Anesthesia for the Patient with Tracheal Stenosis

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The larynx ends at the inferior edge of the cricoid cartilage. The cervical trachea starts below the inferior border of the cricoid and extends to the sternal notch, and the thoracic trachea is located from the sternal notch down to the carina. In total, the trachea is composed of 18 to 22 cartilaginous rings, each being 0.5 cm wide in the adult. The trachea is approximately 12 cm long in men and 10 cm long in women. Normal diameter is about 1.8 to 2.5 cm. With hyperextension, up to 50% of the trachea can be above the sternal notch. Blood supply to the trachea is from the inferior thyroid, the internal thoracic, the supreme intercostal, and the bronchial arteries.

Stenosis of the trachea signifies a functional impairment, with peak expiratory flow rates changing from 100% in a normal trachea with a diameter of 2 cm to 30% in a trachea with a diameter of 5 mm.[1] Tracheal stenosis has multiple causes. Pediatric tracheal stenosis can be divided into acquired versus congenital forms. Acquired forms of pediatric tracheal stenosis include trauma, mostly related to endotracheal intubation or from tracheostomy-induced formation of granulation tissue. Up to 8% of the neonates who undergo prolonged intubation can go on to develop tracheal stenosis. Congenital forms of tracheal stenosis are classified by Cantrell and Guild[2] as generalized hypoplasia, funnel-like stenosis, and segmental stenosis. They can also be categorized based on the length of the stenotic segment and associated anomalies.[3] Congenital tracheal stenoses include tracheal webs, tracheal agenesis/atresia, tracheomalacia, and vascular malformations that extrinsically compress the trachea.[2,3]

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In adults, tracheal stenosis may result from trauma, benign or malignant neoplastic conditions, chronic inflammatory diseases (such as sarcoid and amyloid), collagen vascular diseases (such as Wegener granulomatosis), or external compression from mediastinal masses.[4] Also possible is idiopathic laryngotracheal stenosis, a rare disease, which occurs almost exclusively in women with no identifiable cause of airway stenosis.[5] The most common cause of tracheal stenosis, however, continues to be trauma from prolonged endotracheal intubation.[6] This sequela was first recognized during the poliomyelitis epidemic in the 1950s when prolonged mechanical ventilation was used.[7]

Cooper and Grillo[8,9] first studied tracheal stenosis at the cuff site and showed that the main cause was the pressure exerted by the cuff. Cuff pressures greater than 30 mm Hg cause mucosal ischemia by exceeding mucosal capillary perfusion. This area of ischemia can develop chondritis and granulation tissue, then heal by fibrosis, leading to progressive tracheal stenosis. Cuff-related strictures assume an eccentric circumferential configuration, in contrast to tracheostomy-related strictures, which have a V shape on account of the loss of the anterior cartilaginous arch. The incidence of tracheal damage can be reduced with the use of high-volume, low-pressure cuffs that minimize the pressure exerted on the trachea via a larger area of contact. However, despite the use of these cuffs, up to 11% of patients may still develop some degree of tracheal stenosis as shown in a prospective study of 150 critically ill patients.[10] As compared with tracheal stenosis, glottic stenosis as a result of intubation is usually present posteriorly and can be associated with arytenoid cartilage dislocation, vocal cord paralysis, and granulomas. Bogdasarian and Olson[11] have classified this type of glottic stenosis based on location and involvement of vocal cords.

Another way of categorizing tracheal stenosis is on the basis of spectrum of fixed to dynamic collapse.[3] A fixed stenosis is typically a fibrotic segment of the trachea. As discussed in the ''Physics of airflow'' section, the Bernoulli principle holds that as air accelerates through a small orifice, the pressure will drop. Because the membranous surface of the trachea is flexible and mobile, it can be drawn up into the trachea. When this occurs, the actual airway compromise is greater than that which is simply predicted by the stenotic opening. Dynamic collapse from a prolapsing membranous wall can produce tracheal stenosis in patients without any fixed tracheal narrowing. These patients often have increased work of breathing as a result of chronic obstructive pulmonary disease (COPD), reactive airways disease, and/or obesity. Airway collapse can be on inspiration because of the pressure drop of airflow or on expiration because of the increased velocity of the airflow and the increased abdominal pressure, causing herniation of the membranous wall into the airway.

Tracheomalacia, literally ''softening of the trachea,'' is a condition in which there has been a weakening of the cartilaginous structures of the trachea.[12,13] Typical causes are rheumatic (polychondritis), infectious, secondary to external beam radiation, and secondary to trauma or surgery.[12] There can be stenosis from partial collapse. Airflow can be limited on inspiration and on forced expiration, the latter being due to increased velocity of the airflow and increased abdominal pressure, causing prolapse of the membranous wall into the airway.

PHYSICS OF AIRFLOW

Principles of Flow

Flow through a tube, whether through the trachea, circulation, or any other orifice, can be difficult to measure. It is important to distinguish between flow and velocity, which are often confused. Flow, often denoted as Q, is defined as the volume passing

a particular surface area per unit of time and is often denoted as mL/s or L/min, whereas velocity is the speed of fluid at a particular point in space. Flowing fluids in tubes possess velocity and pressure that can be used to analyze incompressible flow by using the Bernoulli equation, which states that

$$P+\frac{1}{2}\rho U^2 = P_0$$

where P is pressure, ρ is fluid density, U is velocity, and P_0 is a constant pressure. In this form of the Bernoulli equation, pressure decreases as velocity increases. In the flow inside a tube, velocity decreases as area increases.

Flow can be measured using the average velocity of the fluid across a tube. In laminar flow, the velocity has a parabolic shape, with slower velocities along the edges because of friction (**Fig. 1**), whereas turbulent flow has a flattened velocity profile. Turbulent flow is inefficient, and the energy needed to move a given volume is greater for turbulent flow than for laminar flow.

