Late Complications of Tracheostomy

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Tracheostomy may be associated with numerous acute, perioperative complications, some of which continue to be relevant well after the placement of the tracheostomy. A number of clinically important unique late complications have been recognized as well, including the formation of granulation tissue, tracheal stenosis, tracheomalacia, tracheoinnominate-artery fistula, tracheoesophageal fistula, ventilator-associated pneumonia, and aspiration. The clinical relevance of these complications is considerable, as their manifestations range from minimally symptomatic to failure to wean from the ventilator (tracheal stenosis) to life-threatening hemorrhage (tracheoinnominate fistula). Treatment modalities vary depending upon the nature of the complication. For the most frequent complication, tracheal stenosis, a multidisciplinary approach utilizing bronchoscopy, laser, airway stents, and tracheal surgery is most effective. *Key words: tracheostomy, complications, mechanical ventilation, weaning, extubation, upper-airway obstruction, tracheomalacia, tracheoinnominate artery erosion, tracheoesophageal fistula, tracheal stenosis.* [Respir Care 2005;50(4):542–549. © 2005 Daedalus Enterprises]

Introduction

Tracheostomy can be associated with numerous acute, perioperative complications, including hemorrhage, infection, pneumothorax, tube obstruction, and accidental decannulation.[1,2] Some of these complications continue to be relevant well after the placement of the tracheostomy. A number of clinically important unique late complications have been recognized as well (Table 1). The most frequent late complication is the development of granulation tissue, a complication that can be subclinical or may present as failure to wean from the ventilator, failure to decannulate, or may manifest as upper-airway obstruction with respiratory failure after decannulation. Granulation tissue may cause airway occlusion or result in airway stenosis. The other listed complications occur less frequently, but each can be associated with considerable morbidity and mortality.

Assessing the prevalence of late complications of tracheostomy may be particularly challenging. Late complications can occur in up to 65% of patients.[3–5] Because these patients are severely ill with other medical conditions (including multiple organ failure), tracheal compli-

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Table 1.   Late Complications of Tracheostomy

Tracheal stenosis (granulation tissue)
Tracheomalacia
Tracheoinnominate-artery fistula
Tracheoesophageal fistula
Pneumonia
Aspiration

cations may not be readily detectable. Indeed, the extent of airway injury may be greatest for patients with organ failure.[6] In addition, the presence of shock may further compromise mucosal blood flow and lead to airway mucosal ischemia.[7] In addition, follow-up for complications may not occur when patients die in the acute intensive care unit or are lost to follow-up as they transfer to a long-term acute-care facility. Complications may not be appreciated when they fail to yield clinically detectable symptoms. Lastly, it is frequently difficult to separate out the effects of preceding translaryngeal endotracheal intubation on complications.[4] As an example, prolonged endotracheal intubation may itself lead to either tracheal stenosis or tracheomalacia. Indeed, Whited studied 200 patients with prolonged intubation and found an increasing incidence and severity of acute and chronic laryngotracheal stenosis.[8] Moreover, tracheal stenosis may be more common when tracheostomy follows prolonged intubation, possibly a result of tracheostomy introducing bacteria and thereby aggravating chondritis and mucosal and submucosal necrosis and mucosal ulceration.[4,9,10]

Increasingly tracheostomies are placed using the percutaneous dilational approach. In general, most studies have demonstrated a low incidence of long-term complications with the percutaneous approach.[11–14] A meta-analysis of trials (1985–1996) comparing surgical to percutaneous tracheostomy found more frequent perioperative complications with the percutaneous approach but more postoperative complications with surgery.[15] Freeman et al examined 5 studies ($n = 236$ patients) and found that, compared to surgical tracheostomy, percutaneous tracheostomy was associated with a lower overall postoperative complication rate, including less bleeding and less stomal infection, perhaps related to the snug fit of the stoma around the tube.[16] In contrast, Norwood et al used fiberoptic bronchoscopy and computed tomography (CT) to analyze a cohort of patients who underwent percutaneous dilational tracheostomy.[17] Thirty-one percent of patients were found to have > 10% tracheal stenosis. Symptomatic tracheal narrowing was detected after decannulation in 6% of patients. Melloni et al compared surgical and percutaneous (using bronchoscopic guidance) approaches and provided 6-month follow-up.[18] Early postoperative complications were more frequent in the surgical group. Late complications occurred in 2 percutaneous dilational tracheostomy patients (one

with malacia and one with stenosis) and no surgical patients. In 2 prospective randomized controlled trials, complications (including tracheal stenosis and stomal infection) were less likely to occur than in patients undergoing surgical tracheostomy.[11,19]

