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CHAPTER 79

Laryngotracheal Injury from Prolonged Tracheal Intubation

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FREQUENCY AND TYPES OF COMPLICATIONS

The complications of airway intubation are inevitable, as suggested by a study of 1000 patients extubated after standard operative intervention demonstrating a 6% rate of severe lesions including hematoma, mucous membrane laceration, and subluxation of the arytenoid cartilages.[1] Diabetic females and patients with burns are especially predisposed to injuries from prolonged intubation.[2,3] This higher prevalence in females is demonstrated by another study in which both laryngeal dysfunction (48% vs. 18%) and structural lesions (23% vs. 13%) occurred more commonly in women than in men perhaps because of the tracheal tube size[4] and the narrower larynx in women.[5]

Mucosal ulcerations are common and are usually found on the posteromedial areas of the vocal cords, over the arytenoid cartilages, and posteriorly in the larynx in the interarytenoid area and over the cricoid plate (Fig. 79–1*A*). These lesions occur even after short duration of intubation and are caused by pressure from the tracheal tube. Most resolve spontaneously,[6] but laryngeal granulomas and scarring stenoses are not infrequently formed (see Fig. 79–1*B*). The late lesions associated with endotracheal intubation are primarily laryngostenosis of the glottic and subglottic regions, whereas tracheostomy may result in tracheostenosis. Observed lesions are related to duration of intubation and the presence of chronic conditions such as diabetes mellitus, atherosclerotic heart disease, and immunosuppression.[7] The complication rate in healthy, young, head-injury patients was as high as 61% for prolonged intubation in one study but only 20% for early tracheostomy.[7] Pediatric head-trauma patients, on the other hand, demonstrated a higher complication rate with tracheostomy (26%) than orotracheal (10%) or nasotracheal (11%) intubation.[8] Using long-term artificial airway, adult patients with closed head injury have a greater complication rate, mainly sinusitis, vocal cord paralysis, laryngotracheostenosis, and tracheomalacia.[9] The overall complication rate is perhaps highest in those with severe medical illness, equivalent for prolonged intubation and tracheostomy in incidence of laryngotracheal injury (95% vs. 91%) but more common than tracheostenosis in those with tracheostomy (19% vs. 65%).[10]

The complication rate of prolonged intubation is related to duration. For those intubated for periods of 1 week or less the rate was 37%, whereas for those intubated longer than 1 week the complication rate was 52%.[11] These complications may be as subtle as hoarseness and are found in even larger proportion (77%) in those receiving tracheostomy after a period of prolonged intubation.[11] Problems reported by patients are diverse and include dysphonia (57%), aspiration (25%), dysphagia (23%), odynophagia (21%), dyspnea (21%), stridor (17%), and hoarseness (14%).[12]

Adverse sequelae of endotracheal intubation and tracheostomy may be delineated in incidence (i.e., common vs. rare) (Table 79–1). Infection is often encountered during prolonged intubation. Most of these problems occur in the first 2 weeks and more often with nasal (40%) than oral (20%) intubation.[13] Predisposition is established by the presence of a tracheal tube; the cuffed variety decreases mucus flow in the trachea more so than the uncuffed variety.[14] The low-volume, small resting diameter cuff is causing deeper damage to the tracheal mucosa; hence, healing takes longer and a defective mucociliary transport may exist for weeks. This decrease in mucociliary clearance is enhanced by the administration of anesthetic agents[14] and inadequate humidification.[15] Nosocomial infection of the tracheobronchial tree often implicates gram-negative bacilli and is somewhat lessened by adequate humidification.[16] Culture identification of *Klebsiella pneumoniae, Staphylococcus aureus, Escherichia coli, Proteus mirabilis,* and *Pseudomonas aeruginosa* helps to differentiate infection from colonization.[4] The incidence of pneumonia, mostly resulting from aspiration, is highest after emergency intubation (45%), often occurring within 72 hours but without changing outcome.[17]

PATHOPHYSIOLOGY

Aphonia is the result of decreased vocal cord adduction ability often caused by subluxation of the arytenoid cartilages (see Fig. 79–1*C*), ulceration of this area (see Fig. 79–1*D*), and cricoarytenoid ankylosis after long-term endotracheal intubation.[18] Vocal cord immobility may be caused by interarytenoid fibrosis specifically involving the vocal processes and posterior commissure[17,19] (Fig. 79–1*E*). Vocal cord paralysis suggests dysfunction of the recurrent laryngeal nerve, which may be injured if the cuff is inflated in the

TABLE 79–1. Complications Associated with Prolonged Intubation*

Common	*Rare*
Infection	Cricoarytenoid subluxation
Hemorrhage	Cricoarytenoid fixation and scarring
Aspiration	Vocal cord paralysis
Air leak	Tracheal necrosis
Subcutaneous emphysema	Tracheal rupture
Pneumomediastinum	Paratracheal abscess
Pneumothorax	Tracheoinnominate artery fistula
Atelectasis	Tracheoesophageal fistula
Laryngeal edema	
Laryngeal ulceration	
Laryngeal granuloma	
Laryngotracheostenosis	
Tracheomalacia	

*For details, see Hsu et al,[12] Chilla and Gabriel,[18] Cavo,[20] Gibbin and Eggington,[21] Levin,[22] Bein et al,[23] Heath and Peirce,[24] Abbey et al,[25] Rinecker and Schvet,[26] Payne et al,[27] Bishop et al.[28]

