# Extubation and Reintubation of the Difficult Airway

RICHARD M. COOPER | SOFIA KHAN

# I. INTRODUCTION

Tracheal extubation has received relatively limited critical scrutiny compared with that accorded to intubation. Textbooks, reviews, and conferences focusing on the airway frequently ignore this aspect of management, despite the observation that airway complications are significantly more likely to be associated with extubation than intubation.[1] These complications range from the relatively minor, such as coughing and transient breath holding that have little impact on outcome, to those that are life-threatening. The American Society of Anesthesiologists (ASA) Closed Claims Project analyzed adverse respiratory events and found that 17% of brain injuries and deaths occurred after extubation in the operating room or postanesthesia care unit (PACU).[2] Complications associated with extubation in critical care areas are likely to occur more frequently and have more serious consequences.[3,4]

The ASA Task Force on Management of the Difficult Airway and the Canadian Airway Focus Group recommended a preformulated strategy for extubation of the difficult airway and an airway management plan for dealing with post-extubation hypoventilation.[5,6] This chapter classifies the complications associated with routine and more complex tracheal extubation or reintubation, proposes a risk stratification for extubation and reintubation in various clinical settings, and suggests strategies that may prove helpful in reducing serious complications or death.

Low-risk or routine extubations have been reviewed elsewhere and are not the focus of this chapter. Although they are dealt with only briefly, concerns include the adequacy of recovery from neuromuscular blockade, depressant effects of narcotics and residual volatile or intravenous sedatives, level of consciousness, hemodynamic stability, adequate ventilation and oxygenation, normothermia, freedom from noxious stimulation, and airway patency.[7-10] The controversy about deep versus awake extubation has been addressed elsewhere,[10-12] and most of the present discussion concentrates on extubation of more challenging airways and strategies that may increase the probability of success.

# II. EXTUBATION FAILURES AND CHALLENGES

Extubation fails when an attempt to remove a tracheal tube is unsuccessful. Reintubation failure occurs when extubation is followed by an immediate or delayed but unsuccessful attempt to reintubate the trachea. There is no agreement about how to define failure, and because there is no consensus about the time frame, reported incidences may vary. It is reasonable to consider the failed extubation in two separate clinical settings: the intensive care unit (ICU), where such failures are relatively common, and the operating room or PACU, where they are far less common.

In the ICU, the ability to predict readiness for extubation is imprecise despite a host of predictive criteria.[13-16] To minimize the risks, discomfort, and expense of prolonged intubation, a trial of extubation is occasionally attempted, but it may be followed sometime thereafter by a need to reintubate. The incidence of required reintubation ranges from 6% to 20%, and it depends on the clinical mix of patients,[15,17,18] their critical acuity, critical care resources, and the threshold levels for extubation. Even if reintubation is successful, patients who failed their initial extubation had increased rates of ICU mortality and cost of care, prolonged ICU stay, and hospital length of stay.[19] Compared with routine postoperative patients, intensive care patients are more likely to fail extubation because neurologic obtundation may leave them unable to protect their airways. Debilitation and impaired mucociliary clearance may interfere with pulmonary toilet, and diminished strength, altered pulmonary mechanics, increased dead space, and venous admixture may result in hypercapnic or hypoxemic respiratory failure.

Although the complications associated with extubation of postoperative patients may be more common than those associated with intubation, they rarely require reintubation. Studies involving a wide case mix of postoperative patients show a high degree of concordance regarding the incidence of required reintubation. Combining the results of four large studies enrolling more than 150,000 patients, the incidence ranged from 0.09% to 0.19%.[20-23] The reintubation rate appears to be significantly higher (1% to 3%) after selected surgical procedures such as panendoscopy [20] and a variety of head and neck procedures.[24-28]

Postoperative reintubation, although uncommon, may offer some unexpected challenges, including anatomic distortion, physiologic instability, incomplete information concerning the patient, lack of essential equipment and time, and inexperienced personnel. These problems may convert a previously easily managed airway into a life-threatening disaster. A difficult airway adequately managed during a controlled intubation is completely different from the difficult airway in an agitated, hypoxemic, and hypotensive patient.

# III. EXTUBATION RISK STRATIFICATION

Risks related to extubation fall into two broad categories: the risk of extubation failing and the risk of reintubation failing. Within each category, there is a continuum from low to high risk. Like the prediction of a difficult intubation, this is an inexact science; it is probably safest to err on the cautious side. Most extubations are expected and turn out to be uneventful, but even these routine extubations may be associated with complications (Box 50-1).

The causes of a failed extubation can be classified as a failure of oxygenation, failure of ventilation, inadequate clearance of pulmonary secretions, or loss of airway patency. We cannot always predict which patients will require reintubation, and if reintubation is likely to be difficult, it is prudent to employ strategies expected to maximize the likelihood of success. In this discussion, if reintubation is expected to be difficult, we regard such extubations as intermediate- or high-risk extubations, although such designations are based on probability and clinical judgment.

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**BOX 50-1** Complications of Routine Extubations

- Unintended extubation
- Fixation of endotracheal tube
- Hypertension, tachycardia
- Coughing, breath-holding
- Laryngeal injury
- Laryngospasm or vocal cord paralysis
- Stridor, airway obstruction
- Negative pressure pulmonary edema
- Laryngeal incompetence
- Aspiration

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## A. Routine Extubations

A retrospective database review from The University of Michigan analyzed 107,317 general anesthetics administered between 1994 and 1999.[23] It identified 191 required reintubations in the operating room or PACU, 112 (58.6%) of which were for respiratory reasons. The most common respiratory causes were hypercapnic or hypoxemic respiratory insufficiency (60%), respiratory obstruction (20.5%), and laryngospasm or bronchospasm (19.5%). Failed extubations from a respiratory cause occurred at a rate of 112/107,301 or 0.1%. Unintended extubation accounted for 25 of 191 reintubations, all of which occurred in the operating room. Surgical complications, including neck hematoma, pneumothorax, laryngeal nerve paralysis, and bleeding, accounted for 16 of 191 reintubations. Contrary to their expectations, the investigators found that excessive narcotics (9 cases) and prolonged neuromuscular blockade with pancuronium (2 cases) were responsible for only 11 of 191 reintubations. This self-reported information was retrospective, and sometimes required interpretation of the causes from the therapies applied (e.g., narcotic antagonists, additional doses of anticholinergics).

A prospective study from Thailand found a rate of reintubation occurring within 24 hours of 27 per 10,000 patients.[29] This was twice the rate observed in the University of Michigan study.[23] The precipitating factor in the Thai study was thought to be residual neuromuscular weakness in almost three quarters of cases requiring reintubation in the operating room or PACU. In a Taiwanese database that surveyed almost 138,000 patients undergoing general anesthesia between 2005 and 2007, 83 reintubations were performed after intended extubation.[30] Overall, this represented a rate of 6 reintubations per 10,000 patients. Comparing these patients with a matched cohort not requiring reintubation, the investigators identified the following factors as most predictive of a need for reintubation: chronic obstructive pulmonary disease (odds ratio [OR] = 7.17; 95% confidence interval [CI], 1.98 to 26.00), pneumonia (OR = 7.94; 95% CI, 1.03 to 32.78), ascites (OR = 13.86; 95% CI, 1.08 to 174.74), and systemic inflammatory response syndrome (OR = 11.90; 95% CI, 2.63 to 53.86).

### 1. Hypoventilation Syndromes

The ASA Closed Claims Project found that 4% of 1175 closed claims resulted from critical respiratory events in the PACU. The highest proportion was attributed to inadequate ventilation, and many of these patients died or suffered brain damage.[31]

Many clinical conditions may give rise to postoperative ventilatory failure. A multicenter, prospective survey in France that looked at almost 200,000 general anesthetics administered between 1978 and 1982 found that postoperative respiratory depression accounted for 27 of 85 respiratory complications that were life-threatening or had serious sequelae. These complications were responsible for seven deaths and five cases of hypoxic encephalopathy.[32] A respiratory rate of less than 8 breaths/min was observed by PACU nurses among 0.2% of 24,000 patients after general anesthesia.[22]

Hypoventilation may be mediated centrally at the level of the upper motor neuron, anterior horn cell, lower motor neuron, neuromuscular junction, or respiratory muscles. Clinical correlates include central sleep apnea, carotid endarterectomy,[33] medullary injuries, demyelinating disorders, direct injury to peripheral nerves, poliomyelitis, Guillain-Barré syndrome, motor neuron disease, myasthenia gravis, and botulism. Hypoventilation may result from the loss of lung or pleural elasticity, diaphragmatic splinting caused by abdominal pain or distention, thoracic deformities such as kyphoscoliosis, or multifactorial entities such as morbid obesity and severe chronic obstructive pulmonary disease. Rarely, hypercapnia results from excess carbon dioxide production or a marked increase in physiologic dead space.

The residual effects of anesthetic drugs contribute to inadequate postoperative ventilation.[34-36] It may be aggravated by incomplete reversal of neuromuscular blockers,[37] hypocalcemia or hypermagnesemia, or the administration of other drugs, including antibiotics, local anesthetics, diuretics, and calcium channel blockers, which may potentiate neuromuscular blockade.

### 2. Hypoxemic Respiratory Failure

A review of the many causes of postoperative hypoxemia is beyond the scope of this chapter, but it can occur because of hypoventilation, a low inspired oxygen concentration, ventilation-perfusion mismatch, right-to-left shunting, increased oxygen consumption, diminished oxygen transport, or rarely, an impairment of oxygen diffusion. These events are more likely in some clinical situations because of preexisting medical conditions or anesthetic and surgical interventions. If sufficiently severe, there may be a requirement for continuous positive airway pressure (CPAP) or reintubation and mechanical ventilation.

### 3. Inability to Protect the Airway

ICU or postoperative patients may be unable to protect the airway because of preexisting obtundation, neurologic injury, or the effects of residual anesthesia. In the latter case, it may be possible to temporize by turning patients on their sides, altering their positions (head up or head down) or by reversing residual medications with antagonists. These measures may not restore airway competency, and lack of resolution may necessitate reintubation.

### 4. Failure of Pulmonary Toilet

Inadequate clearance of pulmonary secretions may result from a depressed level of consciousness with impaired airway reflexes, overproduction of secretions, alteration of sputum consistency leading to inspissation and plugging, impaired mucociliary clearance, or inadequate neuromuscular reserve. These problems may result in atelectasis or pneumonia with attendant hypoxemic respiratory failure. Alterations in pulmonary mechanics may also lead to hypercapnia, necessitating reintubation.

### 5. Inadvertent Extubations

Inadvertent extubations may result from movement of or by the patient with an inadequately secured tracheal tube. Intraoperatively, this may occur in the prone position, when the airway is shared with the surgeon, when the head and neck are extended, when draping obscures the view, or when drapes adhere to the endotracheal tube (ETT) or circuit and are carelessly removed. In the ICU, extubation may occur when the patient is repositioned for a radiograph or routine nursing care. Patients insufficiently sedated are at greater risk for deliberate self-extubation,[38] but those more heavily sedated are more likely to require reintubation.[39] Fastidious attention to securing and supporting the ETT and breathing circuit is essential. Self-extubation may occur during emergence from anesthesia, when the patient is confused, agitated, and distressed, which promotes premature extubation. In the ICU, it may not be possible to know whether a self-extubation is accidental or deliberate, but many of these patients require reintubation,[40] are more likely to exhibit post-extubation stridor, and may need multiple intubation attempts, increasing the likelihood of esophageal intubation and death.[41-43]

### 6. Entrapment

The tracheal tube may become entrapped due to an inability to deflate the cuff,[44,45] or there may be difficulties with the pilot tube.[46,47] Difficulties include a crimped pilot tube, a defective pilot valve, and fixation of the tracheal tube by Kirschner wires,[48] screws,[49] ligatures,[50] or entanglement with other devices.[51,52] Entrapment can also occur during a percutaneous tracheostomy.[53] Mechanical obstruction of an entrapped tube is a life-threatening complication. Partial transection of the tracheal tube by an osteotome during a maxillary osteotomy has resulted in the partially cut tube forming a barb that caught on the posterior aspect of the hard palate.[54] One report of tube entrapment with fatal consequences involved a Carlens tube that was inadvertently sutured to the pulmonary artery.[55] Lang and colleagues recommended routine intraoperative testing for tracheal tube movement when fixation devices are used in proximity to the airway.[49] Uncertainty about tube movement should prompt fiberoptic examination before emergence from general anesthesia.

### 7. Hypertension and Tachycardia

Transient hemodynamic disturbances accompany extubation in most adults. These responses may be prevented by deep extubation,[56] insertion of a laryngeal mask airway (LMA) before emergence, or attenuated by concurrent medication.[57-59] Most healthy patients not on antihypertensive agents or other cardioactive drugs exhibit increases in heart rate and systolic blood pressure of more than 20% in association with extubation.[60] After coronary artery bypass surgery, these changes tend to be transient, lasting 5 to 10 minutes, and they usually are not associated with electrocardiographic evidence of myocardial ischemia.[61] Coronary sinus lactate extraction measurements, however, indicate that among patients with poor cardiac function, extubation may be associated with myocardial ischemia.[62] Patients with inadequately controlled hypertension, carcinoid, pheochromocytoma, hypertension associated with pregnancy, or hyperthyroidism may be expected to display even greater increases in blood pressure in response to tracheal extubation. The specific strategies needed to attenuate these usually transient changes are dictated by the clinical context. The strategies, which are not universally effective, include the use of intracuff,[63-65] intratracheal,[66] or intravenous lidocaine[61,67,68]; beta-blockers[60,69-71]; dexmedetomidine[72,73]; and nitrates.

### 8. Intracranial Hypertension

Tracheal intubation and suctioning are associated with a rise in intracranial pressure. Extubation probably is associated with comparable or even greater increases in intracranial pressure. There is evidence, albeit contradictory, that intravenous and endotracheal lidocaine attenuate this effect.[74,75]

### 9. Intraocular Pressure

Madan and colleagues compared the intraocular pressure changes of tracheal intubation and extubation in children with and without glaucoma.[76] They observed significantly greater increases 30 seconds and 2 minutes after deep extubation compared with the corresponding times after uncomplicated intubations. These differences were seen in both groups of children. It is likely that significant increases in intraocular pressure observed after deep extubation would have been even higher had extubation occurred after recovery of consciousness. Lamb and coworkers observed similar effects of extubation on intraocular pressure in adults and commented that this increase could be prevented by using an LMA rather than a tracheal tube.[77]

### 10. Coughing

Coughing on emergence from general anesthesia is virtually ubiquitous, particularly when an ETT is used.[78] No difference between smokers and nonsmokers is observed. Although coughing is a protective reflex, it can be particularly troublesome in the setting of ophthalmologic, neurologic, oropharyngeal, and neck surgery.

Several strategies have been proposed to minimize coughing, including deep endotracheal extubation, primary use of or conversion to an LMA,[57,79,80] use of the sedative dexmedetomidine,[73] intravenous or topical application of a local anesthetic to the vocal folds, and use of intracuff lidocaine.[63,65,67,81] However, coughing on emergence is common and relatively benign for most patients.

The emergence of severe acute respiratory syndrome (SARS) and the high prevalence of drug-resistant droplet or airborne diseases in some locales make coughing potentially hazardous to the airway manager. In 2003, Toronto was the North American epicenter for SARS, and coughing assumed life-threatening proportions for medical personnel.[82-87] Patients with cough, fever, and pulmonary infiltrates were dangerous, and coughing on emergence was not protective, but rather posed a threat by dispersing infectious respiratory droplets on those in the patient's vicinity. The strategies adopted at that time have been largely relaxed, although they may again become necessary when new risks threaten patients and care providers.