A number of mechanisms can cause late complications after tracheostomy.[7] Complications can be directly related to placement of the tube, leaving the tube in place for a prolonged period of time, or abnormal healing at the site of injured tracheal mucosa.[4] As with a translaryngeal endotracheal tube, complications may be related to the inflated cuff of the tracheostomy tube or the tip of the tube, especially when it impinges on the posterior tracheal wall. In contrast, the tracheostomy stoma leads to a unique set of airway complications. Once airway injury occurs, other factors may serve as exacerbating factors. As an example, chemical injury from either gastroesophageal reflux or laryngopharyngeal reflux can aggravate the extent of damage of an already injured airway.[20,21] Pooling of inflammatory secretions above the tracheostomy cuff can further injure the airway.

Tracheal Stenosis

Tracheal stenosis, an abnormal narrowing of the tracheal lumen, most commonly occurs at the level of the stoma or above the stoma (suprastomal) but below the vocal cords (subglottic).[4,7,22] Tracheal stenosis may also occur at the site of tracheostomy tube cuff or at the site of the tube's distal tip. Stomal stenosis develops secondary to bacterial infection and chondritis, which conspire to weaken the anterior and lateral tracheal walls. Stomal granulation tissue frequently develops, and nearly all patients have some degree of tracheal narrowing at the site of the tracheostoma. In contrast, only 3–12% demonstrate clinically important stenosis that ultimately requires intervention.[22] This granulation tissue often starts at the cephalad aspect of the stoma and initially is soft and vascular. Indeed, it may cause substantial bleeding at the time of tube exchanges. Granulation tissue may obstruct the airway at the level of the stoma and cause difficulty in replacing the tracheostomy tube if accidental decannulation occurs. Alternatively, this granulation tissue can occlude tube fenestrations and lead to difficulty with successful decannulation. Subsequently, as the granulation tissue matures it becomes fibrous and covered with a layer of epithelium. With the development of fibrosis, stenosis develops as the anterior and lateral aspects of the tracheal wall become narrowed at the level of the stoma.[22] Multiple risk factors are associated with stomal stenosis, including sepsis, stomal infection, hypotension, advanced age, male sex, steroids, tight-fitting or oversized cannula, excessive tube motion (ie, mechanical irritation), prolonged placement, and dis-
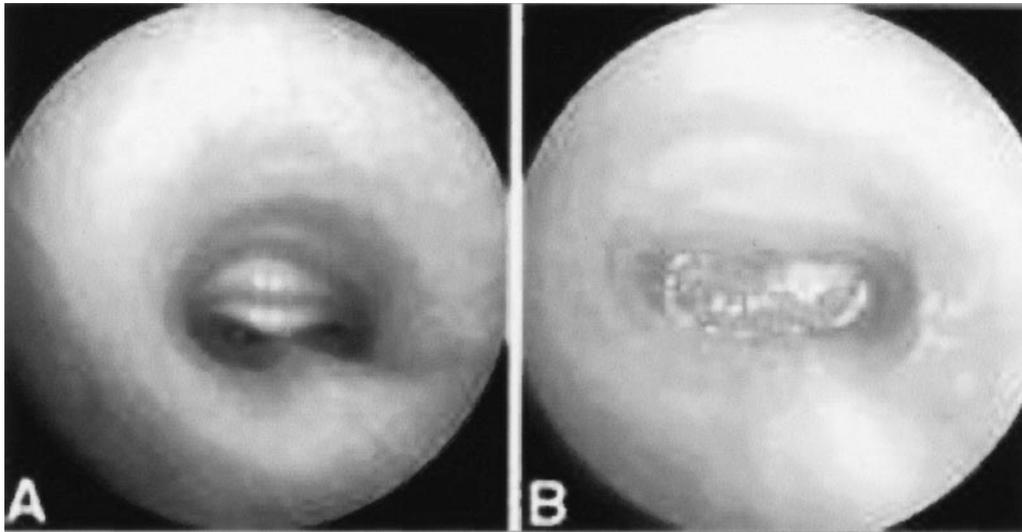
Fig. 1. Panel A shows the invagination of cartilage rings, causing tracheal obstruction after percutaneous dilational tracheostomy. Panel B shows granulation tissue originating from the upper aspect of the tracheostoma. (From Reference 25, with permission.)