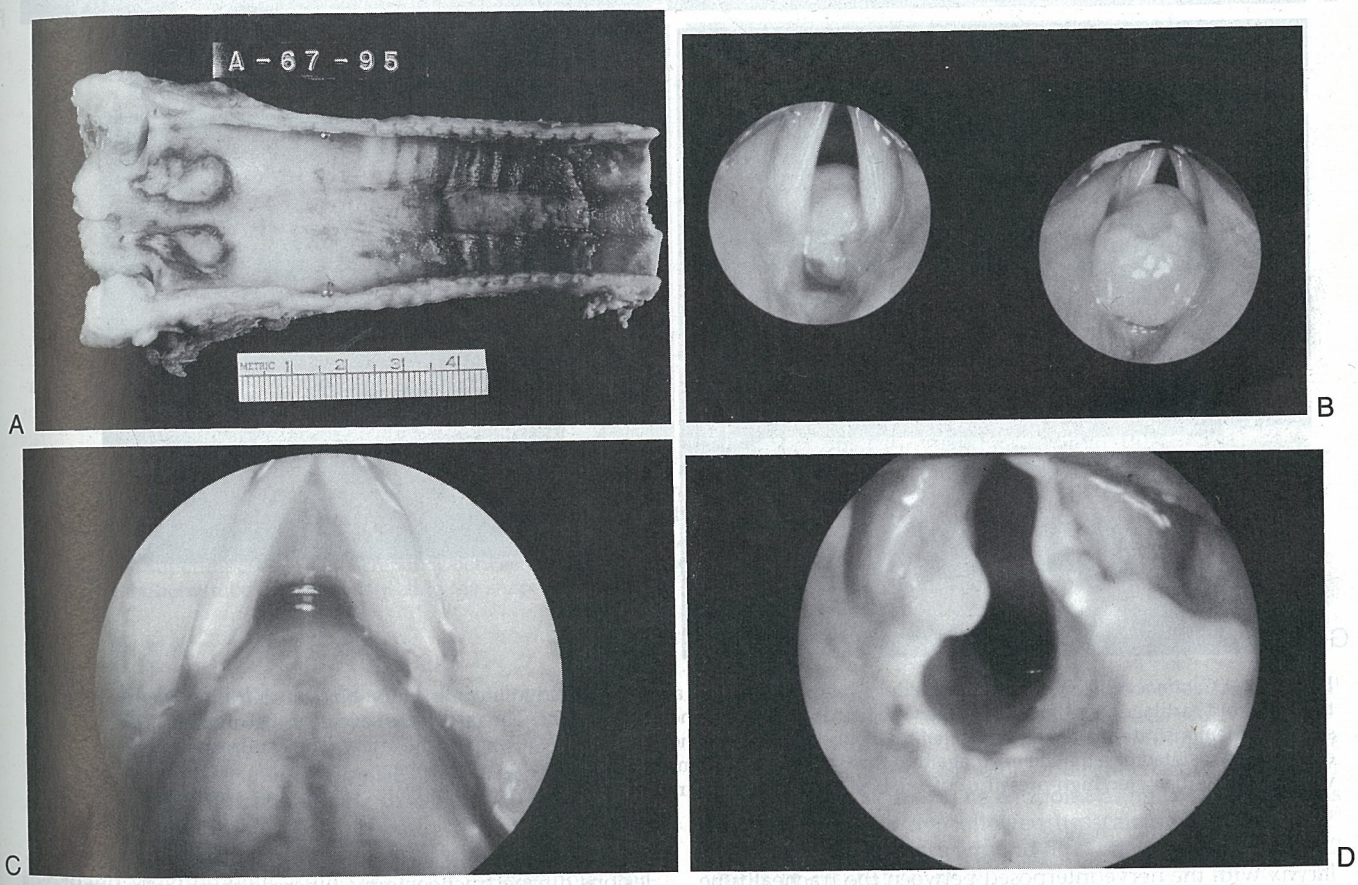

Figure 79–1. *A*, Laryngotracheal specimen, split along the anterior midline, showing necrotic lesions at the medial sides of the arytenoid cartilages and the cricoid plate. The lesions were caused by pressure from the tracheal tube and, further down the trachea, by the cuff. *B*, Endoscopic views of a pendulating postintubation granuloma at the right medial region of the arytenoid cartilage. *C*, Endoscopic view of a tracheal tube that is too wide in relation to the size of the interarytenoid space. *D*, Close-up view of the larynx immediately after extubation. Pressure necrosis and inflammation posteriorly are present where the tracheal tube was located. (See Color Plate Section of this textbook.)

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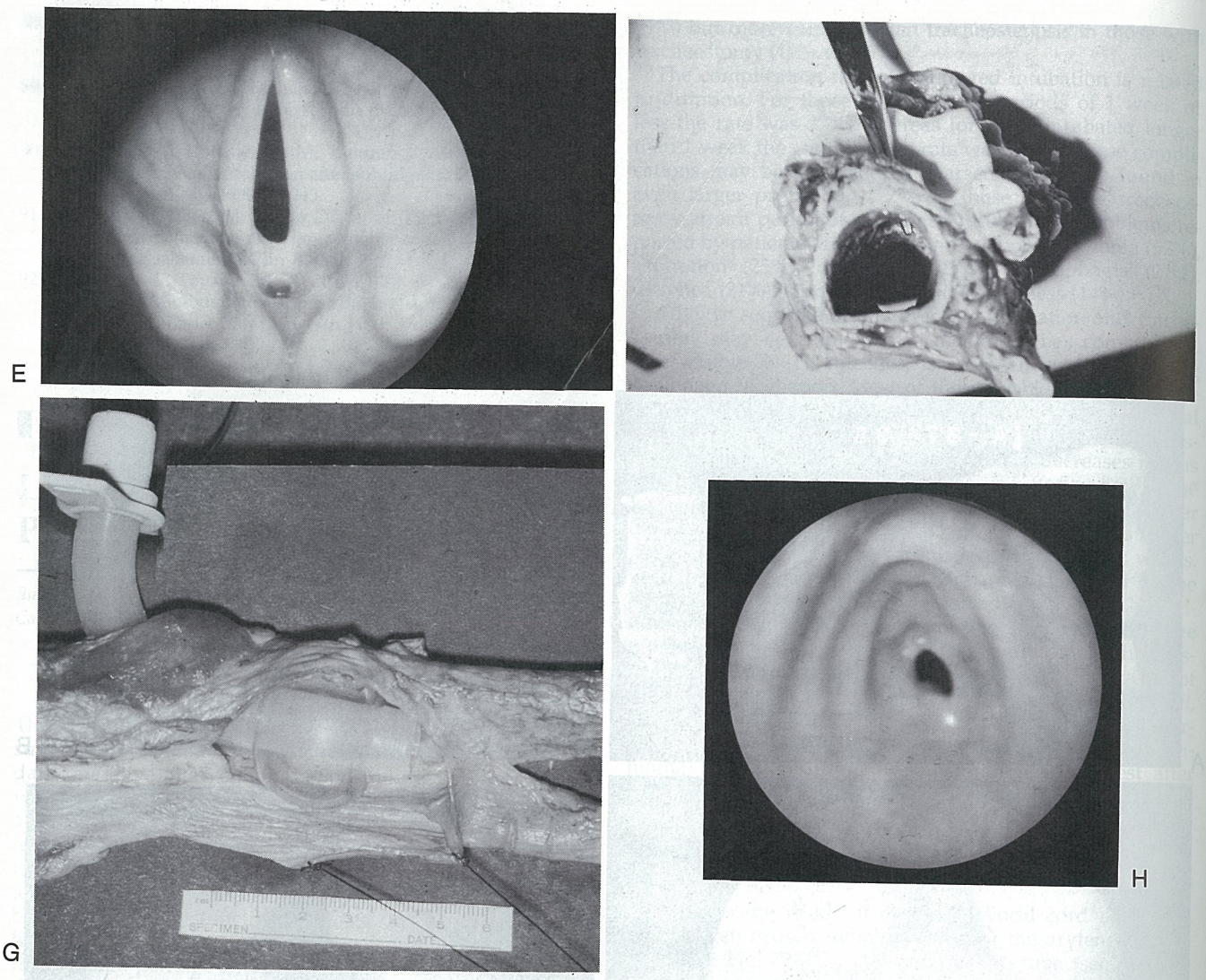

Figure 79–1 *Continued E,* Follow-up laryngeal view showing healing after prolonged intubation injury. Fibrous scarring is present between the arytenoid cartilages, and fixation of the vocal cords can be seen near the midline. The result was severe laryngeal stenosis. *F,* Autopsy specimen demonstrating a fistula (indicated by hemostat) between the trachea and the innominate artery. The fistula was caused by cuff erosion and resulted in lethal hemorrhage into the trachea. *G,* Specimen with a large tracheoesophageal fistula due to erosion by a low-volume, small-diameter, high-pressure cuff. *H,* Severe cuff-induced tracheal stenosis. (See Color Plate Section of this textbook.)

larynx with the nerve interposed between the tracheal tube cuff and thyroid cartilage below the vocal cords.[20] This vocal cord paralysis may be unilateral or bilateral.[21] Function may return because this neuropraxia is sometimes only temporary.