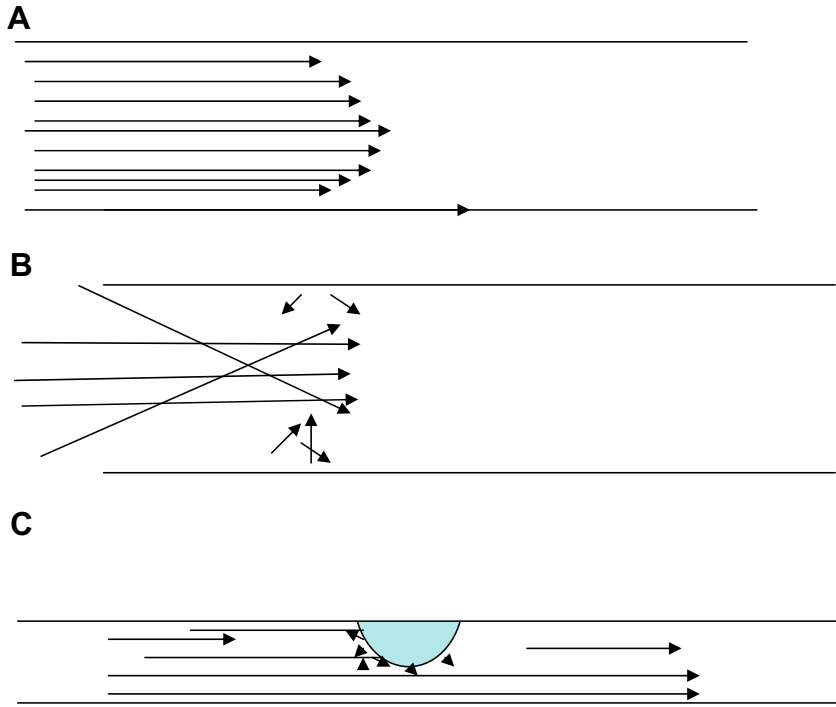

Fig. 1. (*A*) In a smooth-walled tube at low flow rates (ie, small pressure gradients), the flow rate is laminar; that is, flow moves smoothly in concentric circles, with the centermost area having the greatest flow velocity and the area nearest the wall of the tube being virtually stationary. (*B*) As the flow rate (and pressure gradient) increases, the flow transitions from laminar to turbulent. Instead of a neatly ordered flow, the velocities are more randomly distributed, and the energy needed for a given flow rate increases. Many factors govern this transition, including size of the tube, viscosity of the fluid, flow rate, and pressure gradient. These factors are combined in the determination of the Reynolds number. (*C*) Even at low flow rates, the distortion of laminar flow around an obstruction necessitates an increase in the pressure gradient to drive volumes greater than that simply predicted by the decrease in diameter. This is as a result of the effect of turbulent flow.

Most commonly, the flow that needs to be measured by anesthesiologists is not laminar, and the Bernoulli equation cannot be applied because it describes frictionless flows. The transition from laminar to turbulent flow depends on the type of fluid, the speed of the flow, and the shape of the flow. These fluid factors are combined in a ratio called the Reynolds number (Re).

$$\mathrm{Re} = \rho \mathrm{UL}/\mu$$

where ρ is the density of the fluid, U is the mean flow velocity, L is the characteristic length of the flow, and μ is the viscosity of the fluid. For flow through a tube of circular cross section, transition from laminar to turbulent flow occurs at a critical value of Re. A large Reynolds number indicates that viscous forces are not important at large scales of the flow. Using the definition of the Reynolds number, we can see that a large diameter with rapid flow, where the density of the fluid is high, tends toward turbulence. Also, rapid changes in diameter, as occurs in tracheal stenosis, may lead to turbulent flow. If flow in a tube passes through a sudden restriction, the turbulent flow is proportional to the area of the orifice and the square root of the pressure drop through the orifice as opposed to its direct proportionality to pressure gradient in laminar flow. Understanding the physics of airflow becomes an important concept in the diagnosis and treatment of tracheal stenosis.[14]

DIAGNOSIS OF TRACHEAL STENOSIS AND PREOPERATIVE EVALUATION

History and Physical Examination

As previously stated, tracheal stenosis can have numerous causes, with iatrogenic trauma being the most frequent cause. Other conditions that are related to tracheal stenosis should be ruled out. These include amyloidosis, Wegener granulomatosis, sarcoidosis, tuberculosis, and other infectious disease processes.[13] Patients can be tested for antinuclear cytoplasmic antigen to rule out Wegener granulomatosis. Rheumatology is often helpful in interpreting results and evaluating a patient for other collagen vascular diseases. A detailed history of the patient should be obtained, and a review of systems including difficulty clearing secretions, exercise tolerance, and orthopnea should be elicited. Physical examination should include palpation of the trachea, auscultation of the lungs and trachea, and evaluation of neck mobility. Routine blood work is all that is needed. Cardiac screening, such as an echocardiography or stress test, may be needed if the patient has had a change in symptoms or if the stenosis has rendered the patient incapable of activity.

A clear history of any antecedent airway management needs to be collected. A history of traumatic airway management can be surmised from prolonged hoarseness after an operation or from intubation in an emergent situation. All cases of postoperative or intensive care unit ventilation need to be examined for duration and the presence of other comorbidities, such as sepsis and heart failure, that have been associated with the development of tracheal stenosis.

Tracheal stenosis often presents with inspiratory stridor and/or expiratory wheezing. These patients are frequently misdiagnosed with asthma on initial presentation. These patients will be unresponsive to bronchodilators. Also, most patients with tracheal stenosis do not develop symptoms at rest until they have reached 70% stenosis.[15] On physical examination, patients may use accessory muscles or demonstrate tachypnea.