proportionate excision of anterior tracheal cartilage during the creation of the tracheostomy.[4]

Suprastomal stenosis has recently been reported, particularly as a complication of percutaneous dilational tracheostomy.[23–25] Suprastomal injury may occur when there is guidewire-related injury to the posterior tracheal wall and with subsequent development of granulation tissue. In a study of 24 patients undergoing percutaneous dilational tracheostomy, 3 suffered substantial injury to the posterior wall, possibly related to instability of either the guidewire or the guiding catheter.[26] Another report noted intermittent obstruction of a percutaneously placed tracheostomy, related to swelling and/or hematoma of the posterior wall.[27] Additionally, the dilators used to enlarge the percutaneous dilational tracheostomy stoma can injure the anterior tracheal cartilage (including tracheal ring fracture), causing these deformed structures to invaginate and protrude into the tracheal lumen, causing obstruction (Fig. 1).[23,25,28–30] In a retrospective study of patients undergoing percutaneous tracheostomy (Griggs technique), 12 of 19 patients (63%) were found to have > 10% tracheal stenosis, using endoscopic diagnostic techniques (2 had > 25% tracheal stenosis).[31] In 7 patients the cricoid cartilage was affected by the percutaneous dilational tracheostomy. In contrast, tracheal stenosis seems less common when the Ciaglia approach is used.[12,32] Importantly, in a series of 3 cases of substantial suprastomal obstruction after placement of a percutaneous dilational tracheostomy, the trachea was found to be 50% obstructed in one patient and completely obstructed in 2 others.[25]

The third location for tracheal stenosis is at the site of the tracheal-tube cuff, where ischemic injury to the tracheal mucosa can take place. This occurs when cuff pressure exceeds the perfusion pressure of the capillaries of the tracheal wall. Shearing forces from the tube or the cuff may further aggravate injury to the airway. The incidence of cuff (infrastomal) stenosis has fallen 10-fold with the change from low-volume high-pressure to high-volume low-pressure cuffs.[33,34] Unfortunately, overinflation of a high-volume low-pressure cuff can lead to ischemic airway injury. With prolonged ischemia, mucosal ulceration, chondritis, and cartilaginous necrosis may ensue, leading to the formation of granulation tissue. The process is exacerbated by the presence of pooled secretions or gastroesophageal reflux disease.[35] The end result is fibrous narrowing and circumferential stenosis. Risk factors for the development of cuff-site stenosis include female sex, older age, prolonged tube placement, and excess cuff pressure.[4,7]

Finally, stenosis may occur near the distal tip of the tracheostomy tube. Depending on the positioning of the tube, the tip may rub against either the anterior or the posterior tracheal wall. The latter may occur because standard tracheostomy tubes may be too short in patients with abundant soft tissue in the anterior neck. The resulting injury to posterior membranous wall may lead to stenosis or to tracheoesophageal fistula (see below).