Catastrophic complications include perforations in the hypopharynx, posterior to the cricopharyngeal muscle, or in the piriform sinus, which require early surgical repair.[22] Tracheal rupture may be found in those predisposed by musculoskeletal conditions such as the rigid spine syndrome.[23] Retropharyngeal abscesses are found after lacerations caused by emergency nasotracheal intubation and are indicated by rapid onset of fever and odynophagia.[24] Massive tracheal necrosis has been found in patients with hypoperfusion, infection, and excessive cuff pressure.[25]

The tracheoarterial erosion syndrome involving the brachiocephalic trunk often results in rapid patient demise as a result of massive bleeding into the trachea (see Fig. 79–1*F*).[26] Tracheoesophageal fistulas (see Fig. 79–1*G*) can be found acutely in those with traumatic injury, including iatrogenic lesions during tracheostomy, whereas chronically ventilated patients may demonstrate a more indolent course.[27] These conditions require a high index of suspicion to avoid further patient compromise. However, the most germane issue for routine intensive care practice is an immediate diagnosis of tracheostomy-induced pneumothorax, either unilateral or bilateral. Complications of prolonged airway support are better understood by analysis of pathologic findings. Most long-term complications are attributed to the tube or cuff components (see Fig. 79–1*A*). The presence of an endotracheal tube segment affixed in a canine larynx caused erythema at 24 hours, progressing to severe mucosal ulceration and loss of normal architecture by 1 week.[28] Cuff-induced complications, usually stenosis (see Fig. 79–1*H*) or tracheomalacia (Fig. 79–1*I*), occur most commonly below the first tracheal cartilage.[29] Initial changes are functional, with slowing or complete interruption of tracheal mucus transportation. The superficial respiratory epithelium may undergo squamous metaplasia.[30] The epithelium may begin this alteration after as few as 4 hours of intubation. These early

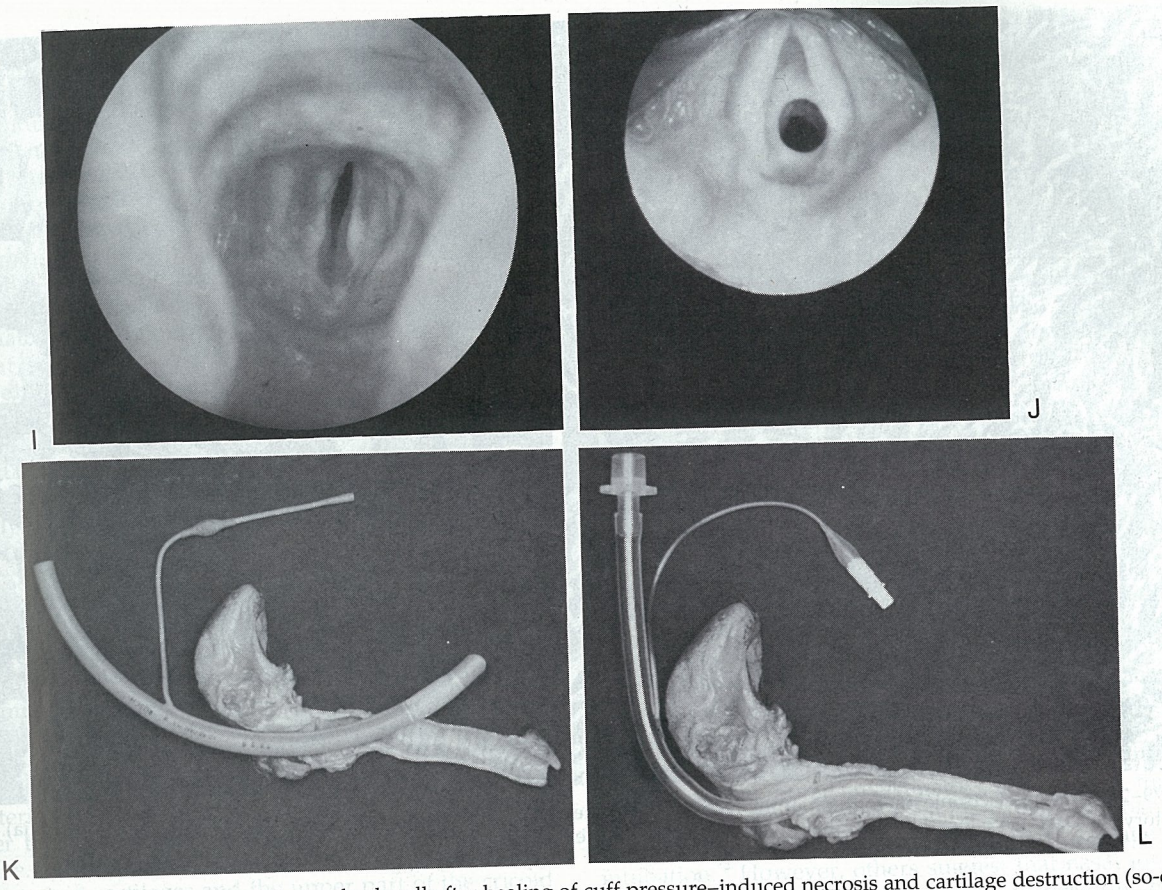

Figure 79–1 *Continued I,* Softening of the tracheal wall after healing of cuff pressure–induced necrosis and cartilage destruction (so-called "tracheomalacia") results in tracheal collapse during exhalation, air trapping in the lungs, and life-threatening respiratory failure. *J,* Endoscopic view of fibrous subglottic stenosis caused by circumferential pressure from a tracheal tube that was too large in relation to the size of the cricoid ring. *K,* An autopsy specimen of the tongue, larynx, and trachea, mainly in their in vivo positions, with a standard tracheal tube superimposed. The discrepancy between the shape of the tube and that of the airway can be seen. *L,* The configuration of the anatomic Lindholm tube corresponds well to the shape of the natural human airway. (See Color Plate Section of this textbook.)

changes consist of flattening, fusion, and erosion of respiratory epithelial cells with ciliary disappearance or compromised function resulting from mechanical abrasion and ischemia (Fig. 79–2).[31] The presence of cuff irritation also results in constriction of smooth muscle of the trachea.[32]

The tracheal arterioles are located in the submucosa and are circumferentially oriented anteriorly between the cartilages and longitudinally in the posterior membranous portion.[33] Capillary perfusion pressure, estimated at 22 mm Hg (30 cm H_2O), is inversely proportional to cuff tracheal wall pressure. Studies using an endoscopic photographic technique suggest continued tracheal blood flow at a cuff pressure of 25 cm H_2O, but the mucosa becomes pale at 40 cm H_2O and blanched at 50 cm H_2O, and flow is absent at 60 cm H_2O.[33] Thus, consideration of cuff to tracheal wall pressure suggests that flow is initially affected at cuff pressure levels of 30 cm H_2O, with complete occlusion at 50 cm H_2O of intracuff pressure.[33] These results have been reproduced by radioactive hydrogen clearance testing, suggesting decreased flow at cuff to tracheal wall pressure of 30 mm Hg (41 cm H_2O) with a practical limit for intracuff pressure of 20 mm Hg (27 cm H_2O).[34] However, at least in rabbit experiments, there is a biphasic response in which normal tracheal blood flow (0.3 mL/min per gram of tissue) is increased 10-fold by the tube irritation because of histamine relaxation of arterioles.[35] Nonetheless, cuff pressure of 30 mm Hg or even levels between 20 and 30 mm Hg may cause significant ischemia of mucosa localized over the tracheal cartilages if a low-volume, small-diameter cuff is used.[35] With a high-volume, large-diameter, thin-walled cuff, the safety margin is greater before total mucosal ischemia over the cartilages occurs.[35] The endpoint of mucosal ischemia is necrosis and infection followed by scarring and stenosis.[19] Subglottic stenosis was found in a canine model after 2 weeks of intubation in which ulceration was followed by granulomatous tissue and scar formation.[36]