## 11. Laryngeal Edema

Several of the complications of endotracheal intubation do not become apparent until after extubation occurs.[88] Glottic or tracheal injury may occur despite a good laryngeal view or during awake fiberoptic intubation.[89-91] Anatomic or functional laryngeal problems are more likely to develop as a consequence of a difficult or prolonged intubation attempts.[22] Possible airway injuries include laryngeal edema, laceration, hematoma, granuloma formation, vocal fold immobility, and dislocation of the arytenoid cartilages.[92]

Glottic edema has been classified as supraglottic, retroarytenoidal, and subglottic.[93] Supraglottic edema may result in posterior displacement of the epiglottis, reducing the laryngeal inlet and causing inspiratory obstruction. Retroarytenoidal edema restricts movement of the arytenoid cartilages, limiting vocal cord abduction on inspiration. Subglottic edema, a particular problem in neonates and infants, results in swelling of the loose submucosal connective tissue that is confined by the nonexpandable cricoid cartilage. In neonates and small children, this is the narrowest part of the upper airway, and small reductions in diameter result in a significant increase in airway resistance. In children, laryngeal edema is promoted by a tight-fitting ETT, traumatic intubation, intubation longer than 1 hour, coughing on the tracheal tube, and intraoperative alterations of head position.[94] Koka and coworkers found an incidence of 1% among children younger than 17 years. Laryngeal edema should be suspected when inspiratory stridor develops within 6 hours of extubation. Management of laryngeal edema depends on its severity. Treatment options include head-up positioning, supplemental humidified oxygen, racemic epinephrine, helium-oxygen administration, reintubation, and tracheostomy.

Clinical studies of children and adults that evaluated the role of prophylactic corticosteroids in the prevention of post-extubation stridor have yielded contradictory findings.[95-98] In the setting of adult ICUs, a large, multicenter, prospective, randomized, double-blind trial of methylprednisolone (20 mg administered 12 hours before and every 4 hours until extubation) versus placebo found that steroids significantly reduced post-extubation laryngeal edema (11 of 355 versus 76 of 343, $P < 0.0001$) and required reintubation due to laryngeal edema (8% versus 54%, $P = 0.005$). Laryngeal edema was defined by any two of stridor, inspiratory prolongation, and use of

accessory muscles. It was classified as severe if reintubation was required within 36 hours. Laryngoscopy was not performed unless reintubation was required. Factors associated with a greater risk of laryngeal edema included female sex, shorter height, larger-diameter ETT, admission for trauma, and shorter duration of intubation. The absence of pretreatment with methylprednisolone was associated with a hazard ratio of more than 8.[99] It is possible that the benefits of steroids are restricted to high-risk populations and require administration of multiple doses.[100, 101] Contradictory findings may relate to risk factors in the study populations and variability of dosage regimens.

An alternative classification has been proposed for laryngotracheal injury after prolonged intubation.[102-104] Immediate post-extubation airway obstruction results from glottic and subglottic granulation tissue, which may swell on removal of the ETT. Posterior glottic and subglottic stenosis due to contracting scar tissue results in increasing obstruction weeks or months after extubation. Benjamin found that fiberoptic evaluation or laryngoscopy with the tube in situ was of limited value.[102] An ETT obscures the view of the posterior glottis and subglottis. These lesions were best identified using rigid telescopes with image magnification during general anesthesia. This approach permitted anticipation of problems and development of a management strategy.

## 12. Laryngospasm

Laryngospasm is believed to be a common cause of post-extubation airway obstruction, particularly in children.[105] Even in adults, Rose and colleagues found that it accounted for 23.3% of critical postoperative respiratory events, although the diagnosis was presumptive.[106] Olsson and Hallen observed an increased incidence among patients presenting for emergency surgery, those requiring nasogastric tubes, and patients undergoing tonsillectomy, cervical dilation, hypospadias correction, oral endoscopy, or excision of skin lesions.[105] A variety of triggers are recognized, including vagal, trigeminal, auditory, phrenic, sciatic, and splanchnic nerve stimulation; cervical flexion or extension with an indwelling ETT; or vocal cord irritation from blood, vomitus, or oral secretions.[107] A risk assessment questionnaire was used in a study to prospectively evaluate almost 10,000 children undergoing general anesthesia. A positive history of nocturnal dry cough, exertional wheezing, or more than three wheezing episodes in the prior 12 months was associated with a fourfold increase in the risk of laryngospasm in the PACU and a 2.7-fold increased risk of airway obstruction during surgery or in the PACU.[108] Twice as many children were managed with an LMA than an ETT, and an equal number had their devices removed awake and asleep. The depth of anesthesia at the time of device removal did not influence the incidence of laryngospasm.

Laryngospasm involves bilateral adduction of the true vocal folds, vestibular folds, and aryepiglottic folds that outlasts the duration of the stimulus. This is protective to the extent that it prevents aspiration of solids and liquids. It becomes maladaptive when it restricts ventilation and oxygenation. The intrinsic laryngeal muscles are the main mediators of laryngospasm, and they include

the cricothyroids, lateral cricoarytenoids, and thyroarytenoid muscles. The cricothyroid muscles are the vocal cord tensors, an action mediated by the SLN. Management of laryngospasm consists of prevention by either extubating at a sufficiently deep plane of anesthesia or awaiting recovery of consciousness.[56] Potential airway irritants should be removed and painful stimulation should be discontinued. If laryngospasm occurs, oxygen by sustained positive pressure may be helpful, although this may push the aryepiglottic folds more tightly together.[109] Larson described a technique of applying firm digital pressure anteriorly directed to the "laryngospasm notch" between the ascending mandibular ramus and the mastoid process and observed that this technique is rapid and highly effective.[110] Very small doses of a short-acting neuromuscular blocker with or without reintubation may be necessary.[111,112]

## 13. Macroglossia

Massive tongue swelling may complicate prolonged posterior fossa surgery performed with the patient in the sitting, prone, or park bench position.[113-116] It is also seen with very steep or prolonged Trendelenburg positioning, hypothyroidism, acromegaly, lymphangioma, idiopathic hyperplasia, metabolic disorders, amyloidosis, cystic hygroma, neurofibromatosis, rhabdomyosarcoma, sublingual or submandibular infections, and chromosomal abnormalities such as the Beckwith-Wiedemann syndrome.[117]

The most common and dramatic presentation of macroglossia results from angioedema.[118] It can be congenital or acquired. Hereditary angioedema results from a deficiency of C1 esterase inhibitor; acquired C1 esterase deficiency is associated with histamine release, a physical stimulus, or most commonly, a reaction to angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.[118,119] Although involvement of the tongue is the most obvious manifestation, the uvula, soft tissues, and larynx may also be involved.

In the ICU setting, macroglossia may be seen as a complication of extreme volume overloading or tongue trauma, particularly when it is further complicated by a coagulopathic state. If this occurs or progresses after extubation, it can lead to partial or complete airway obstruction, making reintubation necessary but difficult or impossible.[114] Lam and Vavilala postulate that positioning in most cases results in venous compression leading to arterial insufficiency and subsequent reperfusion injury.[115] Alternatively, local compression may cause venous or lymphatic obstruction with resultant immediate and typically milder tongue swelling. The latter form is less severe but more apparent, and extubation is likely to be postponed.

## 14. Laryngeal or Tracheal Injury

Airway injuries, such as lacerations, edema, arytenoid dislocation, and vocal fold paralysis, may occur from the lips to the distal trachea. The lip or tongue may become entrapped between the laryngoscope blade and the mandibular teeth, resulting in swelling or bleeding, although this is unlikely to be severe enough to complicate extubation. The glottis may be injured as a result of insertion of a round tube through a triangular opening. The trachea can be lacerated or penetrated by the ETT or its introducer or by ischemic compression by the cuff on the tracheal mucosa. Palatopharyngeal injuries have been described as a consequence of blind insertion of an ETT during video laryngoscopy.[120-127] Although these injuries are not often apparent at the time of laryngoscopy, they have been managed conservatively and should not complicate extubation. The epiglottis can be downfolded during intubation, but the consequences of downfolding, even if prolonged, are unknown.[128]

Laryngeal injuries accounted for 33% of all airway injury claims and 6% of all claims in the ASA Closed Claims Project database.[129] They range from transient hoarseness to vocal fold paralysis. Even when direct laryngoscopy provides a satisfactory glottic view or intubation is facilitated by fiberoptic instrumentation,[89,91] airway injury can occur and go unsuspected until after the ETT is removed. Airway injuries are presumed to be less likely if intubation is easy, but analysis of the ASA Closed Claims Project revealed that 58% of airway trauma and 80% of laryngeal injuries were associated with intubations that were not difficult.[129,130] Judging from the findings of the Closed Claims Project, difficult intubations were more likely to result in pharyngeal and esophageal than tracheal injuries.

Vocal fold immobility may result from injury to the recurrent laryngeal nerve or the arytenoid cartilages.[92,131-139] Arytenoid immobility has resulted from seemingly uneventful Macintosh and McCoy direct laryngoscopy,[137,140] double-lumen tube insertion, and lightwand intubation.[134] The mechanism of this injury is uncertain. It may be a consequence of a subluxation or a hemarthrosis with subsequent resolution or fixation. Prolonged or stressful contact between the ETT and the posteromedial aspects of the vocal cords, arytenoids, or posterior commissure may result in ulceration of the perichondrium, which heals with fibrous adhesions that produce an immobile glottis. This complication may be more common than the literature indicates.[92,141] Persistent post-extubation hoarseness, a breathy voice, and an ineffective cough should prompt assessment by an otolaryngologist. The diagnosis is confirmed by endoscopic visualization of an immobile vocal cord associated with a rotated arytenoid cartilage.[88] If the diagnosis is made early, before the onset of ankylosis, it may be possible to manipulate the arytenoid back into position.

Vocal fold paralysis results from injury to the vagus or one of its branches (i.e., recurrent laryngeal nerve [RLN] or external division of the superior laryngeal nerve [ex-SLN]) and may resemble arytenoid dislocation or ankylosis. Differentiation may require palpation of the cricoarytenoid joints under anesthesia or laryngeal electromyography.[88] When vocal fold paralysis occurs as a surgical complication, it is usually associated with neck, thyroid, or thoracic surgery. The left RLN can also be compressed by thoracic tumors, aortic aneurysmal dilatation, left atrial enlargement, or during closure of a patent ductus arteriosus. Occasionally, a surgical cause cannot be implicated. Cavo and coworkers postulated that an overinflated ETT cuff might result in injury to the anterior divisions of the RLN.[142]

The RLN supplies all of the intrinsic laryngeal muscles except the cricothyroid, the true vocal cord tensor, which is innervated by the ex-SLNs. Unilateral ex-SLN injury results in a shortened, adducted vocal fold with a shift of the epiglottis and the anterior larynx toward the affected side. This produces a breathy voice but no obstruction and usually resolves within days to months. Bilateral ex-SLN injury causes the epiglottis to overhang, making the vocal folds difficult to visualize. If seen, they are bowed. This produces hoarseness with reduction in volume and range but no obstruction. Unilateral RLN injury causes the vocal fold to assume a fixed paramedian position and produces a hoarse voice. There may be a marginal airway with a weak cough. Bilateral RLN injury results in both vocal folds being fixed in the paramedian position and inspiratory stridor, often necessitating a surgical airway.[143]

Pharyngeal, nasopharyngeal, and esophageal injuries include perforation, lacerations, contusions, and infections. These injuries may be associated with difficult laryngoscopy or intubation, but they may also result from the blind passage of a gum elastic bougie,[144] nasogastric tube,[145] nasotracheal tube,[146] suction catheter, esophageal stethoscope transesophageal echo probe, or temperature probe.[147] Penetrating injuries can communicate with the esophagus, resulting in a tracheoesophageal fistula, or with the mediastinum, which may go unrecognized and result in mediastinitis, retropharyngeal abscess, and death.[131] After a brief intubation, soft tissue injuries resulting in airway obstruction are more likely to result from edema or hematoma than infection. Most of the described injuries do not significantly complicate extubation. Laryngeal and tracheal stenoses are serious complications, but they are rarely evident at the time of extubation.

## 15. Airway Injury

Burn patients can have intrinsic and extrinsic airway injuries. Circumferential neck involvement is an example of an extrinsic injury. Smoke inhalation or thermal injuries are examples of intrinsic injuries. Burn patients are at particular risk of requiring reintubation. They can have bronchorrhea, impaired mucociliary clearance and local defenses, laryngeal and supraglottic edema, increased carbon dioxide production, and progressive acute respiratory distress syndrome. Carbon monoxide may also diminish their level of consciousness and the ability to protect their airway. It may be difficult to secure the tracheal tube because of involvement of the adjacent skin, and burn victims may be agitated or uncooperative, increasing the risk of unintended extubation.[148] Kemper and coworkers reported their management of 13 burn patients younger than 15 years, 7 of whom exhibited post-extubation stridor. Patients treated with helium-oxygen mixtures had lower stridor scores than patients treated with an air-oxygen mixture.[149] They found that 11 of 30 extubated burn victims required treatment for stridor after extubation, consisting of racemic epinephrine, helium-oxygen, reintubation ($n = 5$), or tracheostomy ($n = 1$). The absence of a cuff leak was considered to be the best predictor of failure, with a sensitivity of 100% and a positive predictive value of 79%.[149]

A variety of conditions may lead to airway edema severe enough to result in post-extubation stridor. It occurred in 2% to 37% of ICU patients after "prolonged intubation."[150] A test was sought to predict patients with sufficient airway swelling to compromise safe extubation. The cuff-leak test was initially proposed for children with croup.[151] The concept is that marked airway swelling is likely if air does not escape around the deflated ETT cuff when the ETT is occluded as the patient exhales. The cuff-leak test was the best predictor of successful extubation in a pediatric burn and trauma unit, and although sensitive, it was not specific for predicting stridor, necessitating reintubation in 62 adults.[152,153]

The test has been refined by evaluating cuff-leak volume as the difference between inspiratory and expiratory tidal volumes during assist-control ventilation after cuff deflation.[41,154] Two studies found the cuff-leak volume could predict post-extubation stridor. In one study, 8 of 45 patients exhibited stridor, 4 of whom required reintubation.[154] In the other study involving 88 adult medical ICU patients, 6 patients exhibited stridor, 3 of whom required reintubation.[41] They observed a significantly smaller cuff leak in patients who subsequently developed stridor, concluding that this measurement was the best predictor of the presence or absence of stridor. However, a study of 561 consecutive cardiothoracic patients who were extubated within 24 hours (median, 12 hours) defined a the cuff leak as the difference between the inspired and expired tidal volumes during assist-controlled ventilation. None of the (20) patients with leaks <110 mL developed stridor whereas one patient with a leak of 350 mL did and required reintubation. This led the investigators to conclude that the quantitative cuff-leak test was not reliable in this patient population.[155]

In another study involving 110 trauma victims, cuff leaks of less than 10% had a 96% specificity for predicting post-extubation stridor or the need for reintubation.[156] De Bast and colleagues studied 76 adults in a combined medical-surgical ICU.[157] An equal number of patients had been intubated for more or less than 48 hours. Assist-control ventilation was reinstituted after cuff deflation, and the percentage of cuff leak was determined. Receiver operating characteristic (ROC) curves yielded a cutoff value of 15.5% cuff leak to equalize the false positives and false negatives for reintubation due to laryngeal edema, but this study was relatively small. Among patients intubated for more than 48 hours, only 2 of 22 with large-volume leaks and 6 of 16 with small-volume leaks required reintubation for laryngeal edema.

Kriner and colleagues evaluated the cuff-leak test for its ability to predict post-extubation stridor among 462 adult patients intubated for longer than 24 hours.[150] They evaluated the two thresholds previously described; a positive test was defined as a cuff-leak volume of 110 mL or less or of 15.5% or less of the exhaled tidal volume. Ten of 82 patients with leak volumes of 110 mL or less developed post-extubation stridor, and 10 of 380 with larger leaks developed post-extubation stridor, giving a positive predictive value for stridor of 0.12 and negative predictive value of 0.97. The sensitivity and specificity of the test under these conditions were 0.50 and 0.84, respectively.[150] With leak volumes of 15.5% or less, 7 of 48

patients developed post-extubation stridor, and 13 of 414 patients with volumes greater than 15.5% developed stridor, yielding positive and negative predictive values of 0.15 and 0.97, respectively. The sensitivity and specificity of this test for post-extubation stridor were 0.35 and 0.91, respectively. The prevalence of post-extubation stridor was 20 of 462. Seven of these patients required reintubation; 15 were managed with racemic epinephrine or helium-oxygen mixtures, or both, and 2 of the 15 failed, requiring intubation. The investigators concluded that neither threshold adequately predicted post-extubation stridor or could justify delaying extubation.