The most important aspect in diagnosing tracheal stenosis is for the clinician to have a high index of suspicion, especially when a patient with a history of previous intubation or tracheostomy is under evaluation. Indeed, this may explain why diagnosis of tracheal stenosis is often delayed. Tracheal stenosis may present early—that is, while the patient is still undergoing mechanical ventilation. For example, the presentation may be that of a patient who cannot wean from mechanical ventilation or who cannot be decannulated. Alternatively, tracheal stenosis may present as "unexplained" dyspnea weeks to months after decannulation. In a recent study, symptoms developed < 6
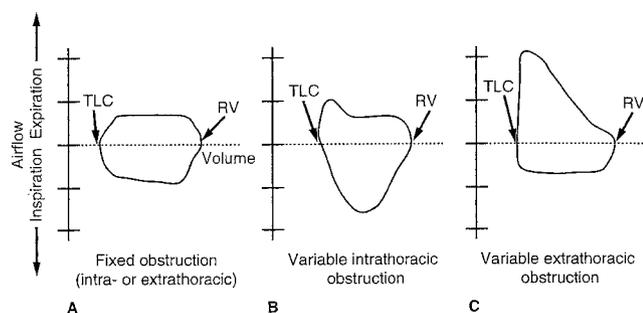
Fig. 2. Flow-volume loops. Panel A shows the pattern of inspiratory and expiratory flow limitation characteristic of fixed upper-airway obstruction (tracheal stenosis). Panel B shows the expiratory flow limitation characteristic of variable intrathoracic upper-airway obstruction, as seen with tracheomalacia. Panel C shows inspiratory flow limitation characteristic of variable extrathoracic upper-airway obstruction. TLC = total lung capacity. RV = residual volume. (From Reference 36, with permission.)

weeks after extubation in more than half of the patients and in < 2 months in two thirds of the patients.[24] In 2 patients, stenotic symptoms occurred very late after extubation: 6 and 18 years, respectively.

Tracheal stenosis may produce no symptoms until the lumen has been reduced by 50–75%.[4] The initial manifestations may be increased cough and difficulty clearing secretions. Once the tracheal lumen has been reduced to ≤ 10 mm, exertional dyspnea occurs. When the lumen is narrowed to ≤ 5 mm, dyspnea at rest or stridor is noted. There are several ways to image the tracheal air column, including chest radiography, tracheal tomography, CT, and magnetic resonance imaging. Yet most patients ultimately undergo either laryngotracheoscopy or bronchoscopy to define the exact site of stenosis, the cause of stenosis, and the length of the involved trachea. In spontaneously breathing patients, the flow-volume loop can show evidence of upper-airway obstruction (Fig. 2). With most forms of tracheal stenosis, there is limitation of both inspiratory and expiratory flow, producing a rhomboidal shape to the curve.

As noted earlier, tracheal stenosis can occur while the patient remains mechanically ventilated. Rumbak et al conducted a retrospective study of 756 patients at a long-term care facility who had been ventilated for at least 15 weeks (3 weeks with an endotracheal tube followed by 12 weeks with a tracheostomy tube).[37] Thirty-seven patients (5%) developed failure to wean secondary to tracheal stenosis or obstruction from granulation tissue, often manifested as higher peak airway pressures or difficulty in passing a suction catheter. Intervention (a longer tube in 34 and airway stenting in 3) led to successful weaning in 34 of 37 patients within 1 week. This study raises the question of whether all patients should undergo bronchoscopic investigation of the trachea prior to tracheal-tube capping or decannulation. In fact, one such study found a high incidence of tracheal granulomas.[38] Yet, the clinical impor-

tance of such lesions is debatable. One way of identifying patients more likely to have substantial stenosis is to restrict evaluation to patients having trouble tolerating tracheostomy-tube capping. As an example, in one study, > 50% tracheal obstruction was detected in a cohort of patients not tolerating 30 min of tracheostomy-tube capping.[39]

Prevention of stomal stenosis starts with limiting the size of the tracheal defect created during surgery. Using a vertical excision of the second and third tracheal rings and avoiding excessive removal of tracheal cartilage can decrease the risk for stenosis.[22] Further efforts to prevent the formation of granulation tissue have centered on strategies to avoid excess mechanical irritation, placement of tubes of proper size, and the use of swivel adapters and ventilator tubing support (thereby preventing abnormal traction on the stoma). Prevention of stomal infection is likely to be important, because infection may impair tissue healing. Meticulous stomal care has been recommended to prevent bacterial contamination.[9] In one animal study, animals receiving prophylactic antibiotics had less severe stenosis than control animals.[40] Alternatively, early tracheostomy-tube changes may keep the stoma clean.[41] A recent observational study conducted in a long-term acute-care facility examined the incidence of clinically important granulation tissue 1 year before and 2 years after the implementation of a new tracheal-tube exchange policy.[42] The latter consisted of careful stomal assessment and changing the tube every 2 weeks. In the year prior to implementation, 7 patients required laser excision of granulation tissue, compared to 5 in the 2 years after institution of the new policy.