In one study, 33% of patients intubated more than 24 hours healed with granuloma formation and concomitant hoarseness, but at 3 months' follow-up only 10% of these granulomas persisted and necessitated removal.[37] Human studies in neonates reveal a 1% to 8% incidence of such lesions in the subglottic region at the cricoid cartilage ring beginning with mucosal necrosis and progressing to full-thickness erosion, perichondritis, and scar formation proportional to the duration of intubation.[19]

Another outcome study demonstrated that 78% of laryngeal intubation lesions re-epithelialized and healed by 8 weeks. Only 7% were left with residual granuloma.[38] Retrospective autopsy observations include inflammation of the perichondrium of the vocal cord processes and arytenoid and cricoid cartilages as well as bacterial invasion beginning at 48 hours and progressing to ulceration by 96 hours.[39] In a prospective study of patients intubated for a mean of 48 hours, most patients had mucosal ulcerations over the ary-

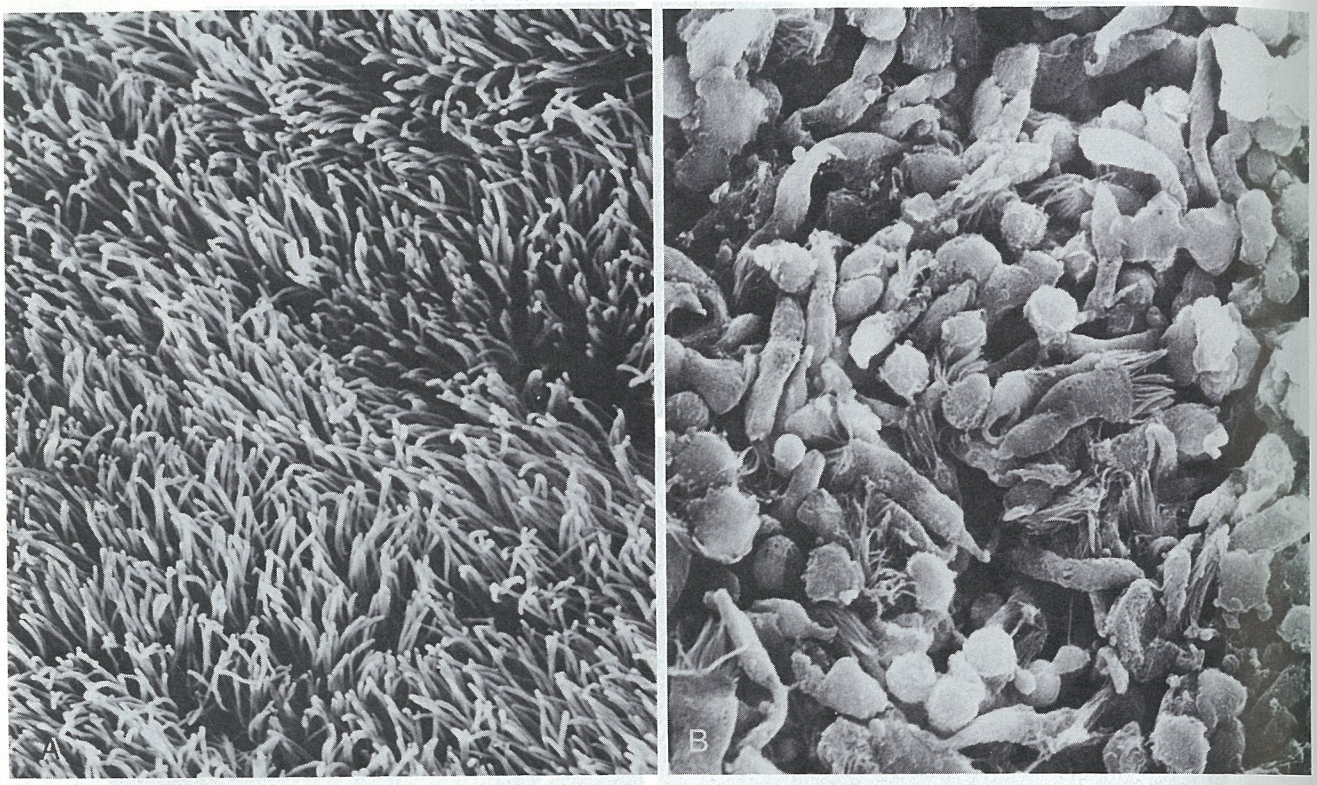

Figure 79–2. Scanning electronic micrograph of the respiratory epithelium. *A,* Normal endothelium (the cells are covered by cilia). *B,* Respiratory cells that have lost their cilia due to erosion by the tracheal tube cuff.

tenoid and cricoid cartilages.[37] Comprehensive endoscopic evaluation suggests inflammation of the true vocal cords in 68%, posterior commissure in 29%, false vocal cords in 29%, glottis in 21%, closed folds in 18%, and arytenoid cartilage area in 11% of the patients examined.[20] Using a grading scale, such lesions have been described as mild in 36%, moderate in 24%, severe in 22%, and complete subglottic obstruction in 15%.[11]

Tracheal Tube Problems

Complications of tracheal intubation can be attributed to the separate tube and cuff components. The presence even of a cuffless tube has resulted in severe laryngeal injury, discovered at autopsy.[40] These effects may be due to tracheal tube characteristics such as size, tip design, rigidity, position, shape, or other distinguishing qualities.[19] There is a clear association between endotracheal tube diameter and complications (Table 79–2).[41] The tracheal tube injury is primarily localized to the posterior glottis, where pressures of 200 to 400 mm Hg are exerted by the elastic recoil force of regular polyvinylchloride (PVC) tubes, somewhat lessened by a narrower tube. The nondeformable round tube exerts lateral pressure on the arytenoid cartilages, where it is wedged between the vocal cords (see Fig. 79–1*C* and *D*).[42] This relationship depends on the external diameter of the tube compared with the available distance between the arytenoid cartilages. The narrowest rigid portion of the larynx is found at the cricoid cartilage, the only solid complete ring of the airway. Particularly in children, a tracheal tube that is too wide will result in circumferential necrosis, healing with serious stenosis, which is difficult to treat surgically (see Fig. 79–1*J*). Thus, even a 7-mm internal diameter (ID) tube is sometimes too large for women, whereas an 8-mm ID tube is often acceptable for the larger interarytenoid space in men. Clinical examination of tubes of various sizes suggests that, although laryngeal wall pressure is decreased by smaller tubes, the sealing cuff pressure required for effective ventilation may increase as tube diameter decreases. Because the smaller tubes have a smaller cuff resting diameter in relation to the trachea, they fail to act as large-diameter, high-volume cuffs.[43] Thus, the most effective regimen

TABLE 79–2. Tracheal Tube Sizes

Inner Diameter (mm)	*Approximate Outer Diameter (mm)*	*Length (mm)*
2.0	3.0	140
2.5	3.6	140
3.0	4.3	160
3.5	4.9	180
4.0	5.6	200
4.5	6.2	220
5.0	6.9	240
5.5	7.5	270
6.0	8.2	280
6.5	8.9	290
7.0	9.5	300
7.5	10.2	310
8.0	10.8	320
8.5	11.4	320
9.0	12.1	320
9.5	12.8	320
10.0	13.5	320

(Modified with permission from International Standards Organization: International standard 5361-2: Tracheal tubes: Part 2. Orotracheal tubes of Magill type [plain and cuffed]. Geneva, Switzerland, International Standards Organization, 1993.)

is to use an appropriately sized tube based on laryngeal diameter with a cuff diameter of more than 30 mm in adults, which is required for an effective seal at approximately 25 cm H_2O of intracuff pressure.