The reported variability of post-extubation stridor (2% to 37%) results in part from inconsistent diagnostic criteria,[150] but undoubtedly there are many other factors at play. Factors increasing the risk of post-extubation stridor include longer duration of intubation,[150] trauma and burns,[14,148,154,158] pediatric age group,[158] female gender,[95,97,150] traumatic or emergent intubations, periods of hypotension, agitation, persistent attempts at phonation, inadequate ETT fixation, aggressive tracheal suctioning, highly positive fluid balance, low plasma oncotic pressure, gastroesophageal reflux, increased ETT diameter, and the presence of a nasogastric tube.[95,97,150]

In summary, it appears that despite the intuitive appeal of the cuff-leak test, neither the qualitative nor quantitative test adequately predicts adult patients who will develop stridor after extubation.[150,155,159] It did not predict where patients were likely to require reintubation. Deem argued that absence of a cuff leak might result in an unnecessary delay of extubation, whereas a large cuff leak might produce false reassurance that there will be no difficulties.[159] At least in adults, the value of this test is uncertain.

### 16. Postobstructive Pulmonary Edema

Severe airway obstruction from any cause may complicate extubation and lead to postobstructive pulmonary edema, also called negative-pressure pulmonary edema.[160] This edema occurs when a forceful inspiratory effort is made against an obstructed airway (i.e., Mueller maneuver), often a closed glottis, generating large negative intrapleural pressures that promote venous return. The increase in venous pressure is aggravated by a lowered alveolar pressure, resulting in transudation of fluids into the pulmonary interstitium and alveoli. It may also result in a rightward shift of the interatrial and interventricular septa, raising left atrial and ventricular pressures. Some instances may be complicated by a permeability defect with exudative fluid and inflammatory cells.[161-166]

Postobstructive pulmonary edema usually occurs in adult patients with upper airway tumors, severe laryngospasm, or rarely, bilateral vocal cord palsy,[167] whereas in children, it occurs most commonly as a complication of croup or epiglottitis.[168] The onset may be within minutes of the development of airway obstruction. It typically resolves with relief of the obstruction and supportive treatment for pulmonary edema.[166]

### B. Higher-Risk Extubations

Although the previously described complications may follow a routine extubation, two additional groups of patients may be affected: those with a higher risk for reintubation and those in whom accomplishing reintubation can be challenging or impossible. Patients at higher risk for reintubation have preexisting medical conditions that reduce their physiologic reserve, ranging from moderate disability to an extremely marginal state. The possibility of higher-risk extubations also exists for a continuum of patients, from those in whom mask ventilation and reintubation should be easily achieved to those in whom both would pose a significant challenge. They may have their jaws wired shut or have a neck that is very poorly suited for emergent surgical access.[169,170]

Chronic pulmonary or cardiac disease may compromise spontaneous ventilation and necessitate intubation. The patient with an ineffective cough or increased secretions may have a need for pulmonary toilet. An obtunded patient may be unable to protect his airway. A list of higher-risk extubations is provided in Table 50-1.

Any reintubation is fundamentally different from the original intubation, because it is likely to occur in an urgent or emergent setting with limited information, personnel, and equipment. The patient is more likely to be hypoxic, acidotic, agitated, and hemodynamically unstable, and the procedure may be done in haste by the available personnel. A preemptive strategy is appropriate to manage these patients.

## IV. CLINICAL SETTINGS OF COMPLICATIONS

### A. Operative Conditions

#### 1. Laryngoscopic Surgery

Mathew and colleagues looked at 13,593 consecutive PACU admissions from 1986 through 1989.[21] Twenty-six (0.19%) of these patients required reintubation while in the PACU; seven of them had undergone ear, nose, and throat procedures. Of the seven patients, three had laryngeal edema, one was obstructed from a large thyroid, two bled at the operative site, and one developed postobstructive pulmonary edema after a tonsillectomy.

Patients undergoing laryngoscopy and panendoscopy (i.e., laryngoscopy, bronchoscopy, and esophagoscopy) are at an increased postoperative risk for airway obstruction and are approximately 20 times as likely to require reintubation as patients undergoing a wide variety of other surgical procedures.[20] Reviewing the records of 324 diagnostic laryngoscopies and 302 panendoscopies, Hill and colleagues found that patients who had undergone laryngeal biopsy were at the greatest postoperative airway risk. Thirteen (5%) of 252 patients required reintubation, most within 1 hour of extubation. Twelve of 13 had undergone laryngeal biopsy. Most of these patients had chronic obstructive pulmonary disease, and their need for reintubation was attributed largely to this.

Robinson prospectively studied 183 patients who had 204 endoscopic laryngeal procedures.[171] Seven patients had tracheostomies before or after their surgery because of high-risk airways. Two of the remaining patients developed postoperative stridor; one required reintubation, and the other required a delayed tracheostomy. Indirect

**TABLE 50-1**

**Complications of Higher-Risk Extubations**

| Complication | Surgical and Medical Settings |
|---|---|
| **Inability to Tolerate Extubation and Required Reintubation** | |
| Airway obstruction | Laryngeal injury or hypopharyngeal swelling after laryngoscopy |
| | Paradoxical vocal cord motion |
| | After thyroidectomy, anterior cervical surgery, or carotid artery surgery |
| | Wound swelling, hematoma |
| | Vocal cord injury (e.g., recurrent laryngeal nerve) |
| | Hypoglossal nerve injury |
| | After palatoplasty |
| | Maxillofacial swelling |
| | Macroglossia |
| | Obstructive sleep apnea |
| | Rheumatoid arthritis |
| | Parkinson's disease |
| | Prolonged intubation |
| Inadequate ventilation | Central sleep apnea |
| | Severe chronic obstructive pulmonary disease |
| | Residual sedation or neuromuscular blockade |
| | Preexisting neuromuscular disorder |
| | Diaphragmatic splinting |
| | Relative hypoventilation (e.g., increased $CO_2$ production) |
| Inadequate oxygenation | Inadequate inspired oxygen concentration |
| | Ventilation-perfusion mismatch |
| | Right-left shunt |
| | Increased $O_2$ consumption |
| | Decreased $O_2$ delivery |
| | Impaired pulmonary diffusion |
| Failure of pulmonary toilet | Obtundation |
| | Pulmonary secretions |
| | Quantity |
| | Quality (inspissated) |
| | Impaired mucociliary clearance |
| | Neuromuscular impairment |
| Inability to protect airway | Obtundation |
| | Neuromuscular disorder |
| **Difficulty Reestablishing the Airway** | |
| Airway injury | Thermal injury, smoke inhalation |
| | Blood or trauma obscuring the view |
| | Blood or trauma obstructing the airway |
| Previous airway difficulties | Known prior difficulties (e.g., multiple attempts, devices, or operators) |
| | Cormack-Lehane class $\geqq$3 for laryngeal view |
| Limited airway access | Maxillomandibular fixation |
| | Cervical immobilization, unstable cervical spine, or halo fixation |
| | Tracheal resection (e.g., guardian suture) |
| | Major head and neck surgery |
| Emergent setting | Lack of knowledge regarding prior or potential difficulties |
| | Lack of expertise |
| | Insufficient time to prepare personnel, equipment, and medications |

laryngoscopy carried out 4 to 6 hours after surgery revealed mucosal hemorrhage or laryngopharyngeal swelling in 32% of cases. Because the patients undergoing tracheostomy were not described, it is possible that the low incidence of reintubation resulted from an aggressive approach to preemptive tracheostomy.

## 2. Thyroid Surgery

A variety of airway-related injuries can be associated with thyroidectomies, including SLN and RLN injuries, wound hematoma, and tracheomalacia. Lacoste and colleagues retrospectively reviewed the records of 3008 patients who underwent thyroidectomies between 1968 and 1988.[27] The RLN had been identified intraoperatively in 2427 of these patients. Indirect laryngoscopy was performed on the third or fifth postoperative day. The RLN was damaged in 0.5% of patients with benign goiters and 10.6% of patients with thyroid cancer. RLN injury produces hoarseness, persistent coughing with phonation, and risk of aspiration. Unilateral RLN palsy was observed in 1.1% of patients. Three patients had bilateral RLN palsy and required tracheostomy. Six of a total of 16 deaths during the first 30 postoperative days were attributed to respiratory complications. One death occurred after failed intubation due to a deviated, constricted trachea. A second death was attributed to difficulties performing a tracheostomy. Two deaths resulted from aspiration or pneumonia, possibly related to RLN dysfunction.

SLN injury is more challenging to diagnose. It produces dysphonia and vocal fatigue, particularly in the higher registers. In a 5-year, multicenter study involving 42 centers and almost 15,000 thyroid operations, the diagnosis was suspected in 3.7% and confirmed in 0.4% of patients.[172]

Local hemorrhage or hematoma occurs postoperatively in 0.1% to 1.6% of patients undergoing thyroid surgery and in 0.36% of the patients cared for by Lacoste and colleagues.[27,172-174] These complications occurred 5 minutes to 3 days postoperatively. Re-exploration within the first day was required only twice. Airway obstruction may result from significant laryngeal and pharyngeal edema, and wound evacuation may be of limited value in the relief of airway obstruction.[173,175,176] It may result from or be aggravated by ligature slippage, coughing, vomiting,[174] coagulopathies, and reoperation.[173] The prophylactic placement of surgical drains likely reduces the incidence of this complication. Wound evacuation may result in significant improvement; however this is not always the case. If time permits or intubation fails, wound evacuation should be considered.[176] Laryngeal edema may persist after the wound has been evacuated, necessitating postoperative intubation.

Tracheomalacia is rarely diagnosed after thyroidectomies, even in patients with significant retrosternal tracheal compression, although it may exist subclinically.[177-181] Although symptoms, computed tomography (CT), and pulmonary function test results make it easy to recognize airway compression preoperatively, tracheomalacia may be difficult to predict or even detect from the surgical field, and it does not become apparent until after the ETT is removed and spontaneous ventilation has resumed.[182]

## 3. Carotid Artery Surgery

Neck swelling or hematoma formation after carotid endarterectomy may be relatively common. The New York Carotid Artery Surgery (NYCAS) study analyzed 9308 procedures performed between 1998 and 1999 at 167 hospitals.[183] A hematoma was identified in 5% of patients, substantially increasing the risk of death (OR = 4.30; 95% CI, 2.72 to 5.00) and stroke (OR = 3.89; 95% CI, 2.82 to 5.38). Hematoma occurrence reported in the literature ranges from 1.2% to 12%, depending on the definition used.[183] The overall rate of wound hematomas in the North American Symptomatic Carotid Endarterectomy Trial (NASCET), involving 1415 patients was 7.1%, 3.9% of which cases were considered to be mild (i.e., no delay in discharge), 3.0% were moderate (i.e., delay in discharge), and 0.3% were severe (i.e., permanent functional disability or death). The moderate and severe cases required re-exploration or wound evacuation (3.3%). Hematoma contributed to the death of four patients.[184] When wound hematomas are identified by a comparison of preoperative and postoperative CT scans, it occurs far more frequently (26%).[185] The postoperative reintubation or exploration rate is 1% to 3.3%.[184,186]

Kunkel and colleagues described 15 patients who developed wound hematomas after carotid endarterectomy.[187] Eight of these were evacuated under local anesthesia. In six of seven cases in which general anesthesia was induced before opening the wound, difficulties arose with airway management, resulting in two deaths and one patient with severe neurologic impairment. O'Sullivan and coworkers reported a similar experience for six patients with airway obstruction after carotid endarterectomy.[188] Stridor was not relieved by wound evacuation. Administration of muscle relaxants made manual mask ventilation and tracheal intubation virtually impossible due to marked glottic or supraglottic edema. Cyanosis and extreme bradydysrhythmias or asystole occurred in four patients. The providers endorsed Kunkle's recommendation for wound evacuation but thought that much of the airway compromise was caused by edema from venous or lymphatic congestion. They emphasized that the outward appearance may lead to an underestimation of the situation's gravity. Voice changes are early signs of danger and may be relatively subtle. Rapid clinical deterioration can occur after stridor develops.[189]

Studies by Carmichael and colleagues provided additional evidence for the role of swelling and bleeding in a small but elegant study. They compared the CT scans of 19 patients before and after carotid endarterectomy surgery.[185] Clinically, 1 patient had severe swelling, 4 had moderate swelling, and 3 had mild swelling, but 10 were deemed normal. However, postoperative CT scans demonstrated significant swelling of the retropharyngeal space and a reduction of the anteroposterior and transverse airway diameter, particularly at the level of the hyoid. Compared with preoperative CT scans, the calculated volume reduction averaged 32% ± 7% for extubated patients. The scans revealed a wound hematoma estimated to be greater than 10 mL (range, 44 to 94 mL) in 5 of 19 patients. Patients who remained intubated postoperatively showed a significantly greater volume reduction of 62% ± 9% ($P < 0.025$). Those remaining intubated as a result of swelling had contralateral extension of the swelling. These observations may help to explain why opening the wound frequently fails to provide benefit in many patients. Nonetheless, it may be difficult to clinically differentiate bleeding from swelling. After radiologic demonstration of swelling, the same group evaluated the benefits of prophylactic dexamethasone but failed to demonstrate any clinical benefit.[190]

A 10-year, retrospective review of 3224 carotid endarterectomies performed at The Mayo Clinic revealed that 44 (1.4%) patients required wound exploration within 72 hours of surgery, despite the nonreversal of heparin.[191] In two patients, re-exploration occurred before the initial extubation. The decision to re-explore was made in the PACU for 7 patients; the remaining 35 were identified in the ICU or ward. Only one patient required a surgical airway when direct laryngoscopy failed in the ICU. Several techniques were initially employed: awake bronchoscopic intubation (15 of 20 of which were successful), direct laryngoscopy after induction (13 of 15 were successful), and awake direct laryngoscopy (5 of 7 were successful). When awake bronchoscopy failed, direct laryngoscopy was successful whether the patient was awake (3 of 3) or asleep (2 of 2). When direct laryngoscopy initially failed after induction in two patients, it succeeded after opening the incision. When awake direct laryngoscopy failed, one patient required a surgical airway; in the other, direct laryngoscopy succeeded after opening the incision. Despite the size of this series, it is not possible to draw conclusions about which techniques are most successful. Success likely depends on the skill and judgment of the airway manager. It is also possible that this study differed from the other studies in that the decision to re-explore was made earlier and patients had less airway distortion.

Several nerve injuries can result from carotid artery surgery or the anesthetic technique. The range reported in the literature is 3% to 23%, although most of these cases resolve within 4 months of surgery.[192] In the NYCAS study, cranial nerve palsies occurred in 514 (5.5%) of 9308 patients and involved, in descending order, the hypoglossal nerve (170), producing tongue deviation to the operative side; a branch of the facial nerve (126), resulting in lip or facial droop; the glossopharyngeal nerve (41); a branch of the vagus, which may involve the RLN and produce vocal cord paresis (31); the trigeminal nerve (19); a branch of the cervical plexus (10); or more than one nerve group (117).[183]

Bilateral vocal cord and bilateral hypoglossal nerve palsies have been described after staged, bilateral carotid endarterectomies.[24,25] In the latter case, the first procedure, performed under regional anesthesia, had been complicated by a wound hematoma, resulting in numbness over the anterior neck and diminished sensation in the C2 and C3 distribution. The subsequent endarterectomy, done 4 weeks later under deep cervical plexus block with subcutaneous infiltration, caused intraoperative airway obstruction and asystole. The airway was secured, but repeated attempts at extubation resulted in persistent obstruction due to bilateral hypoglossal nerve palsy. In another case, performed under cervical plexus

block, the patient developed bilateral vocal cord paralysis that required intubation and subsequent tracheotomy. It is suspected that she had a previously unrecognized contralateral vocal cord palsy from a prior thyroidectomy.[193] In this case, the vocal cord dysfunction was co-incident with retraction of the carotid sheath—although it can also be induced by a cervical plexus block—but it raises the importance of the preoperative assessment of patients who have had prior head and neck surgery.

## 4. Cervical Surgery

Cervical spine procedures may be followed by airway-related complications, including vocal cord paralysis and airway obstruction. Vocal cord dysfunction was seen in 5% of 411 patients undergoing anterior cervical discectomy and fusion.[194] Stridor was observed in one patient with bilateral vocal cord paralysis who required a tracheostomy. Fifteen of 17 patients had recovered by 12 months. One additional patient had recovered by 15 months, and the remaining patient was lost to follow-up.