Flow-Volume Loops

Flow-volume loops can be useful in evaluating these patients. The flow-volume loops may show several anomalies: a delay in reaching peak expiratory flow, a truncation of

peak expiratory and peak inspiratory flow, and/or an abrupt drop of expiratory flow at the end of expiration. In a patient with asthma, one can see flattening of the expiratory curve. This occurs predominantly late in expiration, with slowing of terminal flow rates. Peak flows are maintained, but the flow-volume curve becomes more convex toward the horizontal axis.[16]

On a flow-volume loop in a patient with a fixed upper airway obstruction such as tracheal stenosis, there is flattening of the inspiratory and the expiratory phases. The primary effect occurs early in expiration and results in a truncated, flat-topped flow-volume curve, with a normal late expiratory portion of the flow-volume curve.[17,18] It is important to remember that the quality of the flow-volume loops is totally dependent on the patient's effort and cooperation and that tracings obtained may not have the shapes presented earlier.[19]

Imaging

Imaging can be very useful in evaluating these patients. Chest radiographs can seem normal. Sometimes, a circumferential lesion can be seen on plain radiographs. Computed tomography (CT) scans can be an extremely useful diagnostic tool in these patients. However, it is important to obtain an airway CT rather than a neck or chest CT. A CT dedicated to the airways will evaluate the entire airway with thinner cuts. If thicker cuts are used, one can underestimate or miss the area of stenosis. Advances in imaging allow for 3-dimensional (3D) reconstruction of the trachea.[20] Multidetector CT is the imaging modality of choice. Multiplanar reformations and 3D images allow surgeons and interventional pulmonologists to select the adequate procedure for the patient. In tracheal stenosis secondary to prolonged intubation, one will see eccentric or concentric thickening, with associated luminal narrowing. Volume rendering techniques provide information on the length of stenosis and patency of the airways.[4] One may see a characteristic ''hourglass'' shape. In stenosis secondary to other causes, other findings consistent with the disease process can be evident on the CT scan: enlarged lymph nodes in fibrotic tuberculosis, thickening and calcification of tracheal rings in Wegener granulomatosis, and bronchial wall thickening in tracheobronchial amyloidosis. CT scans may demonstrate specific findings that can help narrow the differential diagnosis.

Endoscopy

Bronchoscopy remains the primary procedure in the diagnostic workup of tracheal stenosis and is the key in defining the characteristic features, extent, and location of the stenosis. Bronchoscopy in a patient with postintubation stenosis shows (1) circumferential luminal narrowing less than 2 cm in length, (2) a thin membrane that extends into the lumen, or (3) a long segment of eccentric soft tissue thickening.[12,21] Patients with stenosis related to tuberculosis will have granulation tissue with friable and ulcerated mucosa, whereas those with sarcoidosis may demonstrate a raised, cobblestoned appearance of the mucosa. Patients with Wegener granulomatosis may have inflammatory ulcers, plaques, or granulomatous tissues. Bronchoscopic findings in patients with amyloid may reveal thickened tracheal segments corresponding to areas of amyloid deposits. Patients may also have generalized mucosal edema and erythema on bronchoscopy.[22] Performing a fiberoptic bronchoscopy in an awake patient allows the physician to examine vocal cord function to determine if recurrent laryngeal nerve damage is present; it also allows for the evaluation of dynamic airway collapse with respiration. Bronchoscopic findings are not pathognomonic, and biopsies are often required to include or exclude an inflammatory disease process.

MANAGEMENT

The use of endoscopic treatment for the initial management of tracheal stenosis started in the 1980s, and bronchoscopic examination is paramount in patients with tracheal pathology. This examination will define the nature of the lesion, length, location, and degree of obstruction. If the obstruction is moderate to severe, bronchoscopic evaluation may be deferred until definitive treatment is chosen.[23] Rigid bronchoscopy is usually chosen in the nonsurgical treatment of these patients. Rigid bronchoscopy allows coring out of tumors, provides a means for ventilation, and may tamponade a source of bleeding.

Irradiation

Squamous cell carcinoma and adenoid cystic carcinoma respond to radiation. However, used alone, it is not a definitive treatment, and most tumors recur after a few years. However, radiation can be used as an adjunct to other methods.[24]

Dilatation

Dilatation can be used for the management of tracheal obstruction in emergency situations or in a planned approach to determine if surgical resection will be needed. Simple dilatation is often not a definite treatment as recurrence occurs. It should be used as a temporizing measure until more definitive treatment can be undertaken. Dilation is not recommended for mature, firm stenosis or those with cartilaginous components.[23] Ideally, it is performed under direct visualization and with a means of securing the airway. Dilatation can be performed with smooth round dilators, gradual dilation with ventilating bronchoscopes of increasing diameter, rigid bronchoscopes of increasing diameters, or dilating balloons passed through flexible bronchoscopes.[15,24] The risk of edema and mucosal trauma increases with multiple dilations, making this method of treatment less popular. Steroids can be used to minimize edema after these dilatations. Steroids can delay the synthesis of collagen in the early stages of scar formation. However, they can also delay wound healing and cause cartilage resorption.[23] In addition, local injection of steroids or mitomycin C may staunch the recurrence of scar formation at the site of stricture.

Laser

Lasers deliver energy to achieve cutting, coagulation, and vaporization with great precision and microhemostasis. The end result is less perioperative edema. The CO_2 laser has a wavelength of 10.6 μm and is used for lesions involving soft tissues. It can deliver power from less than 1 W to 100 W. When operating on the airways, it is used in the range of 3 to 6 W. However, the CO_2 laser cannot coagulate vessels larger than 0.5 mm in size and depends on an optic delivery system. Endoscopic laser treatment includes radial incision and dilation and excision.[21] Radial cuts are made into the fibrotic segment. This allows a planned tear into the stenosis and can potentially provide better results with dilatation before stenting (see later section). Lasers are also used for the debridement of obstructive lesions of recurrent respiratory papillomatosis and produces minimal scarring despite repetitive use. The CO_2 laser is also used as a treatment for glottic webs or for acquired subglottic stenosis.[21,25]

The neodymium:yttrium-aluminum-garnet (Nd:YAG) laser has become the laser of choice in the treatment of obstructing tracheal lesions because it is able to reach distant corners of the tracheobronchial anatomy. This laser can be used in concentric

stenoses and in palliation of tracheal malignancies. However, endoscopists are unable to control the depth of penetration (up to 10 mm, depending on wattage and exposure time) with this type of laser, putting patients at risk for perforation.[25]

The potassium titanyl phosphate (KTP) or argon laser has also been used in the airway. The KTP laser is frequently used in the small pediatric airway because of its small diameter fibers and its coagulating effect on soft tissue.[23] It has a tissue penetration of 4 mm and is useful for vascular lesions as well. The KTP laser can be used with a fiberoptic delivery system, making its delivery more precise.[25] Lasers cannot destroy the root of the tumor, and recurrence is frequent after laser tumor ablation. If definitive surgical treatment is to be undertaken for patients, lasers should not be used because they can destroy healthy tissue that is adjacent to the lesion, which could compromise future anastomotic sites for the surgeon.[24]

Although not the focus of this article, it needs to be stressed that before any laser or cautery use in the airway, typical safety precautions need to occur. These include lowering the FiO_2 to below 30%, ensuring that the endotracheal tube (ETT) or other combustible materials are well away from the laser, and the universal donning of laser protective glasses.