Conventionally shaped, semicircular tubes do not conform with the patient's airway anatomy and exert undue high pressure posteriorly in the larynx (see Fig. 79–1*K*). A most promising new development in minimizing the complication rate inherent to the tube itself is the anatomically shaped tube (see Fig. 79–1*L*). This tube design uses an anteroposterior configuration that conforms with the human airway anatomy and minimizes those posterior deformation forces that result in laryngeal damage.[44] This relationship was first explored by Lindholm, who suggested that deformation forces were minimal with an anatomically shaped tube (30–41 g) compared with PVC (230–296 g) and red rubber (1000 g) tubes. These differences were accentuated as tracheal tube size increased.[45, 46]

Recently, laboratory viscoelastic tests to predict the mechanical consequences of intubation were described by a French group that mainly confirmed previous findings (see Table 79–2).[47] Furthermore, when used clinically, the anatomic tube resulted in a decreased incidence of moderate and severe laryngeal injury and less postintubation complaints of hoarseness and sore throat.[48, 49] Use of soft tracheal tubes composed of silicone rubber also results in decreased posterior laryngeal pressure.[46] However, one clinical trial suggested equivalent isolated arytenoid and tracheal damage (mainly because of tube diameter and cuff pressure), but posterior laryngeal damage to the interarytenoid area and over the cricoid plate is still minimized by the Lindholm tube.[15] This is crucial because these lesions posteriorly at the arytenoid cartilages and the upper part of the cricoid cartilage are the cause of laryngeal scarring, fixation, and stenosis.[19] Such sequelae are almost impossible to correct and lead to lifelong impairment of breathing and phonation. It is the authors' opinion that the result of a peg board test giving the approximate value of the recoil pressure at the cricoid cartilage should be provided by tracheal tube manufacturers so that the clinicians may choose the least traumatizing tube for each situation.[46]

The limiting factor in minimizing tube size is the increasing respiratory gas flow resistance, imposing increased work for the patient with each spontaneous breath. There is a linear relationship between the pressure gradient and flow. The pressure gradient required is decreased with a larger diameter, shorter, and straighter tube. The position of the tube in the airway is also significant; a concentric intratracheal location sufficiently above the carina to prevent obstruction is clearly advantageous.[50] The resistance of gas flow may be quantified as work of breathing, which is inversely proportional to the tube diameter. Thus, the work of breathing is greater with tubes of 6- to 7-mm ID, sometimes used for nasal or emergency intubation, compared with tubes of 8- to 9-mm ID.[51] In other words, one must balance the more traumatizing effect of larger tubes against increased work of breathing through narrower tubes.

The next significant correlate is the tube composition. These intubation devices are tested for safety and designated IT (implantation tested) according to the U.S. Pharmacopeia XVII or Z79-certified by the American National Standards Institute.[52]

The issues regarding the nasotracheal intubation route are complex, with a clear dichotomy between those that support or reject its use. The most common complication is bleeding, occurring in roughly 45% of nasotracheally intubated patients, usually without adequate nasal mucosa preparation.[53] However, in the intensive care unit (ICU), the most important issue is iatrogenic sinusitis. Among the causes is decreased nasal mucociliary clearance, which can be detected after extubation by a radiopaque disk method with correlation to difficult or prolonged intubation, resulting in mechanical trauma to the surface of the epithelium.[54] The incidence of sinusitis is considerably higher with nasal (43%) than with oral (2%) tubes.[55] Infection occurs on the ipsilateral side in 42% and on the contralateral side in 27% of patients. Sinusitis may require drainage but does not seem to affect mortality.[55] Nasotracheal intubation is most often associated with maxillary or sphenoid sinusitis, followed by ethmoid and frontal sinus problems.[56] The incidence is related to the duration of intubation, with about 33% affected after 3 days and 100% of patients involved after 1 week. However, the majority of cases (88%) resolve within 1 week of extubation.[56]

The clinical significance of sinusitis needs to be examined in the context of bacteremia and sepsis. The incidence of bacteremia is common with nasotracheal intubation but rarely occurs with orotracheal intubation.[57] True sepsis occurs in 7% of those with paranasal sinusitis and more commonly after emergency intubation than elective cases.[58] Patients are predisposed by a history of diabetes mellitus or steroid use. Diagnosis is achieved by computed tomography or plain radiography. A decision must be made whether to extubate, change to orotracheal intubation, or tracheotomize. Topical decongestants, antihistamines, and antibiotics are indicated in these cases.

The debate concerning the advisability of nasal intubation remains active. Some investigators believe that the nasal route may be safe for long-term (3–63 days) intubation and may be associated with half the laryngeal damage of oral intubation.[59] However, others suggest that nasal intubation takes two and one-half times longer to perform than oral intubation and has a significant rate of bleeding (45%) and septicemia (9%). Were it not for greater comfort and less laryngeal injury, it would offer no significant advantage.[53] The development of laryngeal injuries caused by tracheal tubes has been summarized by Benjamin (Fig. 79–3).[19]

Cuff Problems

Traditionally, the cuff of the tracheal tube has caused more severe complications than the tube itself. However, these cuff problems have decreased, both in frequency and severity, with the common, almost exclusive use of large-diameter, large-volume cuffs with soft, moderately compliant, thin walls. These are inflated to 25 cm H_2O at the end of exhalation. The intracuff pressure should be monitored frequently or continuously to avoid tracheal mucosal injury and, as mentioned previously, should not be allowed to exceed 30 cm H_2O. During the inspiratory phase of positive-pressure ventilation, the cuff pressure will passively increase to or just above peak airway pressure. The cuff volume simply decreases with a concomitant elevation of pressure, but the cuff still expands its diameter to seal the trachea because of excess cuff resting diameter.

Standard monitoring uses a three-way stopcock and a mercury sphygmomanometer to sense cuff pressure (Portex, Keene, NH), a simple system that is 99% accurate to within ± 2 mm Hg.[60] Direct cuff to tracheal wall pressure sensing has been described in research protocols using a Teflon envelop positioned between the cuff and tracheal wall, which is able to document pressures as low as 1 to 2 mm Hg.[61]

The effect of peak airway pressure is usually not appreciated as an important factor in cuff-induced mucosal injury if only exhalation cuff pressure is considered.[50] In addition, the monitored proximal airway pressure does not reflect