Emery and colleagues studied the records of 133 patients who underwent cervical corpectomies with arthrodesis between 1974 and 1989.[26] The patients had undergone an anterior approach to achieve a three-level vertebral body and disc resection with bone grafting. This surgical approach requires tracheal and esophageal retraction toward the opposite side to permit exposure. Drains were placed, and all patients were immobilized by a halo vest or a rigid head-cervical-thoracic orthosis. They identified seven patients (5.3%) who required postoperative reintubation, and although they did not compare these patients with those not requiring reintubation, they attempted to identify common features that increased the risk of postoperative airway compromise. Three patients were immediately reintubated in the operating room, and four were reintubated 12 to 91 hours postoperatively. Severe hypopharyngeal swelling was observed at reintubation in four of seven patients and possibly in a fifth. Five of the seven reintubations had no serious sequelae; these patients were extubated within 2 to 8 days. One patient required a cricothyroidotomy, but delay resulted in hypoxic encephalopathy and death. Another patient was reintubated but developed and succumbed to severe adult respiratory distress syndrome. The investigators think that preexisting pulmonary disease, moderate or severe preoperative myelopathy, extensive multilevel decompression with prolonged surgery, and tissue retraction were risk factors for postoperative airway obstruction, but there were no controls.[26] They recommended 1 to 3 days of elective intubation postoperatively, a cuff-leak test, and direct laryngoscopy at extubation.

Venna and Rowbottom reviewed the records of 180 patients who had undergone a variety of cervical surgical procedures.[28] Based on the Emery study, they had made the decision to keep high-risk patients intubated until they met specified criteria, including a demonstrable cuff leak and the absence of significant airway edema on laryngoscopy. The average time to extubation was 33.5 hours. Despite the delay and the aforementioned criteria, 12 patients (6.6%) demonstrated post-extubation stridor

and breathing difficulties, and 5 (2.7%) required reintubation. Two patients required tracheostomy, and two deaths were attributed to airway obstruction and unsuccessful reintubation.

Sagi and coworkers conducted a retrospective chart review of 311 anterior cervical procedures in an effort to identify the factors associated with airway complications.[195] In this series, 19 (6.1%) of patients had airway complications, but only 6 (1.9%) required reintubation. Most of these complications were attributed to pharyngeal edema. Risk factors included increased intraoperative bleeding, prolonged surgery (>5 hours), and exposure of more than three vertebral bodies, particularly when they included C2, C3, or C4. Reviewing the literature, these investigators identified an airway complication rate of 2.4% (from 1615 cases), 35 of whom required reintubation or tracheostomy. On average, those requiring reintubation did so at 24 hours.

Epstein and coworkers developed a collaborative protocol involving the neurosurgeon and anesthesiologist. Their objective was avoidance of reintubations.[196] Although their study enrolled only 58 patients, they required high-risk, lengthy procedures involving several cervical levels and significant blood loss. All patients remained electively intubated overnight and underwent fiberoptic airway examination before considering extubation. Most patients were extubated the day after surgery, but three remained intubated until day 7. Only one patient required reintubation. This reintubation rate was essentially the same as that observed by Emery and colleagues,[26] but Epstein's cohort appeared to undergo higher-risk surgery.

In an effort to better understand the mechanism of postoperative airway obstruction, Andrew and Sidhu compared the soft tissue changes on the preoperative and postoperative cervical spine radiographs of 32 consecutive patients after a one- or two-level anterior cervical discectomy and fusion.[197] They found that the swelling was maximal at the C3-C4 level, corresponding to the area where Emery observed pharyngeal edema. None of their patients experienced dyspnea despite mean differences of 9.4 mm (95% CI, 7.41 to 11.09 mm) to 10.7 mm (95% CI, 7. 8.82 to 12.58 mm) between the preoperative and postoperative radiographs at C3 and C4, respectively. It appears that this area is most vulnerable, regardless of the level operated on. Compared with Sagi's study, these patients underwent much shorter and more limited procedures.

Extubation criteria after anterior cervical spine surgery were discussed at length in the Society for Airway Management online Forum in 2011. The groups who perform this surgery frequently tend to extubate most of their patients at the conclusion of surgery, except when the blood loss is high (e.g., >500 mL), the surgery is prolonged (e.g., >5 hours), the operation involves more than three levels, the airway was difficult or expected to be difficult, or comorbidities exist, such as severe cervical myelopathy, obesity, and obstructive sleep apnea (OSA). Patients with multiple risk factors are cared for in an ICU and are extubated over an airway exchange catheter (AEC) (personal communication from C.A. Hagberg, SAM Forum, February 2011).

Patients undergoing posterior cervical surgery may face the risk of macroglossia and significant retropharyngeal and hypopharyngeal swelling, which may be aggravated by fixation of the cervical spine and make intubation more difficult.[198] There is a low probability (1.1% to 1.7%) that reintubation will be required,[196,198] but accomplishing this may be very difficult.

## 5. Maxillofacial Surgery and Trauma

Maxillary and mandibular surgery produces conspicuous and often worrisome swelling. Anxiety regarding postoperative care may be heightened by limited airway access, fear that airway intervention may disrupt the surgical repair, and anecdotal reports of near misses or actual fatalities. Because many of these patients are young and otherwise healthy and are undergoing elective surgery for functional or cosmetic improvement, there may be concerns about litigation. It is speculative about whether this results in more or less aggressive care.

Although these concerns demand special attention, deaths rarely occur. In a review of 461 perioperative deaths reported to the Ontario, Canada, coroner between 1986 and 1995, the investigators found only one death associated with orthognathic surgery, although they were unable to determine how many such cases had been performed (see "In Vivo Studies").[199] They were unable to identify nonlethal complications. Meisami and others performed magnetic resonance imaging (MRI) approximately 24 hours after maxillary or mandibular surgery in 40 patients.[200] Despite the significant facial swelling seen in almost all the patients, none exhibited soft tissue swelling from the base of the tongue to the glottis.

Complete airway obstruction after elective orthognathic surgery has been reported. Dark and colleagues described a case involving a young woman who underwent seemingly uneventful mandibular and maxillary osteotomies with submental liposuction.[201] Immediately after extubation, she developed airway obstruction requiring reintubation. Repeated fiberoptic examination and CT showed severe and extensive edema from the tongue to the trachea, which was maximal at the level of the hyoid. By the fourth postoperative day, a cuff leak was detected, and the patient was successfully extubated over a tube exchanger. Hogan and Argalious described a patient in whom maxillomandibular advancement was performed for OSA.[202] The procedure lasted 9 hours, during which he received 7200 mL of crystalloid and 500 mL hetastarch. He remained intubated overnight, and after demonstrating adequate spontaneous ventilation and a cuff leak, he was extubated (over a 19-F AEC). Extubation was immediately followed by clinical evidence of airway obstruction, and he was reintubated. The obstruction was attributed to fractured hardware and a hematoma in the piriform fossa that caused extrinsic compression. This could easily have resulted from periglottic edema. The investigators concluded that patients undergoing this type of surgery face a high risk of airway complications and recommended nasopharyngolaryngoscopy before extubation.

Clinical assessment of airway edema is unreliable,[200] and studies indicate that the cuff-leak test is neither sufficiently sensitive nor specific to determine when to extubate these patients. Endoscopic assessment may help to identify patients who harbor occult clots (i.e., "coroner's clot") behind the soft palate or adjacent to the glottis, but it may miss or itself give rise to troublesome bleeding.

Maxillofacial injuries often result from unrestrained occupants of motor vehicles encountering an unyielding dashboard, windshield, or steering wheel. Gunshot wounds or physical altercations also cause maxillofacial injury. Airway obstruction is a primary cause of morbidity and mortality in these patients, and many die before they reach the hospital.[203] Those with less life-threatening injuries are likely to present with a full stomach, and many have associated head and neck injuries, lacerations, loose or avulsed teeth, intraoral fractures, and fractures extending into the paranasal sinuses, into the orbit, or through the cribriform plate. They may also have an unstable cervical spine or damage to the neural axis. Injuries to the lower face raise the possibility of a laryngeal fracture. Intermaxillary fixation may be part of the surgical plan, necessitating a nasal intubation or a surgical airway. Timing of tracheal extubation is complex and must take into consideration factors such as the patient's level of consciousness, ability to maintain satisfactory gas exchange, coagulation status, and integrity of protective airway reflexes. Attention must be paid to the difficulties originally encountered in securing the airway and an evaluation of whether reintubation would be easier or more difficult after surgery and resuscitation. Most of the trauma literature about airway management addresses intubation and offers little help with extubation, making cooperation between the anesthesiologist,[204,205] surgeon, and critical care physician essential. Intermaxillary fixation requires wire cutters to be immediately available and personnel to know which wires to cut. A flexible bronchoscope, provisions for an emergency surgical airway, and the required expertise should be immediately available at the time of extubation. Alternatives include prophylactic tracheotomy, submental intubation,[206-208] nasal intubation, and bronchoscopic airway evaluation performed before extubation,[209] although assessment may be limited to supraglottic structures and exclusion of tube entrapment. Ideally, extubation should be accomplished in a reversible manner, permitting supplemental oxygenation, ventilation, and reintubation if needed (see "Extubation Strategies").

## 6. Deep Neck Infections

Infections involving the submandibular, sublingual, submental, prevertebral, parapharyngeal, and retropharyngeal spaces are significant airway management challenges, whether intubation is achieved for surgical drainage or for protection during medical management. In expert hands, bronchoscopy-assisted intubation can often be achieved.[210] When this is unsuccessful or constitutes a significant risk of rupturing the abscess, a surgical airway before incision and drainage may be called for.[211] Potter and colleagues retrospectively compared the outcomes of 34 patients in whom a tracheotomy was performed with 51 patients who remained intubated after surgical drainage.[212] All patients had undergone surgical drainage for impending airway compromise and required airway

support postoperatively. It was not always evident to the investigators why a particular strategy was chosen, and these groups were not likely identical. Airway loss occurred more commonly in the intubated patients, but this characteristic was not statistically significant. Two deaths occurred, one resulting from an unintended extubation and the other from post-extubation laryngeal edema and an inability to reestablish the airway. The latter patient had a cuff leak before extubation, and signs of obstruction developed 30 minutes after the ETT was removed. Surgical drainage rarely results in immediate airway improvement, and reintubation or emergent placement of a surgical airway, if required, may be complicated by edema, tissue distortion, and urgency.

## 7. Posterior Fossa Surgery

Posterior fossa surgery can cause injury to cranial nerves, bilateral vocal cord paralysis, brainstem or respiratory control center injury, and macroglossia.[114,115,167,213-216] Because the nerve roots may be very close to the operative site, the resultant injuries may be bilateral, extensive, and transient or permanent. Gorski and coworkers suggested that tolerance of the ETT and the absence of a gag reflex on oral suctioning should arouse suspicion of such an injury.[214] Howard and colleagues described a patient with a recurrent choroid plexus papilloma involving the fourth ventricle.[215] Preoperatively, the patient displayed bulbar dysfunction. His extubation on the first postoperative day was complicated by complete airway obstruction, hypoxia, and a seizure. Laryngoscopy performed after neuromuscular blockade revealed mildly edematous vocal cords. After reintubation and elective tracheostomy, fiberoptic examination showed the vocal folds in a neutral position. Nocturnal ventilation and tracheostomy were still required at 1 and 3 months, respectively. This patient demonstrated central apnea and bulbar dysfunction with hypoglossal paralysis and unopposed vocal fold adduction.

Artru and colleagues described a patient with a cerebellar mass, severe papilledema, and bulbar signs.[213] Despite recovery of consciousness and strength, the patient remained apneic and required ventilatory support for 7 days. The investigators cautioned that the dorsal pons and medulla are the sites of the cardiovascular and respiratory centers that control hemodynamics and ventilation. The area is also host to several cranial nerve nuclei. Damage to these areas can result from edema, disruption, ischemia, or compression and may cause a loss of respiratory drive or airway obstruction.

Dohi and coworkers described a patient who developed bulbar signs, including bilateral vocal cord paralysis after excision of a recurrent cerebellopontine angle tumor.[167] Negative-pressure pulmonary edema developed as a consequence of a bilateral, presumably central RLN injury, and a tracheostomy was required until recovery 3 months later. During the initial intubation, the glottis could not be seen by direct laryngoscopy, and blind intubation was performed. The details of three subsequent unsuccessful extubations and reintubations were not described. Trials of extubation in a patient known to be a difficult (direct) laryngoscopy case are life-threatening and cannot be justified. A tracheostomy

was performed, and vocal cord function recovered after 3 months.

Early vocal cord evaluation after extubation has been advocated along with the involvement of a neurosurgeon, otolaryngologist, speech therapist, and intensivist to manage patients who have developed laryngeal dysfunction.[216] A tracheostomy and an enteral feeding tube may be needed. A more preemptive approach (described later in more detail) involves flexible laryngoscopic assessment through a supraglottic airway (SGA) after removal of the ETT.

## 8. Stereotactic Surgery and Cervical Immobilization

Stereotactic neurosurgical and neuroradiologic procedures are finding increasing applications. When head frames are used, they may impede access for SGA placement or laryngoscopy. Similarly, patients in cervical immobilization devices for spinal cord protection may undergo high-risk surgical procedures.[116] Careful planning for their extubation is critical because reintubation may be difficult, and rapid surgical access may be virtually impossible. Full recovery of strength and consciousness, persistence of respiratory drive, the presence of a cuff leak, preservation of protective reflexes, and absence of significant tongue swelling are the essential prerequisites for extubation. Postoperative seizures, vomiting, elevated intracranial pressure, and neurologic obtundation may make extubation particularly hazardous. Several of the strategies described subsequently should be given serious consideration in managing these patients.

## 9. Tracheal Resections

Patients with moderate or severe tracheal stenosis may come for surgical tracheal resection. These patients usually have tracheal stenosis or tracheomalacia, often caused by prolonged intubation or occasionally caused by a retrosternal mass. Some patients may have compromised preoperative respiratory function. After an end-to-end anastomosis, the surgeon may elect to place a "guardian suture" from the chin to the chest, maintaining the head and neck in flexion and thereby minimizing traction on the suture lines (Fig. 50-1).[217,218] The preference is for early extubation to avoid positive pressure, coughing, and presence of a foreign body in the airway.[217-221] A cough-free extubation is highly desirable, as is avoidance of a need for reintubation, which if required could prove very challenging.

## 10. Palatoplasty

A variety of surgical procedures have been employed to treat OSA, including uvulopalatopharyngoplasty, midline glossectomy, mandibular advancement, limited mandibular osteotomies with genioglossal advancement, and hyoid bone suspension.[222] Pepin and colleagues published a critical analysis of the literature on the risks and benefits of surgical treatment of snoring and OSA.[223] They identified "at least five deaths" after uvulopalatopharyngoplasty and found that few studies had adequate numbers to allow conclusions to be drawn regarding their outcomes. Less than one half of the studies commented on the frequency of complications. A retrospective review of 101 uvulopalatopharyngoplasties identified an early postoperative respiratory complication rate of 10%.[224] Ten of
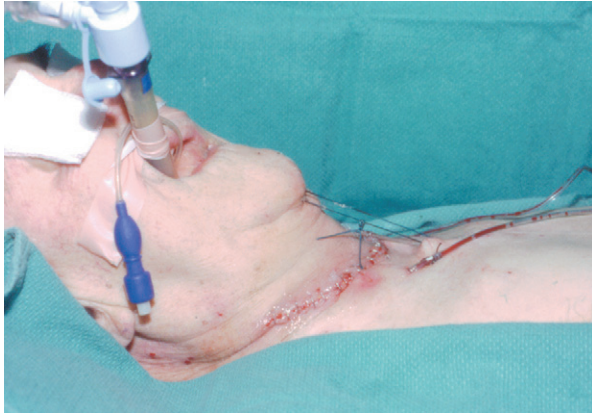
**Figure 50-1** This patient has undergone a cricotracheal resection. Cervical extension is restricted by a chin-to-chest guardian suture. The patient has been extubated, and a laryngeal mask airway has been introduced before reversal of neuromuscular blockade or awakening. This reduces coughing on emergence, allowing gradual recovery and assessment of spontaneous respiratory function while minimizing the potential distraction of the surgical anastomosis. It also provides the optimal means of performing flexible laryngobronchial examination under controlled conditions. (Courtesy of Patrick Gullane, MD, University Health Network, Toronto, Ont.)

11 patients required reintubation, and 1 death resulted from airway obstruction.

