without flow limitation, always leads to an increase in end expired lung volume, which is paralleled by an increase in peak airway pressure. This, in fact, was seen in our patient.

Having demonstrated that the problem was not one of dynamic airway collapse distal to the sites of surgery, the question arose whether the obstructing lesion was local or diffuse. While endoscopy is the procedure of choice when defining the anatomy of the airways, in this case the inspection was limited to the mid-trachea by massive mucosal edema and tracheal narrowing. Dynamic imaging of the airway by ultrafast CT was very valuable in this case. This technique showed diffuse tracheal narrowing from the tip of the endotracheal tube down to the mainstem bronchi. With this clinical tool, cross-sectional images of the trachea can be examined during different phases of the respiratory cycle, thus providing information on the compliance of the airways. Unfortunately, to date, normal values for tracheal diameter change with lung volume and respiration have not been defined. In our patient, several days after surgery the tracheal lumen remained fixed and independent of intrathoracic pressure.

**Conclusion**

In summary, we have reviewed the surgical options in the management of patients with tracheomalacia. We caution that the preoperative demonstration of segmental dynamic collapse of the trachea does not prove discrete localization of disease. Finally, we have shown that simple bedside measurements of airway pressure and flow provide information on the nature of major airway narrowing (fixed vs variable obstruction) and that anatomy and measurements of airway function can be integrated to optimize clinical assessment and care.

**References**

1. Page BA, Klein EF. Tracheal stent as an aid in weaning from mechanical ventilation in tracheomalacia. Anesthesiology 1977; 47:300-01