Stents

Stents can have multiple uses in this patient population. They can be used as palliation for patients who have tumors that are too extensive for surgery, patients with benign lesions but extensive strictures, or patients with trachea destroyed by multiple reconstruction attempts. They can be used as temporizing measures until the patient is ready for surgery or as an adjunct to surgery to stabilize the newly anastomosed trachea.[24] Ideally, stents must have several characteristics: they should reestablish the airway with minimal morbidity and mortality, have limited migration, be easily removable, maintain luminal patency without ischemia or erosion, induce minimal granulation tissue formation, and be economically affordable.[26] But no stent currently satisfies all of these characteristics.

The advantage of an airway stent is that it supports the airway wall against collapse or external compression and impedes extension of tumor into the airway lumen. Stents can be safely used in patients undergoing external beam radiation therapy or brachytherapy; however, they can burn or break when subjected to Nd:YAG laser energy, the effects of electrocautery, or the effects of argon plasma coagulation.

Silicone stents, such as the Dumon (Endoxan prosthetics, Ayton, France) and Hood brands (Pembroke, MA, USA), protect from collapse and ingrowth of tumors.[26] Silicone is the least irritating of the stent materials, so there is less associated inflammation. Silicone stents are easily removed and exchanged, resist external compression, and cause minimal formation of granulation tissue. However, their more rigid structure cannot conform to irregular airways. The most common complication seen with silicone stent is migration of the stent, followed by granulation formation and mucous plugging. Other complications include malposition, infection, or obstruction related to inflammatory tissue overgrowth.[23]

Metal stents provide an advantage in that they are less likely to inhibit the respiratory cilia compared with silicone stents. In early generations, these metal stents required a balloon to expand after the stent was placed in the proper position. With the use of self-expanding stents, a balloon is no longer needed. Expandable stents are made of woven wire struts or meshes, typically from alloys such as nitinol. They can be positioned using bronchoscopy. However, tumor ingrowth can occur along the length of the stent. Also, these stents are extremely difficult to remove, may collapse or fracture with increased external pressure along the stent, and have ingrowth of granulation tissue that may lead to lumen occlusion.[26]

The covered self-expanding metallic stents are considered hybrids of metallic and silicone stents. They have a decreased risk for tumor ingrowth and granulation tissue because the wire mesh is covered with synthetic material. However, the ends are still uncovered to provide anchoring into the mucosa. As a result of their uncovered metal ends, these stents have less migration than their silicone counterparts. However, they also have an increased risk of granulation tissue formation at the ends compared with the silicone stents.[23,26] Because of the formation of granulation tissue, metallic stents are ideally used in cases of malignant strictures, and silicone stents are reserved for use in the treatment of benign strictures.

All stents can be inserted and removed endoscopically under general anesthesia or while awake and sedated with local anesthetic topicalization of the airway. Stents can be inserted using fluoroscopic guidance with bronchoscopic assistance. Bronchoscopic evaluation during stent placement allows insertion of a guidewire across the stricture. Bronchoscopy can again be used after stent placement to evaluate patency and location of the stent. If incorrectly positioned, the stent may be repositioned with bronchoscopy forceps.[26,27]

Anesthesia for stent placement

An experienced anesthesiology team working in close cooperation with the surgeon or interventional pulmonologist is required for optimal care. Sedative premedication should only be used for very anxious patients because of the danger of hypoventilation and further airway obstruction. An anticholinergic agent can be selected as part of the premedication to decrease excessive airway secretions.[28] It is important to remember that in changing a spontaneously breathing patient to positive pressure ventilation, a partially obstructing lesion can completely obstruct the airway. Also, the bronchoscope may completely obstruct the airway when entering the area of stenosis. Both of these issues make preoxygenation vital before starting the procedure. Because of altered airflow, preoxygenation/denitrogenation will take considerably longer than usual. Tidal breathing may be inhibited, and 5 maximal breaths in a patient with a compromised trachea will be insufficient for effective nitrogen washout.

Sedation can be provided without airway compromise, using agents such as dexmedetomidine or ketamine. Dexmedetomidine was initially described as a possible agent for sedation by Hall and colleagues.[29] They took 7 volunteers and found that hemodynamics, oxygen saturation, and respiratory rate were well preserved during the infusion of the drug, whereas most patients experienced amnesia and sedation. The use of dexmedetomidine for the management of difficult airways has been documented with success in other case reports.[30,31] Candiotti and colleagues[32] describe the use of dexmedetomidine for monitored anesthesia care in a randomized, double-blind trial. This trial found that patients undergoing sedation with dexmedetomidine required less fentanyl, had less respiratory depression, and had increased satisfaction with the anesthesia care. It is important to note that dexmedetomidine can result in significant bradycardia and hypotension.

For initial bronchoscopy, local anesthetics can be used to anesthetize the airway. Practitioners may have different ways of doing this. However, local anesthetics are not without their own consequences. They may decrease forced expiratory volume in the first second of expiration and forced vital capacity, further exacerbating the inspiratory airflow limitation by inhibiting airway dilator responses and may cause deep sedation when absorbed in significant quantity. One must also be cautious of total local anesthetics used because these are easily absorbed through mucosal membranes, and toxic levels can be reached.[33,34] Airflow limitation secondary to local

anesthesia of the airway may lead to complete airway obstruction in a compromised airway.