The development of high-volume low-pressure tracheostomy-tube cuffs has led to a 10-fold reduction in cuff-site stenosis.[3,7,43] Nevertheless, overinflation can convert these cuffs to high-pressure ones that have the potential to cause ischemic tracheal mucosal injury. One way to avoid overinflation is to ensure that the proper size tube has been used (eg, tubes that are too small may predispose to cuff overinflation). Therefore, it is important to carefully monitor cuff pressure to ensure that this remains below 20 mm Hg. This may entail allowing the patient to have a small air leak around the cuff. It is crucial to use a 4-way stopcock during the maneuver, to allow pressure to be measured *during* cuff inflation. If pressure is measured with a manometer attached to an already-inflated cuff, the cuff pressure may be underestimated.[22,33]

A number of therapeutic approaches have been applied to patients who have granulation tissue (Table 2). It is important to note that these therapeutic options have not been subjected to rigorous randomized controlled trials. In symptomatic patients, neodymium-yttrium-aluminum-garnet (Nd-YAG) laser excision, with or without rigid bronchoscopic dilation, may be the preferred approach.[44] Although using a rigid bronchoscope to "core" out or dilate

LATE COMPLICATIONS OF TRACHEOSTOMY

Table 2. Treatment Strategies for Granulation Tissue

Topical Strategies
    Antibiotic cream
    Steroid cream
    Silver nitrate
    Inhaled steroids
    Combination of antibiotics, antifungals, and steroid powder
    Polyurethane form dressings
Surgical Strategies
    Bronchoscopy with either CO_2 or YAG laser excision
    Bronchoscopy with excision via stoma
    Electrocautery
    External exploration

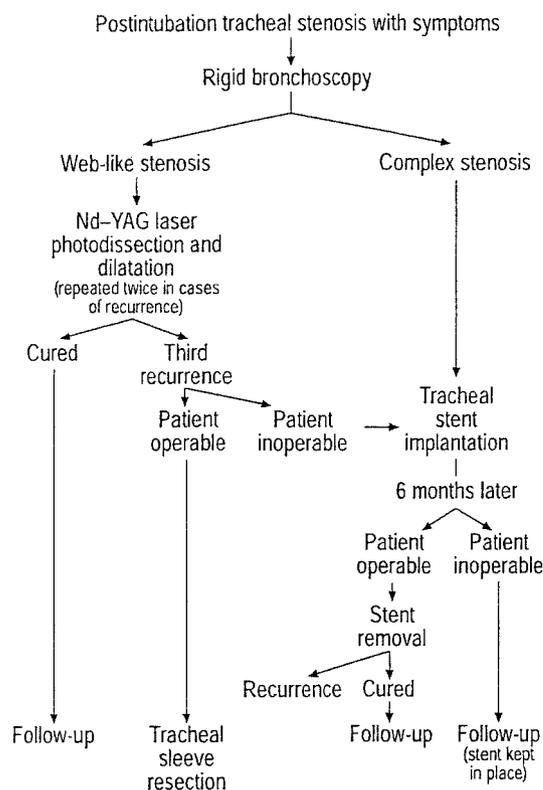YAG = yttrium-aluminum-garnet (Adapted from Reference 41.)

Fig. 3. Multidisciplinary algorithm for managing patients with post-tracheostomy tracheal stenosis. Nd-YAG = neodymium-yttrium-aluminum-garnet. (From Reference 24, with permission.)