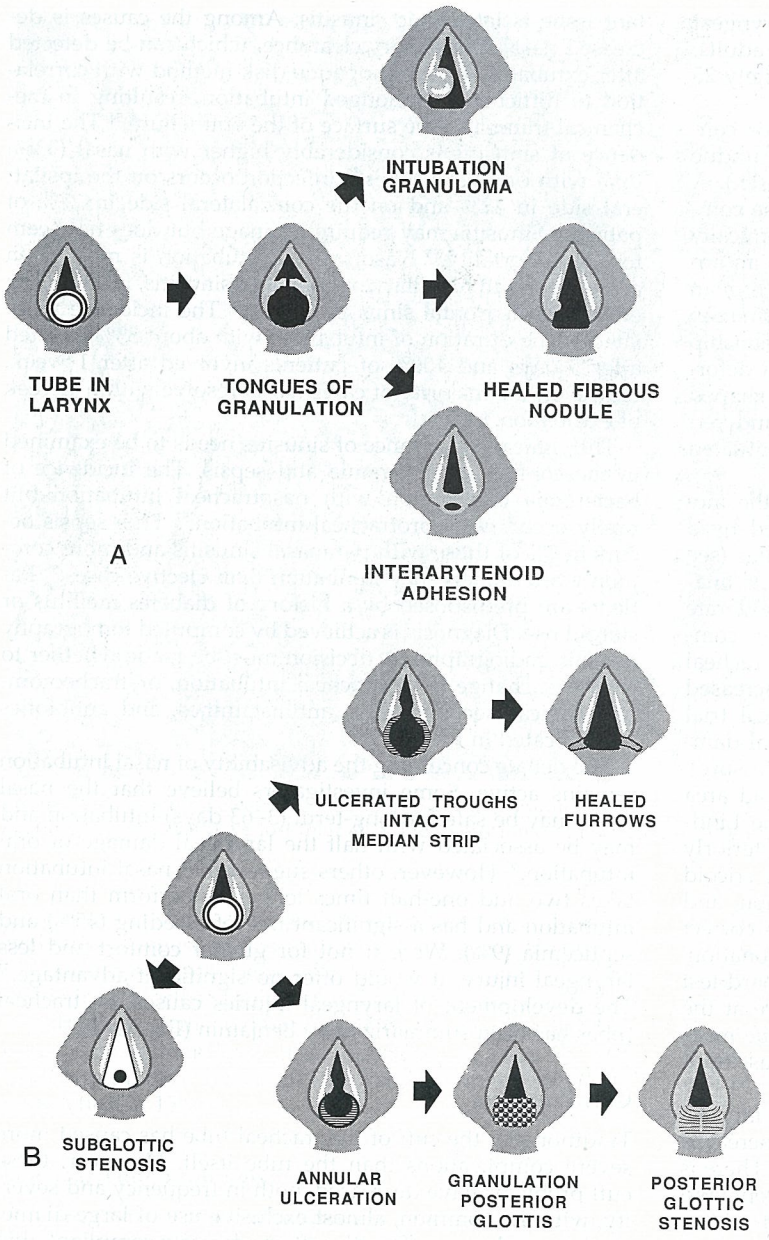

Figure 79–3. *A,* Development of three types of sequelae following endotracheal tube–induced laryngeal injury. Tongues of granulation may cause vocal cord granulomas or fibrous nodules, which are easy to remove without causing functional impairment. As long as interarytenoid adhesions leave the posterior commissure intact, it is usually possible to resect the fibrous band and restore adequate laryngeal function. *B,* Prolonged tracheal intubation may also lead to the formation of ulcerated troughs. Trough healing results in the creation of "furrows," which may cause a problem referred to as "leaking voice," which occurs when air loss causes phonation difficulties. A circumferential subglottic stenosis as well as posterior glottic stenosis are conditions that are extremely difficult to correct surgically. (From Benjamin B: Prolonged intubation injuries of the larynx: Endoscopic diagnosis, classification, and treatment. Ann Otol Rhinol Laryngol Suppl 1993; 160:1.)

actual distal airway pressure (below the tube), which is different (i.e., lower during inspiration and higher during exhalation).[62] Furthermore, an awake patient often fights the ventilator with a significant increase in airway pressure at oral suctioning (twofold), endotracheal suctioning (threefold), coughing, and movement.[63]

Cuff damage initially manifests itself clinically as tracheal dilatation with onset after 4 to 8 days, especially with small-diameter, high-pressure, low-compliance cuffs.[64] Scanning electron microscopy of canine trachea from autopsy sections documents complete absence of cilia after short use of an inflated small-diameter cuff. Mucous membrane damage occurs, especially over the tracheal cartilages with minimal cuff inflation already after 2 hours.[65] In dogs, these changes are partially reversed after 2 days and completely resolved by 1 week. Similar observations have been made in human anatomic specimens using standard tracheal tubes and cuffs.[65]

The first tracheal tube cuffs used were of the low-volume, high-pressure variety. Currently, the standard is a large-diameter, high-volume, low-pressure cuff, which causes a lower and more evenly distributed cuff to tracheal wall pressure with less mucosal damage (Fig. 79–4). A canine model suggests that a low-pressure cuff demonstrates minimal gross or microscopic lesions compared with the high-pressure variety, which, with prolonged use, causes circumferential erosion, mucosal ulceration, and cartilage destruction.[66] Tracheal wall pressure is due to a complex interaction of tracheal diameter and compliance versus cuff diameter, compliance, inflation pressure, and symmetry.

Intubated ICU patients are at risk for further complications after general anesthesia, including use of nitrous oxide (N_2O), which results in spontaneous elevation of cuff pressure. This effect occurs when oxygen and N_2O diffuse into the air-filled cuff. Partial gas pressures inside the cuff will equilibrate with the outside, causing a progressive increase in cuff volume and pressure. Clinical trials have suggested that this effect is minimized in low-pressure, high-volume

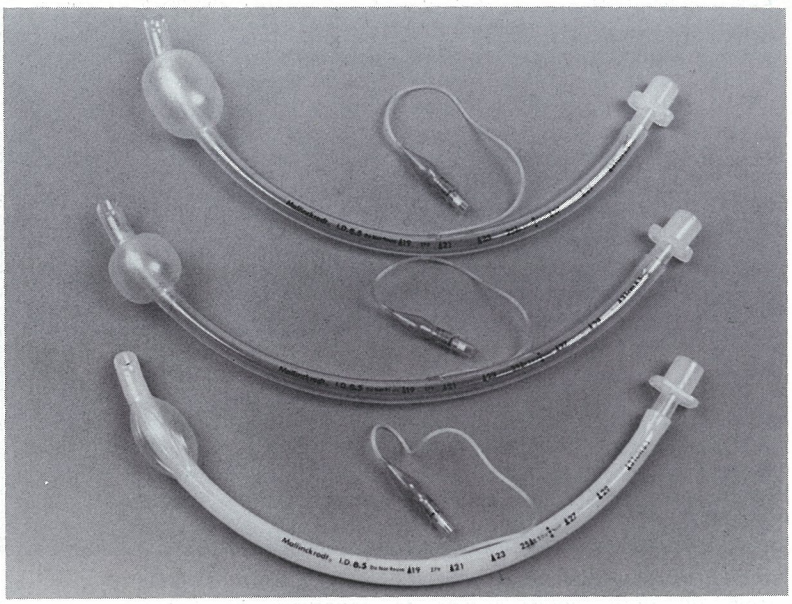

Figure 79–4. Different cuff designs. *Top,* A large-diameter, high-volume, low-pressure cuff. *Middle,* An intermediate-diameter, -volume, and -pressure cuff. *Bottom,* A small-diameter, low-volume, high-pressure cuff.

cuffs and may be prevented by filling the cuff with the used anesthetic gas mixture. Cuff pressure has been monitored and has demonstrated a fivefold increase during general anesthesia with N_2O inhalation. Modern tracheal tubes may reach cuff pressures of 30 to 100 mm Hg within 2 to 3 hours.[67] Gas diffusion is governed by N_2O concentration, wall thickness, and compliance of the cuff. The "rediffusion system" allows cuff-resorbed N_2O to diffuse into a high-compliance pilot balloon in which reversed diffusion takes place, resulting in reduction of cuff pressure toward the original setting.[68]