In a patient with significant stenosis who presents with respiratory compromise, the logical first step is to dilate the stricture under minimal anesthetic. Even though the dilatation will not last, this initial step will produce a patient in whom the airway is not critically narrowed so that other diagnostic tests and preoperative preparation can take place. Further, linear cautery of the stenosis and balloon dilatation may produce less inflammation and edema as compared with extensive laser therapy; so the patient would have less risk of acute airway compromise.[26,35]

Induction of anesthesia needs to be tailored to the patients' lesions and their medical history. Traditionally, it is recommended to avoid neuromuscular blockade and maintain a patient ventilating spontaneously. However, others state that control of the airway is best after an intravenous induction with an agent such as propofol and a short acting neuromuscular blocker.[35] The extent and the fixed versus dynamic characteristics of the stenosis can help guide anesthetic choices. Periglottic dynamic collapse can potentially make intubation difficult, and thus, maintenance of muscular tone and spontaneous respiration may be advantageous. Similarly, immediate subglottic stenoses can make seating of an ETT impossible. In these cases, awake sedated bronchoscopy leading to control of the airway is recommended. Laryngeal mask airways (LMAs) can be placed in patients with proximal lesions to allow ventilation/oxygenation and access for fiberoptic bronchoscopy.

If intravenous induction is chosen, the medications are tailored to the patient's health, the type and duration of the surgery, and the type of airway therapy used. Regimens are typical for all critically ill patients. Total intravenous anesthesia (TIVA) with propofol and remifentanil is useful when rigid bronchoscopy is expected (these should be rapidly available in any case). Obtundation of airway reflexes with opioids needs to be balanced against potential CO_2 retention in patients with COPD. Typical doses of 2 to 4 mg/kg fentanyl help to maintain hemodynamic stability throughout the procedure. Titrated doses of propofol, ketamine, or etomidate can then be added. The focus of induction is shifted toward rapid controlling of the airway, because mask ventilation may be difficult with the higher required pressures causing gastric distension. If mask ventilation is attempted or necessary,a slower rate with longer inspiratory and expiratory times is required to allow for adequate tidal exchange.

For midtracheal stenoses, there is often room to seat an ETT cephalad to the stricture. Jet ventilation, either supraglottic or intraglottic, is an option. However, because of its nature, it is hard to raise the concentration of inspired oxygen, and the narrow orifice of the stenosis can dramatically inhibit the flow.

After the induction of anesthesia, passage of an ETT down the airway can be guided by a fiberoptic bronchoscope. An LMA can be placed to facilitate oxygenation/ventilation and bronchoscopic evaluation/treatment of proximal lesions. Regardless of the mode of induction chosen for the patient, an interventional pulmonologist or surgeon who is familiar with a rigid bronchoscope must always be ready to control the airway. In extreme cases of airway obstruction, percutaneous cardiopulmonary bypass may be instituted if airway control cannot be established with a rigid bronchoscope. Anesthesia for stent cases can be maintained using inhalational agent but is more commonly maintained using total intravenous anesthesia and short-acting agents such as remifentanil and propofol.[5,36] Intravenous agents maintain the depth of anesthesia during the

frequent periods of suctioning, airway dilatation, and stenting when ventilation is interrupted. Patients may be ventilated with positive pressure ventilation of various modes that include common tidal volume or pressure control, jet ventilation, and high-frequency ventilation. Regardless of the type of anesthetic used during a case, a patient must be fully awake with intact airway reflexes at the end of the procedure.

Ventilation

As mentioned earlier, understanding the location and type of lesion will guide ventilatory management. Typical midtracheal stenotic lesions from ETT damage will require a decrease in respiratory rate to allow an increase in inspiratory and exhalation times. With higher inspiratory pressures made possible by intubation, it is reasonable that only the exhalation times would need to be lengthened because exhalation is a purely passive process. If hypotension is encountered after the initiation of positive pressure ventilation, differential diagnosis needs to include breath stacking due to obstructed exhalation, along with the more common issues of pneumothorax, drug effect, myocardial ischemia, and so on.

Jet ventilation is often used for diagnostic and therapeutic procedures. If the jet catheter is supraglottic, care must be taken to aim the jet at the lumen of the stenosis to avoid direct trauma to adjacent tissue. If the catheter is passed through the stenotic orifice, hyperinflation must be considered a constant threat. Prolonged jet ventilation will necessitate arterial CO_2 sampling along with standard monitoring.

The Montgomery T tube

The use of the T tube to treat tracheal stenosis was first reported by Dr Montgomery in 1964. Although considered a silicone stent, the Montgomery T tube is a specific device in that it serves both as a tracheal stent and a tracheostomy tube. The proximal (vertical) portion of the T tube extends above the tracheotomy, ending below the vocal cords in the region of the subglottic strictures. The distal (vertical) limb occupies the lumen of the upper and middle third of the trachea beneath the tracheotomy. The external (horizontal) limb protrudes through the tracheotomy site and serves as a portal for achieving pulmonary toilet and as a means of maintaining access to the trachea in case a tracheostomy tube needs to be placed for ventilatory support.[23,37] The T tube can be used in benign and malignant airway diseases and may be custom-tailored to stent across a stricture either via its proximal or distal limb. Like other airway stents, it may play a role as a bridge to definitive reconstruction, as an adjunct to primary surgical repair, or as definitive treatment in patients who cannot undergo surgical repair. Several techniques have been described for the placement of a T tube. Montgomery's original technique involves grasping the distal portion and advancing it to the distal trachea, doing the same with proximal end, and then pulling the extraluminal limb anteriorly. Other techniques were developed because this original technique could be technically challenging. The extraluminal limb can be plugged to preserve phonation and prevent dryness of respiratory mucosa.