Uvulopalatopharyngoplasty was introduced to deal with retropalatine collapse. However, in approximately one half of the adult patients with OSA, obstruction occurs at the retrolingual pharynx. Tongue suspension is one of several approaches introduced to manage the latter group of patients.[225] The procedure involves placement of an anchoring screw in the genial tubercle and attachment of a suture through the base of the tongue. Szokol described a morbidly obese patient with OSA in whom this procedure was performed.[222] Laryngoscopy and bag-mask ventilation had been difficult. At the conclusion of the procedure, the patient was fully awake, was able to sustain a head lift for 5 seconds, demonstrated a negative inspiratory pressure of 40 cm $H_2O$, and was extubated. Stridor was observed immediately, and bag-mask and laryngeal mask ventilation were ineffective. Attempts to reintubate the patient were unsuccessful, necessitating a cricothyroidotomy. Subsequent direct laryngoscopy showed a markedly swollen epiglottis and grossly edematous laryngeal and hypopharyngeal tissues. The patient developed negative-pressure pulmonary edema, and a tracheostomy was performed 2 days later because of persistent swelling. Tracheal decannulation occurred uneventfully 2 weeks later. The physicians speculated that airway manipulation during the surgery was the cause of this patient's swelling. They did not consider that the swelling might have resulted from or at least been aggravated by repeated attempts at laryngoscopy.

Palatoplasty, alone or in combination with other procedures, may be performed on patients with cleft palates or other congenital abnormalities. In one study, 14 (5.7%) of 247 patients undergoing palatoplasty had postoperative airway problems, 12 of whom required reintubation. One half of the patients experiencing complications had Pierre Robin sequence; three of the patients required reintubation 24 to 48 hours postoperatively.[226]

## B. Preexisting Medical Conditions

### 1. Paradoxical Vocal Cord Motion

Paradoxical vocal cord motion (PVCM) is the quintessential example of a situation wherein reintubation will be required. Intubation is not more difficult; extubation is the challenge. This uncommon and poorly understood condition is frequently mistaken for refractory asthma or recurrent laryngospasm.[227-229] The diagnosis is both overlooked and overused, leading to confusion.[230] It also is called vocal cord dysfunction, Munchausen stridor, psychogenic stridor, factitious asthma, pseudoasthma, and irritable larynx syndrome.[230,231] Normal vocal cord motion involves inspiratory abduction and 10% to 40% adduction on expiration. With PVCM, adduction of the true vocal cords occurs on inspiration or expiration, or both. The false vocal cords and the posterior laryngeal wall may further contribute to the airway obstruction.[231-234] This condition may be associated with psychosocial disorders, stress, exercise, gastroesophageal reflux, irritant exposure, or airway manipulation. Pulmonary function tests show normal expiratory but flattened inspiratory flow loops. It is important to differentiate this condition from asthma, laryngospasm, anaphylaxis, angioedema, gastroesophageal reflux, and vocal cord paralysis. The incidence of PVCM is unknown.

Harbison and coworkers described two patients who had post-extubation stridor after thyroidectomies.[235] This is a particularly challenging situation with a complex differential diagnosis, especially because one of the patients had unilateral vocal cord paralysis preoperatively. In that patient, post-extubation stridor developed 24 hours postoperatively and could be observed while awake and asleep. Fiberoptic examination under sedation showed paradoxical motion of the mobile cord. She was managed successfully with speech therapy. They speculated that these cases might have resulted from surgical manipulation of the RLN during the thyroidectomies.

Hammer and colleagues described a 32-year-old woman with recurrent episodes of stridor,[236] sometimes associated with cyanosis, despite normal flow-volume loops and pulmonary function tests. The diagnosis of PVCM was made endoscopically and managed with relaxation techniques. After preoperative sedation, topical lidocaine, and bilateral SLN blocks, she underwent an awake fiberoptic intubation. At the conclusion of surgery, extubation was performed after she was fully awake, but sustained inspiratory stridor ensued, resulting in reintubation. A subsequent attempt the next day confirmed inspiratory vocal fold adduction, and a tracheostomy was required for 58 days. In the absence of features predicting a challenging intubation, there seems little justification for awake intubation, and it may contribute unnecessarily to an anxiety disorder.

PVCM imposes no special requirements for intubation. The abnormality is functional rather than anatomic. Appropriate management depends on having the correct diagnosis, which requires clinical suspicion and endoscopic confirmation of inspiratory adduction of the vocal cords. Adequate oxygenation, consideration of CPAP or helium-oxygen administration, positioning, reassurance, and support may suffice, although sedation may be

required after the diagnosis is confirmed. Speech therapy, psychotherapy, hypnosis, and reassurance may be helpful in the long-term management,[237] but such is not always the case.[227] Some reports have recommended electromyographically guided botulinum toxin injection into the thyroarytenoid muscle for recalcitrant cases. The optimal anesthetic management of these patients is unknown. Regional anesthesia avoids airway intervention, but it does not ensure that a condition that may be stress related will not occur. Familiarity with this condition, calm reassurance when there is prior suspicion, and perhaps deep extubation seem prudent.

## 2. Parkinson's Disease

Susceptibility to aspiration is common among patients with Parkinson's disease and is the most common cause of death. Dysphonia, most frequently hypophonia, occurs in approximately 70% to 90% of patients with Parkinson's disease.[238,239] Video stroboscopic findings include laryngeal tremor, vocal fold bowing, and abnormal glottic opening and closing.[239] Several neurodegenerative diseases, including multiple system atrophy, have some features in common with Parkinson's disease, including dysphonia, and these patients may exhibit bilateral abductor vocal fold paresis. Typically, symptoms in patients with Parkinson's disease progress insidiously, are not recognized by the patient, and may be associated with nocturnal stridor. These features resemble those of OSA identified by polysomnography. Many of these patients may benefit from nocturnal CPAP or bi-level positive airway pressure (BiPAP).[238]

Blumin and Berke described seven patients, only one of whom presented for surgery. This patient underwent a transurethral prostate resection under general anesthesia, and 2 weeks after surgery, he returned with biphasic stridor that necessitated an emergent tracheostomy. It is unclear whether there was a relationship between the surgery or anesthesia and subsequent airway obstruction.

Patients with multiple system atrophy have daytime hypoxemia associated with abnormal laryngopharyngeal movements, including obstruction at the arytenoids, epiglottis, base of the tongue, and soft palate. The significance of these problems is unclear, but they may contribute to complications after extubation.[240]

Vincken and colleagues studied 27 patients with extrapyramidal disorders.[241] Twenty-four had flow-volume loops, many of which demonstrated saw-toothed oscillations, even in the absence of respiratory symptoms. They observed oscillations with rhythmic (4 to 8 Hz) or irregular movements of the glottis and supraglottic structures. Ten patients exhibited intermittent upper airway obstruction. Four patients had stridor or dyspnea. The investigators believed that the upper airway was the primary site of involvement. In a subsequent report, they observed symptomatic improvement with levodopa despite persistence of the oscillatory pattern on flow-volume loops.[242] Inspiratory and expiratory flows after levodopa increased from 1.40 to 3.50 L/sec and 0.95 to 5.05 L/sec, respectively. Bronchodilators provided no additional benefit. This case may have important implications for the perioperative management of patients with Parkinson's disease.

Easdown and colleagues described a patient with Parkinson's disease who had a respiratory arrest 60 hours after surgery.[243] Before that event, the patient had episodic desaturation, labored breathing, and progressive hypercapnia in the absence of tremor or rigidity. Treatment with bronchodilators produced no benefit, and his condition improved immediately after intubation. With the ETT, compliance and resistance appeared normal. This patient's levodopa or carbidopa had not been resumed postoperatively, and the investigators speculated that this caused or contributed to upper airway obstruction. Because most patients with Parkinson's disease are elderly and may have comorbidities that can make the diagnosis uncertain, it is important to consider involvement of the upper airway and the dramatic effect withdrawal and reinstatement of medications can have on their clinical course. This concern is reinforced by a case report describing a patient who developed airway obstruction and acute respiratory acidosis requiring intubation preoperatively because five doses of his antiparkinsonian medications were withheld while he was being fasted.[244] Easdown and colleagues emphasized the importance of continuing these medications, and avoidance of dopamine antagonists throughout the perioperative period.[243]

Backus and colleagues described a patient with long-standing Parkinson's disease who became aphonic, developed stridor, and suffered respiratory arrest shortly after taking cough medication.[245] Complete upper airway obstruction recurred with vocal fold apposition immediately after extubation. Four days later, the patient extubated herself with no further complications. The investigators interpreted this spontaneous laryngospasm as a manifestation of Parkinson's disease. Others have observed upper airway dysfunction, airflow limitation, and bilateral abductor vocal cord paralysis in association with Parkinson's disease. The first episode might not have been spontaneous but instead a consequence of aspiration of the cough medicine. Nonetheless, there remains a possibility that these patients are more prone to laryngospasm, whether spontaneous or induced by glottic stimulation.

Liu and coworkers described airway obstruction during induction of anesthesia.[246] Despite being unable to visualize the larynx, they attributed the obstruction to laryngospasm. The obstruction resolved with awake, blind nasal intubation but recurred 24 hours later on extubation. At that point, fiberoptic examination showed inspiratory vocal fold adduction, necessitating reintubation. It is unclear whether they were observing manifestations of Parkinson's disease or PVCM, but extubation was uneventful 24 hours later after increasing the dosage of levodopa or carbidopa.

Parkinson's disease is a common disorder, but only 13 cases of stridor have been attributed to it.[239] The pathogenesis of upper airway obstruction is unknown. It may be mediated by the basal ganglia and nucleus ambiguus. A similar phenomenon involving esophageal spasm has been associated with Parkinson's disease. One theory invokes laryngeal hypertonicity, which may be triggered by copious secretions.

### 3. Rheumatoid Arthritis

In patients with rheumatoid arthritis, the airway manager needs to be concerned about three joint areas: the cervical spine, the temporomandibular joint (TMJ), and the cricoarytenoid joint.[247] Autopsy studies suggest that 30% to 50% of patients with rheumatoid arthritis have significant cervical spine involvement. Cervical subluxation has been identified clinically in 43% to 86% of these patients and may represent a serious neurologic risk during intubation with flexion or extension.[145,247-249] The spectrum of cervical involvement ranges from ligamentous destruction with subluxation and impaction to extreme limitations in the range of motion because of fibrosis and ankylosis. These patients may have a narrowed glottic aperture, limited mouth opening due to involvement of the TMJs, micrognathism, laryngeal deviation, and cricoarytenoid and cricothyroid involvement.[250,251] Kohjitani and colleagues retrospectively described four patients undergoing bilateral TMJ replacement; three had glottic erythema and swelling on endoscopy, three had OSA, and three experienced laryngospasm at intubation and after extubation.[251] TMJ involvement may result in loss of ramal height and micrognathia with or without ankylosis and associated OSA.

Cricoarytenoid arthritis and its consequences have long been recognized in the anesthesia and general medical literature.[252-256] Although rheumatoid arthritis is the most common cause of this condition, it also may be associated with bacterial infections, mumps, diphtheria, syphilis, tuberculosis, Reiter's syndrome, ankylosing spondylitis, systemic lupus erythematosus, gout, progressive systemic sclerosis, and other conditions.[257] The cricoarytenoid joint has a synovial lining and bursa. Its mobility is vital for speech, respiration, and protection from aspiration. Inflammatory changes may include effusion, pannus formation, joint erosion, and ankylosis, any of which may compromise the joint's functions. Its involvement may be unsuspected or mistaken for asthma until intubation or after extubation and may necessitate a surgical airway.[258,259] Dysphonia, dyspnea, or stridor should raise suspicion of this possibility. Complete airway obstruction is a well-described but uncommon complication, despite involvement of the cricoarytenoids in 26% to 86% of patients with rheumatoid arthritis.[251,259] Laryngoscopy may reveal a rough and thick mucosa with narrowing of the vocal chink. Although airway obstruction occurs most commonly in patients with long-standing rheumatoid arthritis with polyarticular and systemic involvement, laryngeal stridor has been described as the sole manifestation of this disease.[260]

Keenan and coworkers described tracheal scoliosis, which consisted of tracheal deviation, laryngeal rotation, anterior angulation, and vocal fold adduction seen fiberoptically and on CT scans.[255] It was presumed to result from the loss of vertical height and asymmetrical bony erosions.

Wattenmaker and colleagues studied patients with rheumatoid arthritis undergoing posterior cervical spine procedures.[250] Their primary objective was to compare the perioperative airway complications seen in rheumatoid arthritis patients when intubation was performed by direct laryngoscopic or flexible bronchoscopy. Retrospectively reviewing 128 consecutive posterior cervical procedures, upper airway obstruction characterized by stridor occurred in 9 of 128 patients, 1 of 70 patients intubated with bronchoscopic guidance, and 8 of 58 patients intubated otherwise (i.e., direct laryngoscopy or blind nasotracheal technique). Five patients (all in the nonbronchoscopic group) required emergency reintubation that proved to be very difficult, with two near fatalities and one death. Although the two groups were similar with regard to age, gender, American Rheumatology Association classification, ASA physical status, duration of surgery and anesthesia, fluid balance, and postoperative immobilization, there were significant differences in time to extubation. Seven of the patients could not be intubated by flexible bronchoscopy and were therefore intubated by a nonfiberoptic technique. The patients were not randomized to different methods; criteria for the method of intubation and techniques were not described; all patients were intubated awake; and the study was carried out over an 11-year period.[261] Although it is not possible to draw firm conclusions from this study, there was a high incidence (7%) of post-extubation stridor and difficult or failed reintubations, regardless of the intubation technique.

Patients with rheumatoid arthritis qualify as higher-risk extubation cases because they may have a fixed or unstable cervical spine, TMJ ankylosis, difficult intubations by direct or flexible laryngoscopy, and increased risk of post-extubation airway obstruction. Several investigators have recommended postponing extubation until the patient is wide awake. Unfortunately, this provides increased protection against nothing other than laryngospasm and aspiration. The prevailing wisdom is that patients with limited mouth opening and a potentially unstable cervical spine should be intubated with a flexible bronchoscope.[250] This method involves blind passage of the ETT through the cords, which may be traumatic,[91,261] particularly in the face of preexisting cricoarytenoid arthritis. Regional anesthesia should be considered as an alternative to general anesthesia when appropriate. When intubation cannot be avoided, proposed extubation strategies include a preemptive tracheostomy or a method that increases the reversibility of extubation. Neither strategy has been prospectively evaluated in this population.

### 4. Tracheomalacia

Tracheomalacia is a dynamic airway obstruction resulting from loss of the cartilaginous tracheal support. This results in the posterior membranous wall bulging anteriorly when the intratracheal pressure is reduced or the intrathoracic pressure is increased.[141] Although rare, it should be considered when the patient has dyspnea on exertion with difficulty clearing secretions and a seal-like, incessant cough.[141,262]

Patients frequently are misdiagnosed with asthma and fail to respond to escalating therapy. Pulmonary function tests (i.e., forced expiratory volume at 1 second, forced vital capacity, and peak expiratory flow) show severely diminished expiratory flow with relative preservation of the inspiratory flow. The diagnosis may be confirmed

fiberoptically during spontaneous breathing. Tracheomalacia may be congenital[263] or result from vascular compression,[264] an intrathoracic goiter,[265] chronic obstructive pulmonary disease, or prolonged intubation. The latter may be caused by ETT cuff-induced erosion of the tracheal cartilage with or without extension to the membranous trachea.

The severity of the dynamic obstruction is proportional to the expiratory force. It may be unapparent during quiet breathing but disabling in a distressed patient. Positive pressure or bypassing the lesion with a tracheal tube provides temporary relief while further management options are considered. They may include medical management, surgical resection, or placement of a stent.[262] Additional suggestions for the extubation of a patient with suspected tracheomalacia are described later.