(using a balloon) an obstruction may be effective when used alone, the recurrence rate is up to 90% with lengthy stenoses.[45] Suprastomal granulation tissue can be excised by sharp dissection under bronchoscopic guidance.[46] When a short, web-like band of tracheal stenosis exists, laser excision can prove useful in reestablishing an adequate tracheal lumen, with success rates as high as 60% after 1–3 sessions.[47] Even with laser resection, granulation tissue and stenosis can recur. In cases where laser resection is not feasible, stenting of the airway or surgical repair (tracheal sleeve resection) are the next best options. In the latter approach, the stenotic area is surgically excised, followed by reanastomosis of the remaining elements of the trachea.[22,24,48–53]

A recent European study examined the use of a multidisciplinary, protocolized approach to treating tracheal stenosis (Fig. 3).[24] The diagnosis are first made by rigid bronchoscopy. If a web-like stenosis is detected, laser excision and bronchoscopic dilation are employed. If this approach proves ineffective and the patient is a good surgical candidate, tracheal resection is performed. If the patient is not a good surgical candidate or if the tracheal stenosis is long and complex, a tracheal stent is placed. In some instances, after eventual removal of the stent, the tracheal lumen remains patent. In others, the stenosis persists, necessitating "permanent stenting" (in a nonoperative candidate) or tracheal resection. With this approach in mind the investigators carried out a prospective validation in a small cohort of patients ($n = 32$, 15 with web-like stenosis, 17 with complex stenoses). Interestingly, one third of patients could be managed by laser excision, bronchoscopic dilation, or tracheal stenting (ie, surgery was not required).

Tracheomalacia

Tracheomalacia, or a weakening of the tracheal wall, results from ischemic injury to the trachea, followed by chondritis and subsequent destruction and necrosis of supporting tracheal cartilage.[4,5,54] With the loss of airway support, the compliant tracheal airway collapses during expiration. This can result in expiratory airflow limitation, air trapping, and retained respiratory secretions. In addition, with a loss of cartilaginous support, the trachea may also be compressed by other surrounding structures. In the acute setting, tracheomalacia may present as failure to wean from mechanical ventilation. Alternatively, it may present as dyspnea in a patient with a history of previous tracheostomy.

As with tracheal stenosis, timely diagnosis of tracheomalacia depends on a high index of suspicion. In the patient on mechanical ventilation, bronchoscopy can reveal excessive expiratory collapse of the trachea. Here the best therapeutic approach is to place a longer tracheostomy tube (ie, to bypass the region of expiratory collapse) or a tracheal stent. In the spontaneously breathing patient, the flow-volume loop will show evidence of variable intrathoracic obstruction (see Fig. 2). Another approach is the use of dynamic CT scan images of the trachea, which can depict expiratory tracheal collapse.[55]

The treatment of tracheomalacia depends upon the severity of expiratory upper-airway obstruction.[54] In mild cases, a very conservative approach may be best. In con-

trast, with more severe cases, therapeutic options include placement of a longer tracheostomy tube, stenting, tracheal resection, or tracheoplasty.

Tracheoinnominate-Artery Erosion

One of the most feared complications of tracheostomy is the development of a tracheoinnominate-artery fistula.[4] Risk factors for the development of tracheoinnominate fistula include excessive movement of the tracheostomy, high-pressure (or overinflated) cuff, or a tube that has been placed too low. The innominate artery lies adjacent to the trachea and crosses that structure at approximately the 9th tracheal ring. If the tracheostomy tube is placed too low, below the 3rd tracheal ring, the inferior concave surface of the cannula may erode into the artery. Alternatively, an overinflated tracheostomy cuff balloon or the tip of the tracheostomy tube can severely damage the tracheal mucosa, leading to necrosis and eventual erosion into the innominate artery. This complication occurs in less than 1% of all patients undergoing tracheostomy. The vast majority of cases (approximately 75%) will occur within 3–4 weeks of tracheostomy placement. The mortality rate approaches 100%, even when surgical intervention is undertaken. The most common clinical presentations are bleeding around the tracheostomy tube or massive hemoptysis.

Because of the extraordinarily high mortality associated with this condition, the best treatment is avoiding the complication in the first place. It is therefore recommended that one avoid prolonged or extreme hyperextension of the neck. Furthermore, using lightweight tubing to avoid excessive downward pulling of the tube is also recommended. Treatment of active bleeding from tracheoinnominate fistula includes emergency digital or tube-cuff compression of the fistula to achieve hemostasis and allow for transport to the operating room for immediate surgical repair.[56] The surgical approach consists of interrupting the innominate artery which, if successful, is associated with a low risk of rebleeding.[57]