Patients with T tubes may need general anesthesia for the assessment of tracheal lesions. This may be challenging for the anesthesia practitioner because ventilatory volume may escape the proximal end, risking hypoventilation, and there is no clear adapter for the extraluminal limb. To overcome the lack of proximal seal, a throat pack can be placed to limit volume loss through the proximal end, and the adapter to a 7 mm ETT can be put into the extraluminal (horizontal) limb. To access the proximal limb, an LMA can be placed. This allows a bronchoscope to be inserted via the LMA or the extraluminal limb. Both TIVA and inhalational anesthesia can be used.

SURGICAL MANAGEMENT

Tracheal Reconstruction

Resection and reconstruction with primary anastomosis may be undertaken in patients who fail dilatation and stent therapy. It is recommended that patients have a functional glottis and that those with neuromuscular disorders or profound pulmonary pathology be optimized so as to not require postoperative ventilation. Patients should not harbor active infection or be on steroids because these can lead to wound dehiscence. In tracheal resections, patients are usually positioned supine with the head of the bed elevated and the neck extended. A low-collar incision is made, and the pretracheal plane is entered and developed by blunt dissection. Distal tracheal lesions may require a partial sternotomy and/or right posterolateral thoracotomy for extended mobilization of the trachea and tracheal release maneuvers. These include the suprathyroid laryngeal release, suprahyoid laryngeal release, and right pulmonary hilar release.

The trachea is freed around its circumference only in the area of the stenosis; this prevents devascularization of the tracheal ends used for anastomosis. The trachea is entered below the area of stenosis and transected. At this time, the patient is ventilated by the intubation of the distal trachea across the operative field. Occasionally, it may be necessary for the patient to undergo brief periods of apnea for the surgeons to work. Alternatively, a jet catheter can be passed either through the endotracheal tube or across the field and into the distal trachea. TIVA is typically used during the resection. The surgical technique used must be tailored to the type and site of the stenosis. The surgeon then forms the anastomosis between the 2 free ends of the trachea. This anastomosis is tested for air leaks by submerging it under saline while pressurizing the airway to 25 to 40 cm of water. Either the air leaks are directly repaired or the anastomosis is restructured. A formal tracheostomy or a ''Mini-Trach'' for secretion management can be placed after closure. A chin-to-chest stitch may be placed to prevent hyperextension and excessive tension on the fresh anastomosis.[38]

Attempts should be made to extubate the patient in the operating room to prevent the fresh anastomosis from being exposed to high airway pressures. In the immediate postoperative procedure, patients may require flexible bronchoscopy to clear secretions or for the examination of the anastomosis. Edema is prevented by limiting the intake of fluids. In some cases of edema, patients may require steroids or reduced-density helium-oxygen mixtures.[5,38] Nebulized racemic epinephrine can also be useful to reduce airway swelling.

Anesthesia for tracheal resection

Much like the anesthesia for placements of stents, anesthesia for tracheal reconstruction surgery requires planning and good communication between the surgeon and the anesthesiologist because they must share control of the airway. A detailed preoperative history should include progression of the disease, history of prior intubations, and any other pertinent symptoms that the patient has experienced. Standard monitors of electrocardiogram, oxygen saturation, blood pressure, and capnography should be used. It is recommended to place an arterial line in the left arm in these patients because their innominate artery may be operatively manipulated or compressed, thus making pressure readings in the right arm inaccurate. The premedication and induction of anesthesia should be approached in the same way as that for pulmonary stent placements. Although no adverse effects were seen with an intravenous induction using neuromuscular blocker, others prefer to maintain a patient ventilating spontaneously until the airway is secured.

Besides induction, the other challenge that the anesthesiology team encounters is how to ventilate an open airway. As described by Pinsonneault and colleagues,[24] 5 different modes of ventilation can be used during these procedures. Manual oxygen jet ventilation or low-frequency jet ventilation requires the anesthesiologist to manually trigger O_2 delivery under high pressure. This form of ventilation allows free access to the surgical field, but it can lead to hypercarbia due to hypoventilation. The lungs can also be ventilated using high-frequency ventilation: high-frequency positive pressure ventilation, high-frequency jet ventilation, or high-frequency oscillation ventilation. High-frequency positive pressure ventilation delivers very small tidal volumes using a ventilator at 1 breath per minute. With high-frequency jet ventilation, pulses of gas at 50 psi are delivered. Air entrainment occurs with high-frequency jet ventilation, leading to a lower concentration of oxygen delivery. High-frequency ventilation results in good gas exchange, decreases the risk of atelectasis secondary to auto–positive end-expiratory pressure, and results in minimal hemodynamic changes. The surgeon also has an unobstructed field with this type of ventilation. No single form of ventilation is preferred, but it is important to discuss and plan how to proceed with ventilating the open airway with the surgeon before the induction of anesthesia.

CASE DISCUSSION

MS was a 57-year-old man who was admitted with increasing shortness of breath and a tracheal stricture found on bronchoscopy by a referring pulmonologist. He had a medical history of hypertension, hypercholesterolemia, coronary artery disease, and peripheral vascular disease with claudication. Two weeks before admission, he underwent peripheral vascular surgery at an outside institution. He had multiple intubation attempts. His postoperative course was benign. He was repeatedly treated for reactive airways disease during his 3-day hospitalization. His shortness of breath rapidly progressed during the ensuing week.

MS presented with complaints or dyspnea. He had audible wheezing on inspiration and exhalation. He appeared to be working hard on inspiration and exhalation. He was receiving nasal cannula oxygen at 6 L/min, with an oxygen saturation of 92%. With a known diagnosis of tracheal stenosis, his therapy was changed to helium 75%/oxygen 25%. Although his arterial oxygen saturation did not change, his work of breathing and his complaints of dyspnea decreased. Physical examination at this time revealed mild inspiratory and expiratory stridor greatest over the sternal notch and transmitted to the distal airways. It increased greatly in volume when the patient was taken off the oxygen and placed on nasal cannula. A CT scan with 3D reconstruction (**Figs. 2** and **3**) indicated a narrowing and an anterior shift of narrowed segment. Such a shift in the airway configuration as a result of the fibrosis can cause turbulent airflow and increased work of breathing even in the absence of narrowing. Further, such a change in the axis of the airway can make airway control more difficult because the endotracheal tube may not be able to follow the serpentine airway.