Relapsing polychondritis is an example of extensive tracheobronchomalacia. It is a rare, multisystem disease characterized by episodic inflammation of cartilaginous structures resulting in tissue destruction.[262,266] Laryngeal and tracheal tract involvement occurs in approximately one half of patients. It usually occurs early in the course of the disease and may manifest as hoarseness, nonproductive cough, shortness of breath, and stridor. Upper airway obstruction is usually diffuse and may progress to involve the glottis, subglottic area, trachea, and bronchial cartilages. Histologically, there is evidence of perichondral inflammation and replacement of cartilage by fibrous tissue that manifests as inflammatory swelling and progressive destruction of cartilage. The clinical manifestations range from bronchorrhea and recurrent pneumonia to airway collapse. Medical management consists of steroids, nonsteroidal anti-inflammatory drugs, and immunosuppressant agents, but their benefit varies. Surgical management consists of external airway splinting or self-expanding metallic stents. These patients may present for bronchoscopy, tracheostomy, tracheal or nasal reconstruction, aortic valve replacement, or stent placement.[264,267-271] Airway collapse after extubation should be anticipated and may be temporarily dealt with by CPAP.[272,273]

### 5. Obstructive Sleep Apnea Syndrome

In the ASA Closed Claims Project analysis of adverse respiratory events, 65 of the 156 perioperative events involved obese patients; for the claims specifically related to extubation, 12 of the 18 were obese, and 5 of these patients had been diagnosed with OSA.[2] OSA correlates positively with age and obesity, both of which are becoming increasingly prevalent. The pathophysiology and perioperative airway management of OSA in obese patients has been comprehensively reviewed.[274-276] Many surgical patients have undiagnosed or untreated OSA. OSA syndrome is associated with an increased risk of gastroesophageal reflux, difficult mask ventilation[276-278] and laryngoscopic intubation,[279-282] and accelerated arterial oxygen desaturation.[276,283] The risk of airway obstruction after surgery is increased for patients with OSA; life-threatening post-extubation obstruction occurred in 7 (5%) of 135 patients.[279,284] Rapid desaturation, difficult mask ventilation, and difficult direct laryngoscopy make this a particularly high-risk setting.[285] The ASA practice guidelines

for the management of patients with OSA provided limited guidance beyond a strong recommendation that they be fully awake and that the airway manager verify that neuromuscular blockade is completely reversed before extubation. If possible, extubation and recovery should be carried out in the lateral or semi-upright position,[286] nasal CPAP should be available or routinely implemented, and consideration should be given to extubation over a tube exchanger.[274,276,280,284,287] These strategies have been associated with better outcomes, and anecdotal comparisons are compelling, but they have not been subjected to controlled, randomized trials, and they were not addressed by the ASA Task Force.

### 6. Laryngeal Incompetence

Laryngeal function may be disturbed for at least 4 hours after tracheal extubation.[288] Immediately after extubation, 8 (33%) of 24 patients aspirated swallowed radiopaque dye; 5 showed radiologic evidence of massive aspiration. Four hours after extubation, 4 (20%) of 20 patients aspirated dye; 3 had massive aspirations. At 24 hours, the rate was reduced to 5%. In this study, patients had been intubated for 8 to 28 hours during and after cardiac surgery. Although the investigators did not observe a relationship between duration of intubation (8 to 28 hours) and aspiration, it is unclear whether the presumed laryngeal incompetence occurs after brief intubation or is more common and severe with prolonged intubation. The mechanism of laryngeal incompetence was postulated to be primarily sensory because patients who aspirated dye did not cough.

Residual neuromuscular paralysis is a common problem in postoperative patients and may result in hypoventilation, hypoxemia, pharyngeal and laryngeal dysfunction, or increased pulmonary aspiration.[264,289] Pharyngeal function was impaired in conscious volunteers receiving a continuous infusion of vecuronium and resulted in laryngeal penetration of contrast medium proportional to the degree of blockade.[290] Relaxation of the upper esophageal sphincter was also observed. None of the volunteers coughed or had respiratory symptoms. Berg and colleagues found a higher incidence of postoperative pulmonary complications (i.e., pulmonary infiltrate or atelectasis associated with cough, sputum, or shortness of breath) among patients randomly assigned to receive a long-acting or intermediate-acting neuromuscular blocker.[291] It is intriguing to speculate on how residual neuromuscular blockade may contribute to laryngeal incompetence.

### 7. Pulmonary Aspiration of Gastric Contents

Although more patients are being diagnosed with gastroesophageal reflux, the diagnosis of perioperative pulmonary aspiration has not increased.[292,293] Aspiration is estimated to complicate 1 of 2000 to 3000 general anesthetics and was responsible for only 3 of 156 perioperative events in the ASA Closed Claims Project review.[2,293,294] Nonetheless, it is the leading cause of pneumonia in the ICU and a common cause of acute respiratory distress syndrome.[295] Many of these cases are ventilator-associated pneumonia and occur with the ETT in situ. Factors predisposing a surgical patient to aspiration include

emergency surgery, pain, obesity, narcotics, nausea, ileus, bowel obstruction, pregnancy, some surgical positions, depressed level of consciousness, inadequate depth of anesthesia, postoperative drowsiness, and residual neuromuscular blockade. Despite the ubiquity of these conditions, perioperative aspiration is not commonly identified. Before intubation, difficult bag-mask ventilation may result in gastric distention, which may be further complicated if laryngoscopy proves difficult because it may delay securing the airway. Repeated laryngoscopic attempts may cause edema, thereby increasing glottic resistance. Aspiration may also result from obtundation or conditions that impair vocal cord apposition (e.g., vocal cord paralysis, laryngeal incompetence, residual neuromuscular blockade, granulomas). Aspiration can cause serious morbidity and death.[292-295]

Although most incidents of aspiration seem to occur at induction, many occur during maintenance and recovery from anesthesia.[296] Numerous strategies have been described to reduce the risk at induction, but relatively little information is available on how best to prevent this later. Premature extubation, postoperative nausea, delayed gastric emptying, residual neuromuscular blockade, relaxation of the esophageal sphincters, decreased level of consciousness, gagging on an ETT, supine recovery, and impaired laryngeal competence may make emergence from anesthesia and tracheal extubation as problematic as induction. A kinked or clamped nasogastric tube may promote regurgitation and aspiration. Evidence-based recommendations on an extubation strategy to reduce aspiration are not available. It would seem logical to minimize the contributing factors: postoperative nausea and vomiting, residual neuromuscular blockade, decreased level of consciousness and associated diminished protective airway reflexes, and gastric evacuation. We do not know whether gastric decompression reduces aspiration; a well-seated i-gel SGA[297] or ProSeal LMA may or may not offer some protection from aspiration.[298-300] With the current information, it is not appropriate to recommend the elective use of these devices in a patient at increased risk for aspiration.

## V. FACTORS AFFECTING INTUBATION AND EXTUBATION

### A. Previously Encountered Airway Difficulties

Multiple attempts at laryngoscopy by experienced personnel, a need for alternative airway management techniques due to failure of direct laryngoscopy, and prior difficulty prompting the primary use of alternative techniques are settings in which reintubation may be problematic. In urgent or emergent circumstances, methods that had previously been successful may not be available or appropriate. The required equipment, necessary skills, or time required to perform alternative techniques may not be available. Uncertainty regarding the ease of ventilation or intubation may correctly lead to disinclination to administer paralytic and sedating drugs. Although they may facilitate ventilation and intubation, failure will result in an apneic patient who can neither be ventilated

nor intubated. Knowledge of prior difficulties may result in intubation conditions that are less favorable to success. Repeated attempts at laryngoscopy are associated with a significant increase in the risk of hypoxemia, esophageal intubation, regurgitation, aspiration, bradycardia, and cardiac arrest.[2,301] To avoid this risk, flexible bronchoscopic intubation may be considered, but in an agitated, hypoxic patient with secretions or blood in the airway, it may be difficult to achieve adequate topical anesthesia, and the procedure may be difficult or impossible.

### B. Limited Access

Limited access to the airway is exemplified by intermaxillary fixation, severe cervical restriction, instability, or immobilization, and the chin-to-chest guardian suture (see Fig. 50-1) to prevent traction tracheal resection. More commonly, this situation may arise in the confining space of a PACU, ICU, or patient room where access is limited. In each case, there may be additional risks related to oxygenation, ventilation, airway obstruction, or pulmonary toilet. For example, after cervical fixation or orthognathic surgery, the patient may have macroglossia or supraglottic edema. A patient requiring tracheal resection may be unable to clear blood or secretions from the airway.

### C. High-Risk Cases

A higher-risk extubation exists when there is an increased likelihood that reintubation will be necessary or an increased risk that reestablishing the airway will be difficult. The increased need to reintubate may result from failure of oxygenation, ventilation, pulmonary toilet, or loss of airway patency. These risks cannot always be anticipated, but there are often identifiable patients with less reserve. The clinical conditions previously discussed, including OSA; rheumatoid arthritis; cervical, tracheal, thyroid, or carotid surgery; and intermaxillary fixation, are higher-risk settings because reintubation may be challenging.

The clinical playing field may not be level at all hours of the day. The immediate availability of highly trained primary and support personnel, equipment, and the necessary clinical information may be problematic at night or during periods of intense activity. The ASA Task Force on Management of the Difficult Airway and the Canadian Airway Focus Group recommended a preformulated strategy for extubation of the difficult airway.[5,6] The ASA Closed Claims Project supported the need for such a strategy.[2] Patients at risk for hypoventilation, hypoxemia, and loss of airway patency have been discussed. The remainder of this chapter addresses specific extubation strategies.

## VI. EXTUBATION STRATEGIES

If any of the higher-risk extubation conditions exists or is anticipated, the clinician should consider a strategy that does not cut off access to the airway. Ideally, the strategy should permit continued administration of oxygen or ventilation of a failing patient even while the airway is

being reestablished. These objectives are consistent with the ASA Task Force and Canadian Airway Focus Group recommendations.[5,6]

Extubation risk stratification is largely based on intuition, anecdotal reports, and limited clinical series. The proposed classification and strategies are becoming broader and deeper with time. Because most patients—even those at high risk—can be successfully extubated, any proposed strategy must entail less risk than removing the tracheal tube and hoping for the best. It should also involve minimal discomfort, have an acceptable cost, and facilitate oxygenation, ventilation, and reintubation.

## A. Deep versus Awake Extubation

Extubations may be performed before or after recovery of consciousness. Deep extubation ordinarily occurs after full recovery of neuromuscular function and the resumption of spontaneous ventilation. Its purported advantage is avoidance of the adverse reflexes associated with extubation, such as hypertension, dysrhythmias, coughing, laryngospasm, and increased intraocular or intracranial pressures. The fundamental disadvantage of deep extubation is the patient's inability to protect his airway against obstruction and aspiration. When deep extubation is improperly executed, laryngospasm and its attendant complications are more likely to occur. Although not having to wait for the recovery of consciousness may accelerate operating room turnover, this approach is more difficult to justify when anesthetic agents having a faster elimination time are available. Delays in recovery usually are brief. Unscavenged volatile anesthetic agents may also represent an occupational health hazard. A significant proportion of American anesthesiologists practice the technique, at least some of the time, but there are few data for adults that compare the safety of deep extubation with that of awake extubation.[12] Koga and colleagues compared three small groups of adult patients who underwent deep extubation, awake extubation, or deep extubation after the insertion of an LMA.[80] Straining occurred in a high (but comparable) proportion of patients whether the ETT was removed before or after recovery of consciousness.

Current strategies include extubation on low-dose propofol or remifentanil and intracuff or intravenous lidocaine, which may reduce coughing and straining on extubation. Deep extubation followed by LMA insertion (with 2% to 3% isoflurane) is discussed later. Deep extubation is contraindicated when mask ventilation was or is likely to be difficult, the risk of aspiration is increased, endotracheal intubation was difficult, or airway edema is likely.

## B. Extubation with a Laryngeal Mask or Other Supraglottic Airway

On emergence from general anesthesia, most patients tolerate an LMA with less coughing and changes in intraocular, intracranial, and arterial pressures (see Fig. 50-1).[57,77,80,302,303] Silva and Brimacombe substituted an LMA for the ETT in a small series of patients while still asleep and paralyzed after completion of neurosurgical procedures.[304] Muscle relaxation was then reversed, and the anesthetic was discontinued. The LMAs were removed when the patients resumed spontaneous ventilation and obeyed commands. None of the 10 patients coughed, and changes in the rate-pressure product (indicating cardiac oxygen requirements) were minimal. The investigators concluded that the technique might prove useful in patients undergoing other types of surgical procedures. They stressed that this substitution should be performed only by those skilled in LMA insertion. Patients must be at a sufficient depth of anesthesia or coughing, breath-holding, laryngospasm, and the very pressor responses this substitution is intended to avoid may occur. Bailey and others recommended that the LMA be inserted before removal of the ETT to prevent losing the airway after tracheal extubation.[80,305] Compared with deep tracheal extubation followed by Guedel airway insertion, there was a lower incidence of coughing and requirement for airway manipulation.[305] Koga and coworkers compared this technique with deep and awake tracheal extubation.[80] They observed no difference in recovery conditions between patients in whom the ETT was removed by deep or awake methods; however, they noticed a significant improvement in recovery conditions when the LMA substitution was performed. This technique is useful but can jeopardize a secure airway if not properly executed. It should be practiced on routine airways before use in higher-risk extubations.[306] Brimacombe suggested (personal communication, December 2010) that a ProSeal LMA or LMA Supreme with a gum elastic bougie inserted through the drainage tube could produce a more secure substitution for an ETT.[307]

Sometimes, it is desirable to perform the exchange of an ETT for an SGA in reverse. Several types of tube exchanges have been described. Asai wanted to replace a damaged ETT in a patient who had been a difficult intubation.[308] He inserted an LMA Classic behind the existing ETT. A fiberoptic bronchoscope (FOB) with a replacement 7-mm ETT was introduced through the LMA; the FOB was advanced through the vocal cords, and the original ETT was removed. The new ETT was then advanced over the FOB, which was removed. To extend the length of the ETT to enable its removal from the LMA, another ETT was inserted into the proximal end of the replacement ETT.[308] This technique was complicated and could easily have failed.

Matioc and Arndt wished to substitute an ETT for a ProSeal LMA.[309] Using an Arndt Airway Exchange Catheter Set (Cook Critical Care, Bloomington, IN) (see Fig. 50-6), they introduced an FOB through the ProSeal LMA into the trachea. The set comes with a 144-cm extrastiff Amplatz guidewire, which was passed through the FOB, and the latter was removed. An 11-F, 70-cm, Cook airway exchange catheter (CAEC) was introduced over the guidewire and the ProSeal LMA was removed. The replacement ETT was then advanced over the exchange catheter.

A simpler approach involving the Aintree intubation catheter (Cook Critical Care) has been described with a variety of SGAs, including the cuffed oropharyngeal airway (COPA),[310] the LMA Classic,[311-313] the LMA

ProSeal,[314,315] and the LMA Supreme.[316] The Aintree intubation catheter is 56 cm long and has an internal diameter (ID) of 4.7 mm; its outer diameter (OD) is 6.3 mm (19 F). A bronchoscope (<4 mm) can be inserted through the device, leaving 3 or 4 cm protruding beyond its tip. A 7-mm or larger ETT can be advanced over it. Only the distal 3 to 4 cm of the protruding bronchoscope is flexible, but it is usually sufficient to allow successful maneuvering into the trachea, after which the SGA is removed and a replacement ETT is advanced over the Aintree catheter. A Rapi-Fit adapter is provided to enable positive-pressure ventilation while the substitution is performed. The Aintree intubation catheter and FOB are inserted together through the SGA. There are several advantages of this technique.[315,317] It can be used to facilitate conversion from an unmodified LMA to an oral ETT of adequate size. Sufficient length allows the LMA to be removed with minimal risk of losing the airway. The Aintree intubation catheter fits tightly to the insertion cord of the FOB and to the ETT, thereby reducing the size discrepancy that often results in difficult glottic passage. The catheter can be used as a conduit for manual or jet ventilation during an exchange. An LMA Classic, inserted as a rescue device (for "cannot intubate, cannot ventilate" cases) can facilitate safe tube exchange without the need for an intubating LMA.

## C. Extubation or Reintubation over a Fiberoptic Bronchoscope or Laryngoscope

When tube entrapment is a possibility, extubation over an FOB can avert a disastrous outcome. For a spontaneously breathing patient, extubation over an FOB provides the opportunity of visually assessing the trachea and laryngeal anatomy and function. This can help in the patient suspected of having tracheomalacia, vocal cord paresis, or PVCM. It also permits assessment of supraglottic structures.[318] These opportunities can be maximized by reassuring the patient and by providing judicious sedation, an antisialagogue, and the use of a Yankauer sucker for oral secretions. The oropharynx is suctioned, taking care to avoid inducing a gag reflex. The FOB is placed above the carina, and the cuff is slowly deflated to minimize coughing. The ETT is slowly withdrawn into the oropharynx, followed by very gradual withdrawal of the FOB to the supraglottic region. After the patient is comfortable, the FOB is further withdrawn to a position just above the vocal cords. Even with this deliberate technique, the exercise is frequently frustrated by excessive secretions, coughing, swallowing, or poor tolerance with insufficient opportunity of visualizing the structures of interest.