Tracheoesophageal Fistula

A relatively unusual complication, occurring in less than 1% of patients undergoing tracheostomy, is the development of a connection between trachea and esophagus, a tracheoesophageal fistula.[58] This is an iatrogenic complication resulting from injury to the posterior tracheal wall. Tracheoesophageal fistula can occur because of a perforation of the posterior tracheal wall during placement of a percutaneous tracheostomy. Alternatively, excessive cuff pressure or the tip of the tracheostomy tube can cause posterior tracheal wall injury. The presence of a nasogastric tube, and resulting esophageal injury, may also contribute to the development of this complication.[5,59] Tra-

cheoesophageal fistula may manifest as the copious production of secretions. Additional manifestations include recurrent aspiration of food, increasing dyspnea, a persistent cuff leak, or severe gastric distention (as air moves from the respiratory side to the stomach via the fistula).[4] Modalities used to make the diagnosis include barium esophagography or CT scan of the mediastinum. Treatment includes placement of a double stent (in esophagus and trachea) in nonoperative patients or surgical repair in patients capable of tolerating thoracic surgery.[59–61]

Pneumonia

Older studies suggested that tracheostomy might reduce the incidence of ventilator-associated pneumonia. In a meta-analysis of 3 prospective trials totaling 289 patients, the relative risk of pneumonia was 0.88 (0.71–1.1).[62–64] In contrast, Ibrahim et al prospectively studied more than 3,000 medical and surgical intensive-care-unit patients at a nonteaching community hospital. Twenty-eight percent of these patients were mechanically ventilated and 15% of those developed ventilator-associated pneumonia.[65] Using a multiple logistic regression model, the authors found that the presence of a tracheostomy was powerfully associated with the development of pneumonia, having an odds ratio of 6.7. Other factors associated with ventilator-associated pneumonia included reintubation, multiple central line insertions, and the use of antacids.

In a retrospective study, Georges et al investigated 137 patients undergoing tracheostomy.[66] Ventilator-associated pneumonia occurred in 26%, and in half of these it was diagnosed within 5 days of tracheostomy placement. Interestingly, major risk factors for the development of ventilator-associated pneumonia were fever and tracheal bacterial colonization at the time of the procedure. It is unclear if instituting antibiotic therapy and delaying tracheostomy in such patients will result in reduced risk for pneumonia.

Aspiration

Placement of a tracheostomy tube disrupts swallowing and thereby predisposes to aspiration. This can occur if overinflation of the tracheostomy tube cuff leads to compression of the esophagus. Elpern et al used video fluoroscopic analysis of a barium swallow and found that 50% of 83 patients undergoing prolonged mechanical ventilation (mean duration of tracheostomy 112 d) had evidence of aspiration with the cuff inflated.[67] In 77% of these cases the patients were asymptomatic; that is, the aspiration was silent. Risk was greatest for older patients. Tolep et al found similar results: 50% of tracheostomized patients had clinical aspiration, while 83% aspirated by video fluoroscopy.[68] Schonhofer et al examined a cohort of tracheostomized patients who were difficult to wean from mechan-

ical ventilation.[69] These 62 patients were on average 64 years old, had been ventilated for 49 days, and 61% had chronic obstructive pulmonary disease. With the cuff deflated, 30% had evidence for either clinical or subclinical aspiration. In a study conducted in an acute-care unit, 33% of 52 patients (duration of tracheostomy < 2 months) had evidence for aspiration by endoscopy; in 82% of these cases the aspiration was clinically silent.[70] Based on the high prevalence of swallowing disorders, frequently clinically silent, it is recommended that a formal swallowing evaluation be conducted in all tracheostomized patients in whom oral nutrition is contemplated.

Summary

Tracheostomy is associated with numerous late airway complications. The most common, tracheal stenosis, can be asymptomatic, limit weaning from mechanical ventilation, or present as post-decannulation dyspnea. Meticulous care of the airway may reduce the incidence. For patients with symptomatic stenosis, a multidisciplinary approach is recommended. Other complications are less common, but, in the case of tracheoinnominate fistula and tracheoesophageal fistula, can be associated with considerable morbidity and mortality. Further investigation is warranted to better define the most effective strategies for preventing late complications of tracheostomy.

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