MS was brought to the operating room for initial evaluation with awake flexible fiberoptic bronchoscopy and dilatation. His nasal and oral airways were topicalized in standard fashion. **Fig. 4** is the preinterventional bronchoscopy; this shows 2 areas of narrowing and the nonlinear nature of the trachea. An initial management plan of serial dilatations with rigid bronchoscopy for several weeks was planned with close follow-up to determine if stenosis recurred and resection was needed. For this procedure, the patient was induced and an LMA placed. An LMA was chosen because the authors were uncertain if there was sufficient room for the endotracheal tip and cuff between the vocal cords and the stenosis. Neuromuscular blockade was chosen to allow

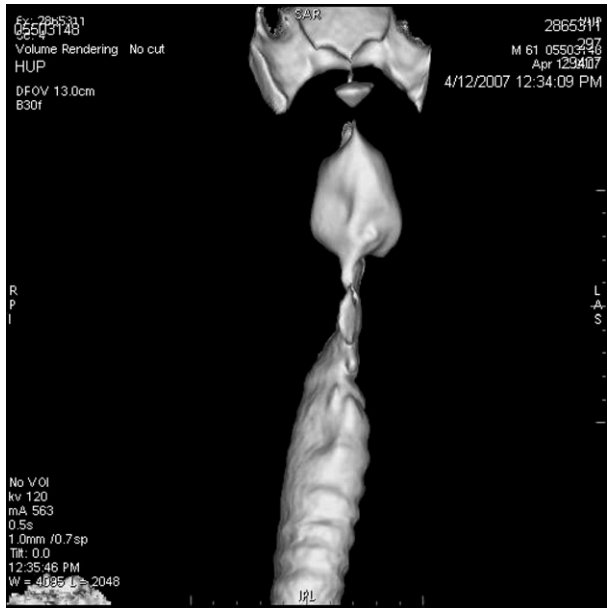

Fig. 2. A 3D reconstructed axial image of a patient with subglottic tracheal stenosis.

a slow respiratory rate (prolonged inspiratory and exhalation time), without the hypotension associated with the depth of anesthesia necessary for lack of spontaneous ventilation. Further, the authors desired paralysis so that there would be no risk of negative pressure pulmonary edema when the airway was obstructed with the dilating

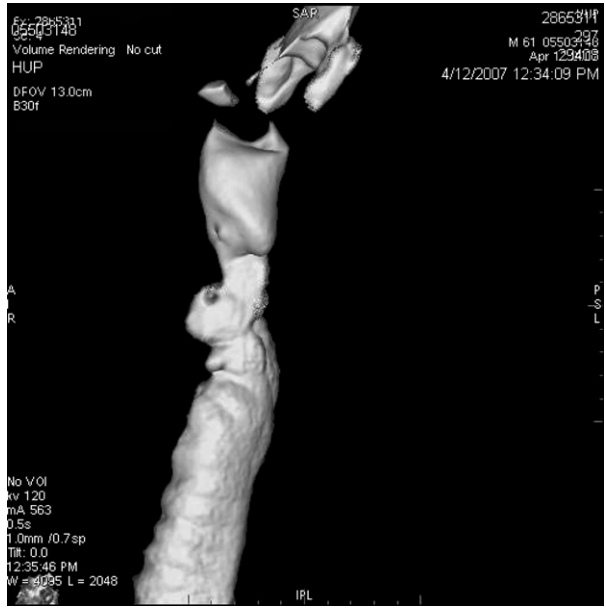

Fig. 3. A 3D reconstructed sagittal image of a patient with subglottic tracheal stenosis.

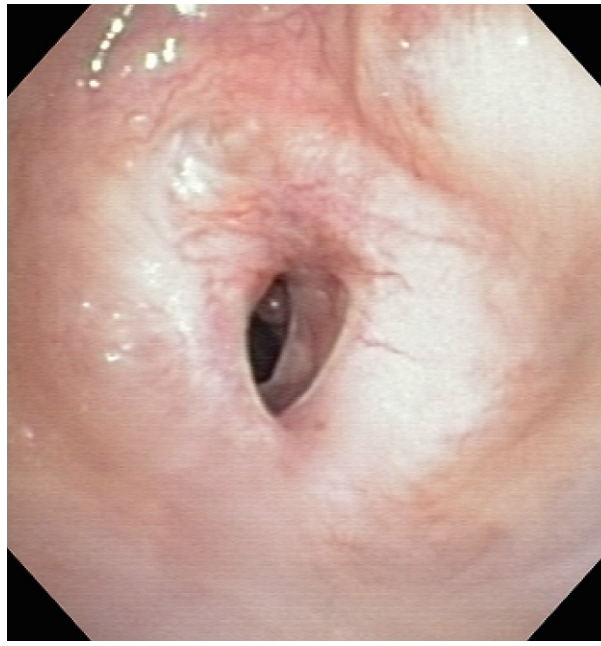

Fig. 4. Bronchoscopic view of tracheal stenosis seen in **Figs. 2** and **3**. The location of the stenosis and its smooth and circumferential presentation suggest an injury from a tracheal balloon.

balloon. The operating room was set up for immediate rigid bronchoscopy should airway compromise occur. The patient received intravenous steroids to prevent or minimize airway edema.

The patient was maintained on 30% oxygen for airway cautery. The stenotic area was incised in 4 places with cautery (3, 6, 9, and 12 o'clock). The patient was then switched to 100% oxygen in preparation for dilatation. Three serial dilatations with a saline-filled balloon resulted in dramatically improved ventilation. The patient was then switched to TIVA for rigid bronchoscopy. Serial dilatation with 5 mm, 6 mm, and 7 mm (internal diameter) rigid bronchoscopes was undertaken. Ventilation occurred by coaxial jet ventilation. After hemostasis was achieved and the airway was suctioned, the rigid scope was removed and an LMA was placed for final flexible bronchoscopy and termination of anesthesia.