If the technique is successful, it may identify problems and anticipate complications. When significant abnormalities are identified, a decision must be made about whether to immediately reinsert the ETT or withdraw the FOB and manage the patient with agents such as corticosteroids, racemic epinephrine, and helium-oxygen.[149,152] This technique is not practical for performing a trial of extubation, in part because such a trial lasts only seconds or minutes.

Watson endorsed the use of an FOB to exchange ETTs, citing the advantages of minimal sedation, risk of aspiration, hemodynamic embarrassment, and uncertainty about tube placement.[319] His technique involved passing the loaded FOB alongside the existing ETT. He had used this technique successfully in 13 of 15 attempts. Dellinger suggested that use of an FOB to perform tube exchanges offered the greatest likelihood of reintubation success and recommended techniques for conversion from nasal to oral, oral to nasal, and oral to oral ETT exchange in addition to extubation.[318] We have found this method of extubation to be unreliable and one that demands immediate and often incorrect clinical judgment.

## D. Extubation with a Supraglottic Airway with or Without a Bronchoscope

Extubation of a difficult airway over an FOB or SGA has the limitations previously described, but the combination of these devices offers significant advantages. Replacement of an ETT with an SGA provides an excellent means of performing a fiberoptic assessment of glottic and subglottic anatomy and function. After the substitution is performed and the patient is under anesthesia or a suitable degree of sedation, muscle relaxation can be reversed and spontaneous ventilation allowed to resume. An FOB is then passed through the SGA, and vocal fold movement and appearance can be assessed while the concentrations of oxygen and volatile agents (if necessary) can be controlled. The view is protected from oral secretions, and inadequate ventilation can be supplemented. PVCM or tracheomalacia can be visually and functionally evaluated, although both may be minimal if the patient is deeply anesthetized.

This technique is useful in patients with recurrent post-extubation stridor or those at risk for static or dynamic tracheal stenosis. We frequently employ this technique in patients undergoing thyroidectomy when tracheomalacia or vocal fold paralysis is suspected.[181]

## E. Use of a Gum Elastic Bougie or Mizus Endotracheal Tube Replacement Obturator

Finucane and Kupshik described an awake, blind nasal intubation in a patient with cervical instability, but the cuff became damaged, requiring a tube exchange.[320] They used the 63-cm-long, 4-mm-OD plastic sleeve from a brachial central venous catheter as a tube exchanger. Others have used a gum elastic bougie to achieve similar objectives.[321-324]

Cook Critical Care designed the Mizus Endotracheal Tube Replacement Obturator (METTRO) for the replacement of endotracheal and tracheostomy tubes (Fig. 50-2). It is available in two sizes: 70 cm long (7.0 F, 2.3 mm) for replacement of ETTs as small as 3 mm and 80 cm long (19 F, 6.3 mm) for passing through tubes 7 mm or larger. The METTRO is a single-use, flexible, radiopaque, solid device with a tapered tip and distance markings. The package insert instructs the user to advance this device until resistance is encountered, but this recommendation can result in coughing, discomfort,

**Figure 50-2** The Mizus Endotracheal Tube Replacement Obturator (METTRO) is a solid device that is tapered at the end. It is available in two diameters (7 and 19 mm) and two lengths (70 and 80 cm). The figure shows the proximal *(bottom)* and distal *(top)* ends of one of these. (Courtesy of Cook Critical Care, Bloomington, IN.)

hypertension, and tachycardia. Tracheal perforation has been reported using different devices but following similar recommendations.[325,326]

The smaller airway obturator has been used to maintain airway access during tracheostomies in 22 patients and for "tentative extubations" in 7 patients.[327] The smaller-caliber device was preferred by Audenaert and colleagues because patient discomfort was minimal during tube exchanges and the device was unobtrusive during surgical tracheostomies. The obturator was removed when it was apparent that the patient was unlikely to require reintubation. The 19-F obturator was not conducive to spontaneous breathing. Chipley and coworkers used a METTRO in an obese patient with a fractured occipital condyle recovering from respiratory failure.[328] They left this in place for 48 hours, removing it when extubation appeared to be successful. They also described the use of the obturator to stimulate coughing, although this might have been ill advised given the possibility of tracheal perforation. The METTRO obturator is being phased out and will soon be unavailable.

In a modification of the Eschmann gum elastic bougie (Intubation Guide, Smiths Medical, Keene, NH), both ends of the bougie were amputated, exposing a hollow core. A cannula, syringe, and ETT connector enabled this assembly to be connected to an oxygen source.[329]

## F. Use of Jet Stylets

The ubiquitous nasogastric tube has been used as an exchange catheter,[330] but these devices are specifically formulated to become softer as they are warmed. This thermolability is not a desirable characteristic for a tube exchanger.

Bedger and Chang coined the term *jet stylet* to refer to a self-fashioned, 65-cm-long, plastic catheter with a removable 15-mm adapter at the proximal end. It could be connected to an anesthesia machine or jet injector.[331] They created three side ports cut into the distal 5 cm to minimize catheter whip during jet ventilation. They used the stylet for extubation or reintubation of 59 patients.



**Figure 50-3** An Endotracheal Ventilation Catheter (ETVC) properly secured tube exchanger.

It also functioned adequately in patients when it was used for jet ventilation and oxygen insufflation. Although no complications were described in this series, in an earlier report, Bedger and Chang described tension pneumothoraces in 3 of 600 patients ventilated through a 3.5-mm-OD pediatric chest tube at 15 psi.[332] This stylet had been used to provide airway access and ventilation during direct laryngoscopy. They speculated that the pneumothoraces might have resulted from endobronchial migration of the catheter. They did not consider the possibility that barotrauma occurred as a result of jet ventilation against apposed vocal cords as the patients were recovering from neuromuscular blockade.

## G. Use of Commercial Tube Exchangers

Several commercial products incorporate many of the features described by Bedger and Chang.[331] They are long, hollow catheters that may include connectors for jet or manual ventilation. Most have distance and radiopaque markers. They also have end and distal side holes. They can be introduced through an existing ETT, permitting its withdrawal. Oxygen insufflation or jet ventilation can be provided through the tube exchanger. Respiratory monitoring can be achieved by connection to a capnograph. Spontaneous breathing may take place around the device. In most reports, these catheters have been tolerated well enough that they can be left in situ until it is unlikely that reintubation will be required. They must be properly secured to ensure that they do not come out prematurely (Fig. 50-3). Even with the catheter in place, most patients will be able to talk or cough.

If reintubation or a tube exchange is required, it can be facilitated with gentle laryngoscopy, not necessarily to reveal the glottis but to retract the tongue. Reintubation using a tube exchanger is similar to intubation over an FOB, and the difference of diameters between the tube exchanger and the advancing ETT may predict the relative ease of tube advancement. It is wise to delegate someone to secure the exchange catheter during reintubation. If resistance is encountered, ETT rotation may successfully release the tube from the piriform fossa, "vocal cord," or arytenoid cartilage, but it is best to

minimize the size discrepancy by choosing a larger-diameter tube exchanger or a smaller-diameter ETT.

These devices are consistent with practice guidelines from the ASA Task Force and Canadian Airway Focus Group recommendations regarding the extubation of the difficult airway.[5,6] They increase the probability that reintubation will succeed; if difficulty is encountered, the device can provide a conduit for oxygen insufflation. Jet ventilation, if necessary, can be accomplished while alternative techniques are explored. This may be thought of as a reversible or staged extubation. With the device in place, other options can be pursued, including evaluation of the benefits of helium-oxygen mixtures or inhalation of racemic epinephrine. Knowing that the patient is satisfactorily oxygenated and ventilated, the airway manager can recruit additional information, equipment, and expertise.

The differences between these commercial products are far less important than the concept of a reversible extubation. In our opinion, reintubation of the high-risk patient may be unlikely, but it must have a high probability of success.

## 1. Tracheal Tube Exchangers

The most basic commercial tube exchangers are the Sheridan T.T.X. (Hudson Respiratory Care Inc. [RCI], Temecula, CA) (Fig. 50-4) and the JEM Endotracheal Tube Changer (Instrumentation Industries Inc., Bethel Park, PA). The T.T.X. devices are available in four ODs (2.0-, 3.3-, 4.8-, and 5.8 mm) and two lengths (56 and 81 cm). The smallest can be inserted into tracheal tubes with IDs as small as 2.5 mm. A longer, 100-cm endobronchial exchanger (Sheridan E.T.X.) is available for exchanging double-lumen tubes. They are firm—Shore hardness of 85, the same durometer measurement as the CAEC and Cardiomed International's Endotracheal Ventilation Catheter (ETVC)—although thermolabile and therefore subject to softening with heat. They are frosted to minimize drag and have a radiopaque stripe and distance
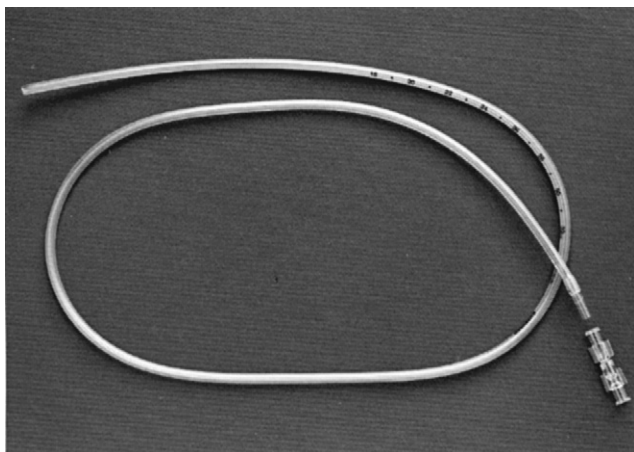


**Figure 50-4**   The tracheal tube exchanger (Sheridan T.T.X., Hudson RCI) is a simple catheter with no proximal or distal modifications. These devices are available in four diameters and two lengths. If the device is to be used for ventilation, it must be adapted by the user. There are no distal side ports, which makes jet injection potentially hazardous. (From Cooper RM: The difficult airway–II. *Anesthesiol Clin North Am* 13:683–707, 1995.)

markings. They have no side holes or connectors. An alternative product, the Sheridan JETTX Exchanger, is a longer device (100 cm), but it is available in only a single OD that is suitable for ETTs greater than 6.5-mm ID. It incorporates a proximal slip-fit connector that can be connected by a Luer-Lok to a jet ventilator. As with the T.T.X., there is only a single, distal end hole.

The JEM devices are available in nine sizes and are compatible with ETTs from 2.5- to 7.5-mm ID (JEM 325 to 400). These are single-use, high-density, polyethylene devices without proximal connectors or distal side holes.

## 2. Cook Airway Exchange Catheters

Cook Critical Care has developed a family of hollow stylets known as CAECs (Fig. 50-5A). They are available in 8.0-, 11-, 14-, and 19-F sizes, corresponding to 2.7-, 3.7-, 4.7-, and 6.3-mm ODs, respectively. They can be used to exchange ETTs with IDs of 3, 4, 5, and 7 mm, respectively. The 8-F CAEC is 45 cm long, and the others are 83 cm long. The devices are radiopaque, have distance markings between 15 and 30 cm from the distal end, and have two distal side holes and an end hole. Proximally, two types of connectors are secured and released by a patented Rapi-Fit adapter (see Fig. 50-5B). They provide a 15-mm connection or a Luer-Lok jet ventilation attachment and were designed for rapid removal and reattachment while the tracheal tube is being off-loaded and replaced. The length and IDs (1.6 to 3.4 mm) make manual ventilation with a resuscitation bag possible but useful only for short periods because resistance is very high. The Luer-Lok jet Rapi-Fit connector allows jet ventilation, but the paucity of distal side holes potentially increases catheter whip and the risk of barotrauma.[333]

Atlas and Mort examined the relationship between the diameter of the two larger CAECs and tolerance, as well as the ability to phonate and cough.[334] It is unclear whether their patients were randomly assigned to specific sizes of catheters. Phonation and discomfort were similar in both groups, with only 3 of 101 patients experiencing significant discomfort. Cough effort tended to be reduced with the larger CAEC, but this did not achieve significance. Atlas and colleagues also looked at a larger tube exchanger (JEM 400 Endotracheal Tube Changer, Instrumentation Industries Inc.), which they reasoned would have a higher degree of success as a tube exchanger. This device has an OD of 6.35 mm and is said to be stiffer. They adapted the JEM 400 using the Rapi-Fit connector from the CAEC 19-83 to enable jet ventilation.[335] This device, like the Sheridan JETTX, has only a single end-hole and is not recommended for jet ventilation.

Mort evaluated the concept of reversible extubation in patients with difficult airways.[336] From an institutional database, he identified patients who were extubated in the operating room, PACU, or ICU with a CAEC. The tube exchanger was left in place until the need for reintubation was considered unlikely. Over a 9-year period, 354 patients qualified. Two groups emerged: those who required reintubation while the AEC was still in place and another group who required reintubation within 7 days but after the AEC had been removed. Airway-related complications were compared for the two groups. The AEC dwell time was a mean of 3.9 hours (range, 5
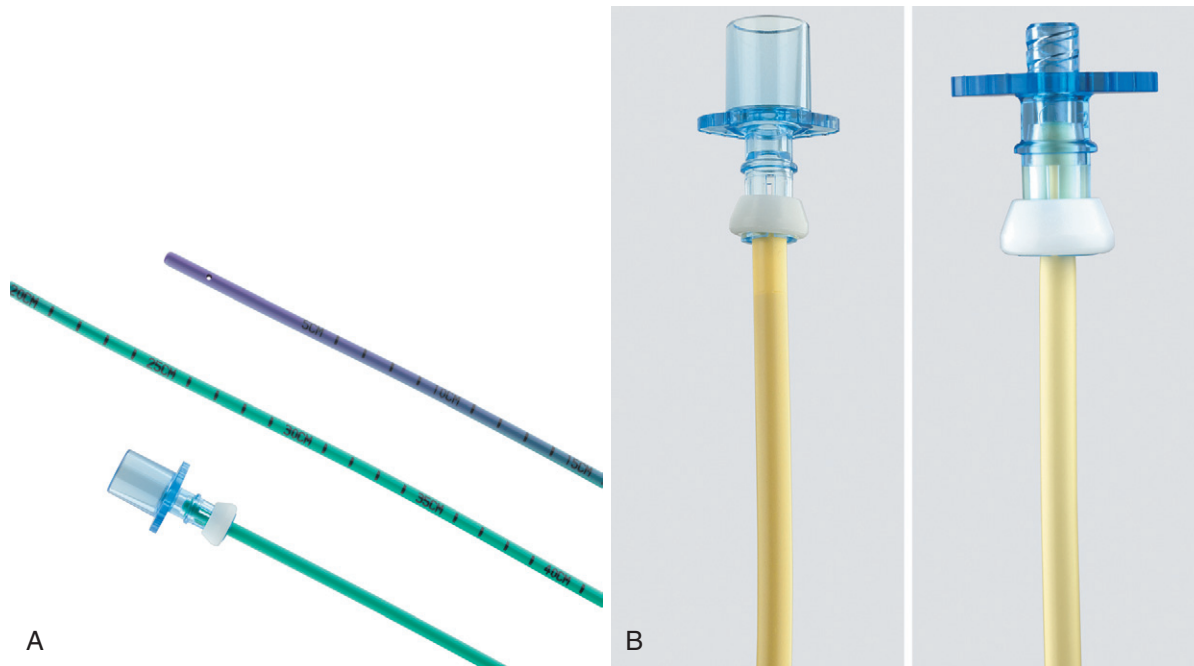
**Figure 50-5** The Cook airway exchange catheters are available in four diameters and two lengths. They are radiopaque and have distance markings at each centimeter throughout the working length. **A,** The proximal Rapi-Fit adapter, the middle section, and the distal end of a flexible-tip exchange catheter (available only on the double-lumen exchange catheter). Notice that there are two distal side holes and one end hole. **B,** Two Rapi-Fit adapters: a Luer-Lok jet adapter *(right)* and a 15-mm connector *(left)*. (Courtesy of Cook Critical Care, Bloomington, IN.)

minutes to 72 hours). Of 354 patients, 288 were extubated in the ICU and had previously required three or more laryngoscopic attempts or alternative devices to achieve intubation. Comparing the overall success rate in the two groups, 47 of 51 patients in the AEC group were successfully reintubated, 87% on the first attempt. The four failures in this group resulted from inadvertent removal of the AEC in three; one patient was rescued with an intubating LMA and flexible bronchoscope. Mild ($SpO_2$ < 90%) and severe hypoxia (<70%) were experienced by three and four patients, respectively. Of the 37 patients requiring reintubation without an AEC, the first pass success rate was only 14%, with mild and severe hypoxia in 50% and 19%, respectively; three or more laryngoscopy attempts in 28 of 36 patients; and the need for a rescue device in 32 of 36 patients.[336] In all cases, reintubation was attempted by a member of an anesthesia airway team: an attending anesthesiologist or a resident under direct supervision. Although reintubation over an AEC does not guarantee first-pass success, this strategy was strikingly more effective (87% versus 14% first-pass success) and had far fewer life-threatening complications.