Lomholt tested different resting-diameter cuffs in a tracheal model with a compliance similar to that of the human trachea. The lung model was ventilated with different peak airway pressures up to 6 kPa (60 cm H_2O).[69] The lateral wall pressure (LWP) at end-expiration was registered when the cuff achieved a leak of 10 mL ± 1 mL per inspiration. For cuffs with a resting diameter of 31 mm or greater, a seal was present below 3 kPa LWP at end-expiration even when peak airway pressures reached 6 kPa. These cuffs could be regarded as true low-pressure, high-volume, large-diameter cuffs. Cuffs with diameters of 25 to 29 mm had to be inflated to an LWP of more than 3 kPa to provide a seal at peak airway pressures exceeding 3 kPa. These cuffs acted similarly to high-pressure, low-volume, small-diameter cuffs. Lomholt concluded that the minimum diameter for a low-pressure, large-volume cuff should be approximately one and one-half times the tracheal diameter when high inflation pressures are used.[69]

More moderate approaches suggest that the best cuff may be the intermediate-volume variety. However, it is not possible to rely on monitored intracuff pressure as an indication of cuff to tracheal wall pressure if the cuff diameter is too small for such pressure equilibrium in patients with a wide trachea. Both large- and small-volume cuffs may cause experimental damage.[50, 70] The risk of cuff herniation between the vocal cords with a malpositioned large-volume cuffed tube has also been described.[71] Although the enlarged surface area of a large-volume, thin-walled cuff results in less severe damage than the low-volume, high-pressure cuff, large-volume, thick-walled cuffs may still cause specific lesions as a result of redundant excessive cuff folds.[72] The current consensus is that large-diameter, large-volume cuffs should be used in most patients, although occasional individuals may benefit from use of an intermediate-volume cuff or, in rare circumstances, even a low-volume cuff.

In canine trials, the foam cuff has demonstrated a decreased incidence of mucosal ischemia, ulceration, and cartilage damage.[73] This foam cuff (Bivona, Gary, IN) establishes a tracheal seal from recoil pressure of the foamy material, generating a tracheal wall pressure of approximately 15 cm H_2O, but only if a cuff diameter is chosen that is optimal in relation to the size of the trachea (Fig. 79–5). The seal persists at atmospheric airway pressure, thus preventing aspiration without tracheal damage.[72] Trials of foam cuffs compared with high-volume, low-pressure cuffs suggest less mucosal damage with the former.[74]

Mucosal damage caused by the cuff is also minimized by regulation of pressure compared with monitoring alone. The Lanz pressure-limiting device, for instance, allows maximal cuff pressures of 25 cm H_2O (see Fig. 79–5). If overinflated, cuff volume decreases as excess air moves into the more compliant pilot balloon.[33, 75]

Another significant cuff-related complication is aspiration. Secretions pooled above the cuff may slowly and gradually leak into the trachea, causing subsequent infection.[76] Interestingly, mechanical ventilation compared with spontaneous breathing is protective, with aspiration decreasing from 100% to 55%.[77] The addition of positive end-expiratory pressure further decreases aspiration.[77] Similarly, a cuff system that permits removal of subglottic secretions pooling above the cuff (HiLo evacuation tube, Mallinkrodt) decreases tracheal colonization and the incidence of pneumonia.[78]

Clinical evaluation of cuff design demonstrates that the highest rate of aspiration occurs with the high-volume, low-pressure cuff (56%) followed by the intermediate cuff (39%) and the low-volume, high-pressure (20%) design.[79] The most likely explanation for these results is incorrect intermittent monitoring of intracuff pressure. If the manometer is attached to the cuff inflation tube while reading zero, the cuff pressure will drop momentarily because of the dead space in the measuring device, and aspiration may occur. Although the high-volume, low-pressure cuff minimizes mucosal damage, the aspiration rate may be substantial if the cuff pressure is not maintained at all times.

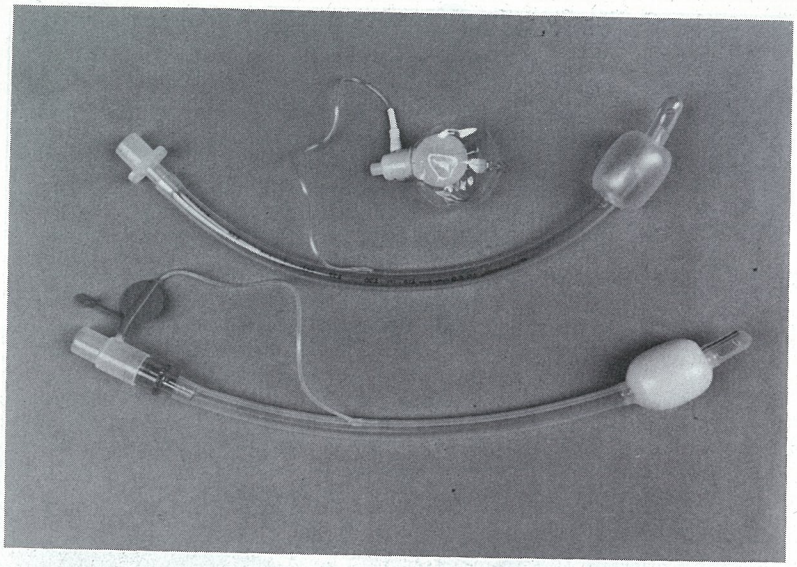

Figure 79–5. Cuff pressure regulating system. *Top,* A Lanz device. *Bottom,* Foam cuff connected to main airway.

SPECIAL TUBES

The double-lumen tube was first described by Carlens.[80] Robertshaw modified the original design because of difficulty with placement and increased resistance with Carlens' tube.[81] Advantages of the double-lumen tube include avoidance of contamination of the unaffected lung from contralateral abscess or hemorrhage during surgery.[82] Double-lumen tubes are increasingly used in patients who undergo single (or bilateral sequential) lung transplantation. Gross difference in compliance between the remaining native and transplanted lung is an indication for continued use of the double-lumen tube postoperatively in the ICU. Disadvantages are due to tube rigidity and include the presence of two cuffs with increased risk of laryngeal and tracheobronchial injury. Air leak and even mainstem bronchial rupture have been reported.[82]

Correct positioning of the double-lumen tube is crucial to its intended function. There are two types of tubes—left sided and right sided—depending on into which mainstem bronchus the distal of the two lumina will enter. With the left-sided tube, positioning is easier because of the longer mainstem bronchus on that side. The distance between the distal opening of the tube and lower cuff margin is 30 mm.[83] The right-sided tube is more complex in its design because the distance between the carina and the right upper lobe bronchus is only about 15 mm. Therefore, this cuff configuration is not symmetric because of the necessary side opening in the distal tube component to permit ventilation of the right upper lobe. This intermediate opening is located 10 to 20 mm from the tube tip. Thus, the right-sided tube has a total of three distal openings, whereas the left-sided tube has only two.