In the recovery room, the patient was immediately treated with racemic epinephrine. He was discharged home on a 5-day Solu-Medrol dose pack. The patient remained well and 1 week later returned for a repeat bronchoscopy (**Fig. 5**). The patient was dilated again. But the patient's symptoms and stenosis recurred, although much less than initial presentation, and a definitive tracheal resection was planned.

For the formal tracheal resection, general anesthesia was induced and an LMA was placed. The surgeons repeatedly measured the distance from the carina to the distal end of the stenosis, the length of the stenosis, and the distance from the proximal end of the stenosis to the vocal cords. This enabled the surgeons to determine how many tracheal rings would be resected and the type, if any, of tracheal release that would be done. An arterial line was placed and the trachea was intubated with a 5-mm ETT. The arms were tucked and padded.

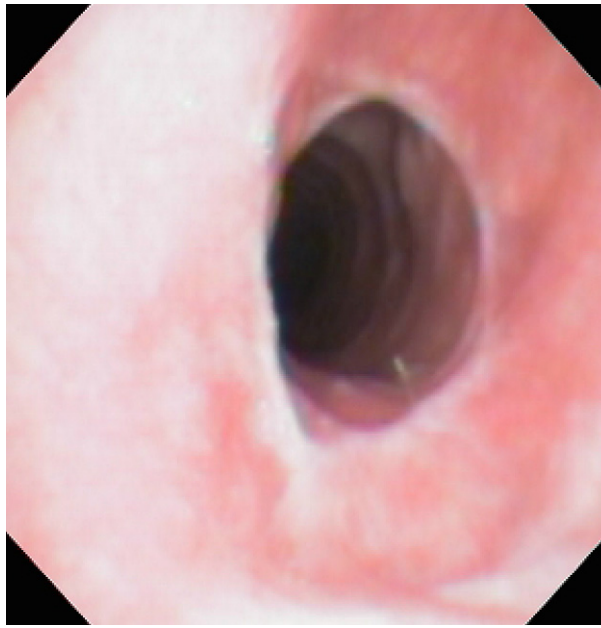

Fig. 5. The stenosis from Fig. 4 1 week after endobronchial therapy. The invasive pulmonologists had made a series of radial cuts into the stenosis to control the tearing when they balloon dilated.

After the trachea was exposed, the bronchoscope was placed into the ETT and slowly withdrawn to the proximal stenosis. Under bronchoscopic view, a needle was placed into the trachea to give an external landmark for the distal end of the stenosis. The distal trachea was divided and TIVA was started. Ventilation was by a 6-mm anode ETT, and a sterile ventilator circuit passed off the field. The transoral 5-mm ETT was retracted and left in place below the vocal cords. Once the stenotic lesion was excised, a jet ventilation catheter was placed through the transoral ETT and into the distal trachea. The narrow diameter of the catheter allowed the surgeon the access to the posterior wall of the trachea for reconstruction. During this portion, the authors were concerned for any arrhythmias as a result of rising arterial CO_2 concentration. If this were to occur, the transoral ETT would have been guided into the distal trachea by the surgeon for tidal ventilation, whereas other causes of instability were ruled out.

Once the posterior wall of the trachea was reapproximated, the transoral ETT was advanced across the suture line until the balloon was distal to the suture line. Tidal ventilation was resumed, and the trachea and neck were closed. Because the trachea was shortened by 2 rings, the patient's neck was flexed to remove tension. A stitch was placed from the patient's chin to the chest to guard the patient against extending their head. The ETT was removed, and the LMA was placed instead. Bronchoscopy showed a widely open tracheal lumen (**Fig. 6**). However, if at this point there was a question about the repair or a concern about the patient's ability to cough and control airway secretions, a tracheostomy or a minitracheostomy would have been placed.

The back of the operating room table was raised and pillows were used to reinforce the neck's flexed position. Spontaneous ventilation was allowed, and the LMA was

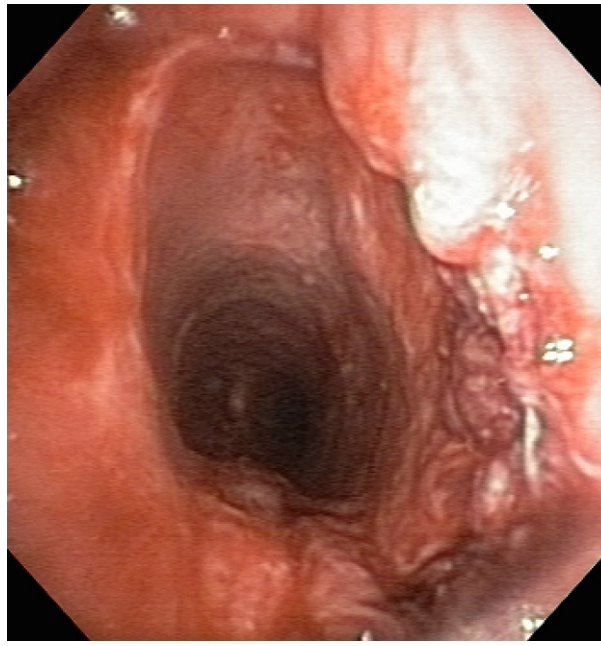

Fig. 6. Because the therapy from Fig. 5 was temporary and the patient suffered a recurrence of stenosis, they had tracheal surgery with resection of 2 tracheal rings and primary reanastomosis. The carina is visible in the distance. There is some granulation tissue along the right suture margin that did not progress to requiring therapy.

removed. Once in the recovery room, the patient received a nebulized racemic epinephrine treatment. The patient was transferred to a high-acuity floor with respiratory rate checks every 15 minutes overnight.

SUMMARY

Tracheal stenosis may occur secondary to trauma, tumors, infection, inflammatory diseases, or iatrogenic causes. Understanding these lesions requires a basic understanding of the physics of airflow. All of these patients must be carefully evaluated and require a series of tests, including pulmonary function tests and radiographic studies. Treatment of tracheal lesions is a multidisciplinary issue and requires the close participation of interventional pulmonologists, anesthesiologists, and surgeons.

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