Cadaver studies have revealed an estimated length of the right mainstem bronchus of only 15 versus 19 mm and of the left mainstem bronchus of 44 versus 49 mm in females and males, respectively.[83] Consequently, fiberoptic bronchoscopy confirmation of tube position is essential to minimize complications. It has been recommended to place the left-sided tube with its distal tip 29 cm ± 1 cm from the teeth with 1 cm added or subtracted for each 10-cm height difference from a 170-cm normal height.[84] The right-sided tube has a decreased margin of safety for anatomic reasons discussed previously. Therefore, the left-sided double-lumen tube is favored in most situations. The double-lumen tube has been used in 48,000 anesthetic procedures with a 0.4% rate of tracheal rupture in patients predisposed for this complication as a result of tuberculosis or bronchitis.[85] The left-sided tube has been associated with acute right mainstem bronchus obstruction by the distal cuff if the tube tip is too close to the carina.[86] Tracheal rupture has also been reported with PVC tubes.[82] Clinical evaluation further reveals that other complications such as unsuccessful intubation, dislodgement, and malposition manifested as hypoxemia are more common with the Carlens (50%) and Robertshaw (23%) tubes than the newer PVC tubes (4%).[87]

The right-sided endobronchial blocker tube (Portex, Ltd., Hythe Kent, UK) has been found to have an efficacy equivalent to that of the standard double-lumen tube.[88] This tube is also used postoperatively for independent lung ventilation with separate regulation of ventilatory parameters in cases of different lung compliance.[89]

TUBE POSITION AND FIXATION

Complications may be minimized by attention to detail concerning tracheal tube position and fixation. Laryngeal damage is worsened in those who undergo head extension positioning, excessive suctioning, and states of agitation.[90] The extent of damage is partly due to the amount of movement of the tube shaft relative to the arytenoid cartilages and posterior cricoid plate. Furthermore, the tube tip may be directed into the anterior tracheal wall.[91]

Initial tube position may be estimated from various anatomic landmarks. The following distances are of importance: from teeth to vocal cords (12–15 cm), from vocal cords to carina (10–15 cm), and from tube tip to carina (2–6 cm).[49, 92] Clinically, Goodman's criteria suggest a 5 cm ± 2 cm tracheal tube tip distance to carina, which has demonstrated no accidental extubation and only 1% endobronchial intubation.[91] Essential to intensive care is the dynamic description of tube variability in which head flexion moves the tube tip approximately 2 cm closer to the carina, extension moves it approximately 2 cm away from the carina, and lateral rotation moves it 1 cm away from carina, suggesting that the appropriate tube tip position is in the middle third of the trachea.[93] However, this means that part of the cuff sometimes is located in the lower half of the larynx because the distance from the tube tip to the upper end of the cuff

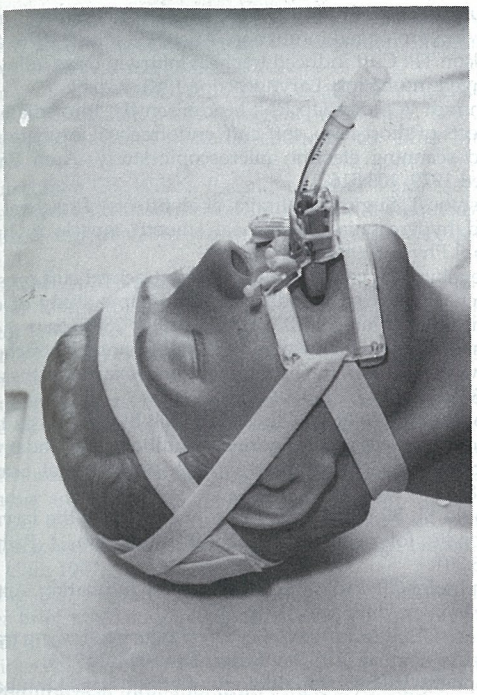

Figure 79–6. The ReviveEasy PtL Airway used with the Secure Easy endotracheal tube holder. (Courtesy of Respironics, Murrysville, PA, and of IPI Medical Products, Chicago, IL, respectively.)

in most tubes is 5 to 6 cm. Therefore, it might be safer to place the tube tip at the border between the lower and middle thirds of the trachea (i.e., 3–4 cm above the carina).

Tracheal tube fixation should be stable and comfortable and allow emergency access to the patient. The most stable fixation is provided by buccal or oral devices used in oral and maxillofacial surgery, but these systems are uncomfortable to the patient.[94] The emergency situation is addressed by tube fixation using various adhesive tape strategies. The Secure Easy fixation device (IPI Medical Products, Chicago, IL) is composed of a Velcro wrap and plastic tube holder (Fig. 79–6). This device has proven superior to adhesive tape in preventing tube displacement, as measured clinically and radiographically.[95] However, cutaneous breakdown may still occur as with adhesive tape.

ASSESSMENT OF INJURIES AND THERAPEUTIC INTERVENTION

The most simplistic analysis of complications may be addressed by monitoring of cuff size on routine chest radiography.[64] The appearance of an overdistended cuff often precedes tracheal dilatation, subcutaneous emphysema, or pneumomediastinum.[96] The most sensitive diagnostic tool is bedside video laryngoscopic assessment, which has been used in the ICU setting, demonstrating vocal cord ulceration or subtle motion abnormalities or paresis.[97] Historically, xerotomography and tracheography have been supplanted by computed tomography, with the capability of diagnosing a wide variety of conditions including fracture or dislocation of laryngeal cartilages and subcutaneous emphysema.[98]

The management of immediate postextubation tracheostenosis includes both medical and surgical approaches. Medical therapy includes inhaled beclomethasone, intralesional triamcinolone, and systemic methylprednisolone, resulting in edema resolution and improved inspiratory and expiratory flow.[99, 100] However, large granulomas must be removed endoscopically. In patients with significant postintubation fibrotic tracheostenosis, surgical therapy has been most successful with resection and end-to-end anastomosis of the trachea.[101] If the stenosis also involves the lower larynx, partial cricoid resection may be combined with tracheal resection. However, endoscopic laser surgery is becoming increasingly common in less pronounced tracheostenosis and in different types of laryngostenosis.[100]

TIMING OF TRACHEOSTOMY

One of the most controversial issues in intensive care medicine is the timing of tracheostomy to avoid complications from prolonged intubation. The incidence of complications is strongly correlated with duration of translaryngeal intubation, but prolonged intubation has been tolerated by the nasal route for up to 63 days and oral route for as much as 155 days and even longer in occasional patients.[59, 102] The optimal timing for tracheostomy has been suggested to range from 24 hours to 3 weeks of translaryngeal intubation based on incidence of complications, with no clear consensus.[103, 104] It has to be emphasized that the less traumatizing the tube, the longer that tracheal intubation may be tolerated. Thus, it is not possible to provide specific time limits for all kinds of tubes. Tube size and configuration play important roles regarding the advisable period of intubation in each individual case. Analysis of tracheostomy timing suggests that 3% of patients are intubated for fewer than 3 days, 29% for 3 to 7 days, 50% for 7 to 14 days, 10% for 14 to 21 days, and 6% for longer than 21 days.[105] Prospective clinical trials demonstrate fewer infection complications in patients with early tracheostomy (within 5 days).[106] Mechanical complications are increased in those patients who remain intubated for more than 7 days.[107] This mechanical complication rate is minimized for those tracheotomized immediately, with most laryngeal damage occurring after 1 week.[108] However, an early tracheostomy trial (3 days) revealed an eightfold increase in the frequency of infectious complications.[109]

We must realize that the complication rate of prolonged intubation may decrease because of improved airway care and tube design. Laryngeal damage of tracheal intubation must also be balanced against the early bacterial colonization and subsequent airway infection associated with tracheostomy. Recommendations suggesting a specific duration of tracheal intubation with the least traumatizing tube followed by tracheostomy should be individualized according to the patient's diagnosis, condition, predicted clinical outcome, and laryngeal appearance at repeated examination.

Clinical trials of available tracheal tubes allow us to draw some conclusions. An evaluation of 500 intubated patients revealed that use of low-cuff pressure, PVC tube composition, and an N_2O diffusion system results in fewer complications, such as voice change or odynophagia with an incidence of 51% and 24%, respectively.[110] Furthermore, the addition of a cuff pressure regulation system results in less tracheal damage and aspiration compared with a high-volume, low-pressure cuff without such pressure regulation system. Thus, the ideal tracheal tube cuff should be thin walled and of moderate compliance with a resting diameter that is 50% wider than the trachea. According to the International Standards Organization, the cuff resting diameter should be indicated on the unit package to allow proper choice.[111]

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