### 3. Arndt Airway Exchange Catheter

The Arndt Airway Exchange Catheter (Cook Critical Care), a radiopaque exchange catheter, consists of an extrastiff Amplatz guidewire with positioning marks, a Rapi-Fit adapter, a bronchoscopic port, and a distally tapered 14-F (4.7-mm-OD), 70-cm-long AEC. It was designed for the exchange of LMAs, endobronchial tubes, and ETTs (Fig. 50-6). Bronchoscopy is performed through the existing airway device. The flexible end of the Amplatz

guidewire is introduced through the working channel of the bronchoscope and under visual control and is advanced to the level of the carina. The bronchoscope is removed over the wire, taking care that it is neither advanced nor withdrawn. The Arndt AEC is advanced over the guidewire to the appropriate depth, determined by aligning the distance markings on the airway device with that of the AEC. The original airway is carefully
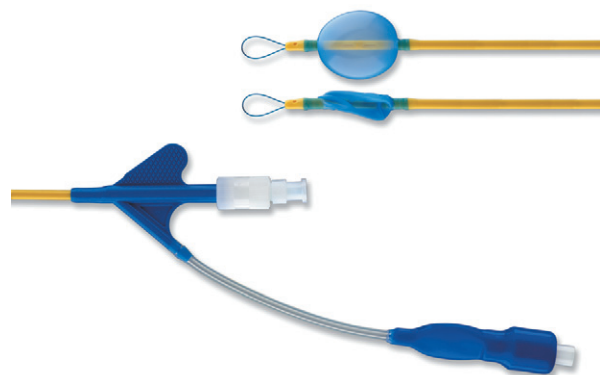


**Figure 50-6** The Arndt Airway Exchange Catheter Set (Cook Critical Care, Bloomington, IN) consists of a bronchoscopic adapter, a stiff Amplatz guidewire, a tapered airway exchange catheter, and two Rapi-Fit adapters. The adapter permits continuation of positive pressure ventilation while a bronchoscope is introduced through the original endotracheal tube (ETT). The guidewire is inserted through the bronchoscope's working channel, and the original ETT and bronchoscope are withdrawn. A tapered exchange catheter is advanced over the guidewire, and it may be connected with a 15-mm or jet Rapi-Fit adapter to provide ventilation. The replacement ETT is then advanced over the exchange catheter.

removed, and its replacement is advanced over the AEC.[309]

## 4. Endotracheal Ventilation Catheter

Cardiomed's Endotracheal Ventilation Catheter (Lindsay, Ontario, Canada) is made of a hybrid plastic (Fig. 50-7). It is 85 cm long and has a 4-mm OD (12 F) and 3-mm ID. It has a radiopaque stripe along its entire length and distance markings at 4-cm intervals. Proximally, it has a male hose barb with a threaded adapter welded into the catheter. These attachments have been constructed to prevent restriction of the catheter's ID. The threaded adapter connects to an easily removed Luer-Lok adapter. Distally, it is blunt ended with one end hole and eight helically arranged side holes to minimize catheter whip and jet ventilation pressures. Unpublished studies by the manufacturer found no significant softening over time at body temperature. This is desirable for a product that may remain in situ and be required to serve as a stylet. A metal guidewire is available to provide additional stiffness, but we have not found this to be necessary.

The ETVC was designed to facilitate reversible extubation.[337] We have used it in at least 600 patients, the



**Figure 50-7**   The Endotracheal Ventilation Catheter (ETVC) is available in one length (85 cm) with an outside diameter of 4 mm. It is nonthermolabile and has a radiopaque stripe along its length. There are distance markings every 4 cm. **A,** Proximally, there is a welded, barbed, plastic connector with a threaded Luer-Lok adapter for jet ventilation. **B,** Distally, there is a blunt end hole with eight helically arranged side holes, which minimize catheter whip and reduce the jet injection pressure.

first 202 of whom has been reported.[338] Although the ETVC had been used to facilitate reintubation, this was not required in most cases. In the original series, reintubation or tube exchange was performed in 32 (16%) of 202 cases, a rate that was very similar to that reported by others.[339] In both series, the ETVC[338] and the CAEC[339] were used primarily to maintain airway access. In the original publication, the ETVC was used for oxygen insufflation (31 patients), jet ventilation (45 patients), and post-extubation capnography (54 patients).[338]

Reintubation was successful in 20 (91%) of 22 attempts. One failure occurred with a softer prototype. The second failure resulted when an inexperienced and unsupervised operator attempted a tube exchange. Difficulty was occasionally encountered advancing the ETT through the glottis, similar to that experienced when using an FOB to intubate.[340] Tongue retraction with a laryngoscope blade should always be attempted when possible. ETT rotation or the use of an ETT such as the Parker Flex-Tip (Parker Medical, Boulder, CO) may prove useful. Because the ETVC has a relatively small OD, use of a smaller-diameter ETT is recommended.

Oxygen insufflation was achieved by connecting the male component of the ETVC to an oxygen flowmeter with 1 to 2 L/min flow rate, which was titrated to the arterial saturation. Jet ventilation is discussed later.

Complications included barotrauma, intolerance, unintended dislodgment, and tracheal perforation. Intolerance occurred in 2 of 202 patients (typically because of carinal irritation) and in 1 patient recently recovered from status asthmaticus. Intolerance should prompt reassessment of the depth of insertion. If the depth is clinically or radiographically appropriate, and the ETVC (or other AEC) continues to be required, tolerance usually can be achieved by instilling lidocaine through the device. Most patients, including those with reactive airways, have tolerated the ETVC without difficulty. Dislodgement occurred when the ETVC was inadequately secured or the patient "tongued" the catheter out. Proper fixation is essential (see Fig. 50-3). Tracheal or bronchial perforation with different instrumentation has been described.[325,326] In our case, it occurred in a patient with obstructing, proliferative tracheal papillomatosis and a chronic tracheostomy. A rigid prototype catheter was inserted alongside the tracheostomy, penetrating the posterior tracheal wall. Jet ventilation resulted in fatal barotrauma. Aspiration and laryngospasm have not been observed.

## H. Exchange of Double-Lumen Tubes

Double-lumen tubes are selected for procedures requiring lung isolation. Although the resistance through a larger double-lumen tube does not preclude postoperative ventilation or weaning, it may be desirable to replace it with a single-lumen tube, particularly if care is to be transferred to an area where familiarity is lacking or ventilatory support may be prolonged. The double-lumen tube may have to be changed because of damage to a cuff or because the initial tube was an inappropriate size. Substitution can often be achieved by direct or indirect (e.g., video) laryngoscopy. While the larynx is in view, the double-lumen tube is withdrawn and immediately
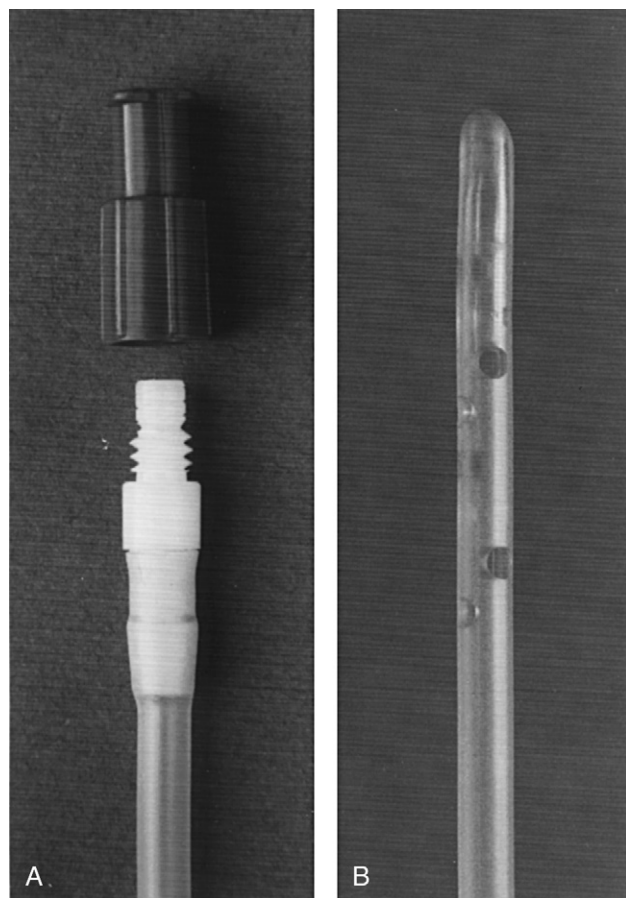
replaced with a single-lumen tube or replacement double-lumen tube. Occasionally, this cannot be accomplished.[341] Whether the substitution is a single- to double-, double- to single-, or double- to double-lumen tube, the requirements are similar, and the previously described tube exchangers may not be sufficiently long or firm.[342,343]

The Sheridan E.T.X. Exchanger is 100 cm long and was designed for use with the Sheri-Bronch double-lumen tube (35 to 41 F). It has one distal end hole. There are distance markings and tracheal and bronchial markings to indicate when the distal tip of the E.T.X. is at the opening of the distal lumen. This device lacks a connector for manual ventilation, and the manufacturer recommends against the use of jet ventilation.

Extra Firm with Soft Tip tube exchangers (Cook Critical Care) are available in 11- and 14-F sizes. They are 100 cm long and were designed specifically for the exchange of double-lumen tracheal tubes (designations C-CAE-11.0-100-DLT-EF ST and CAE-14.0-100-DLT-EF ST). These devices have ODs of 3.7 mm and 6.3 mm, respectively. The *soft tip* refers to the distal 7-cm segment of the catheter.

### 1. Visually Assisted Tube Exchange

It is difficult to imagine a safer or more secure confirmation of a tube exchange than seeing one tube replace another. This is not always possible, but it sometimes may be achieved by combining techniques, such as indirect laryngoscopy (video laryngoscopy) with a tube exchanger, thereby providing security, visual verification, and a means of correcting difficulties with tube advancement.

The recently discontinued WuScope (Achi Corp., Fremont, CA, and Asahi Optical, Tokyo, Japan) has been used to exchange double-lumen tubes and conventional ETTs in patients with difficult airways.[344-346] The WuScope provided a good glottic view in a morbidly obese patient with adult respiratory distress syndrome and permitted insertion of a suction catheter anterior to the existing nasotracheal tube. The nasotracheal tube was withdrawn, and the replacement oral ETT was easily advanced over the suction catheter with minimal interruption of mechanical ventilation. We think that a visually direct tube exchange with a WuScope, Bullard Scope (Circon, Santa Barbara, CA), Upsher UltraScope (Mercury Medical, Clearwater, FL), or video laryngoscope is preferable to blind passage of an ETT over a flexible bronchoscope or tube exchanger. A hollow tube exchanger, however, would have permitted oxygen insufflation or jet ventilation had desaturation or difficulties with tube advancement occurred. A supraglottic view (in contrast to the infraglottic view provided by a flexible bronchoscope) of the exchange enables identification and correction of an ETT impingement or the outward migration of the exchange catheter.[347]

The concept of visually direct tube exchange was assessed retrospectively in a population in whom no view of the larynx could be obtained by direct laryngoscopy.[348] Using one of three indirect laryngoscopes—the Glidescope (Verathon Medical, Bothell WA), Airtraq (Prodol Meditec, Vizcaya, Spain), or McGrath Series 5 (Aircraft Medical, Edinburgh, Scotland)—in conjunction with an AEC, 51 exchanges were performed. Four of the cases involved a double-lumen tube converted to a conventional ETT or a nasal to oral conversion. Thirty-seven patients had previously difficult intubations requiring multiple attempts or a rescue technique. Most of the patients were obese or morbidly obese and had a positive fluid balance of more than 10 L over the previous 4 days. The intention of the study was not to compare the specific devices but rather to evaluate the advantages of visually directing the tube exchange. Mort reported that this was achieved on the first attempt in 47 of 49 patients.[348] In the two failures, use of the Glidescope video laryngoscope afforded an excellent view of the AEC migrating cephalad, allowing rapid reintubation with a styletted ETT. He pointed out that had the tube exchange been performed blindly, the replacement would have failed, with potentially serious consequences. Others have made similar observations.[349,350] These findings and our personal experiences are consistent with Mort's recommendations.

### 2. Conversion from Nasal to Oral Intubation

Blind or bronchoscopically assisted nasal intubation is sometimes performed when oral approaches are difficult or unsuccessful. The nasal tube may have to be converted to an oral one because of complications or the intended surgery. Whenever possible, this should be done under visual control, assisted by a flexible bronchoscope,[351] rigid fiberoptic device, or video or optical laryngoscope.[344,348] The more anatomically or physiologically challenging the patient, the more compelling the case for including a tube exchanger.

Gabriel and Azocar described a patient in halo fixation in whom the connector was detached and the nasotracheal tube was advanced deeper into the trachea.[352] The tube was then grasped close to the uvula with forceps and digitally extracted through the mouth. Novella used a Sheridan T.T.X. to convert a nasal to an oral tube in a patient with Klippel-Feil syndrome who first underwent orthognathic surgery and subsequent septorhinoplasty.[353] After completion of orthognathic surgery, the T.T.X. was inserted into the nasal tracheal tube, and the latter was withdrawn. The T.T.X. was then grasped with two Magill forceps; the caudal one was used to stabilize the catheter, and the cephalad one was used to withdraw the proximal end out of the mouth. An oral tube was then railroaded over the T.T.X. Cooper described a similar maneuver in a patient in whom oral fiberoptic intubation could not be accomplished, but fiberoptic nasal intubation was achieved.[354] He passed an ETVC through the existing nasal tube and removed the latter. The ETVC was then stabilized with caudal Magill forceps and withdrawn through the mouth with the cephalad forceps. Oxygen insufflation was provided through the ETVC, which was then used to thread an oral tube into the trachea. In this case, oxygen desaturation was avoided, although the procedure was easily and quickly accomplished.

### 3. Conversion from Oral to Nasal Intubation

During efforts to convert from an oral to a nasal tracheal tube, Sumiyoshi and coworkers used negative-pressure ventilation during the tube exchange.[355] Their patient was in a halo and chest cast due to a cervical injury, and

laryngoscopy had been unsuccessful. An attempt to introduce a 4.8-mm FOB adjacent to the existing tube (with a tube exchanger through it) was unsuccessful. A subsequent effort involved a 3.5-mm FOB and a 7-F METTRO using negative pressure to achieve ventilation. A smaller, hollow tube exchanger might have been successful and could have avoided the risk of negative-pressure pulmonary edema resulting from an ETT and FOB occupying a small glottic opening.[356] Smith and Fenner performed an oral to nasal conversion using a 4.0-mm-OD, flexible bronchoscope, which they inserted through the glottis and anterior to an oral tube.[357] The oral tube was withdrawn, and a nasal tube was advanced over the FOB. Many of the difficulties in performing a tube exchange can be avoided with the use of indirect (fiberoptic or video) laryngoscopes, ideally in combination with AECs.

### 4. Conversion from Supraglottic Airways to Endotracheal Tubes

A discussion of the conversion of various SGAs to ETTs is beyond the scope of this chapter, but the process is addressed elsewhere. Whenever possible, this conversion should be facilitated by visual guidance, using direct laryngoscopy or an indirect technique. These techniques include the use of a flexible bronchoscope and Aintree catheter or a video laryngoscope.[358,359]

### 5. Changing Tracheostomy Tubes

A tracheostomy tube may need to be replaced because of tube damage, such as cuff rupture, occlusion with secretions, or conversion to another type or size of tracheostomy tube. If the tracheostomy was recently performed, the tissue is friable. In patients in whom a false passage has been created during the original procedure, in those with poor tissue integrity, or those highly dependent on supplemental oxygen, exchanging the tracheostomy tube can be difficult and fraught with complications. The Weinmann Tracheostomy Exchange Set (Cook Critical Care) (Fig. 50-8), consists of a Ciaglia Blue Rhino tracheostomy dilator, a CAEC, Rapi-Fit adapter, and two loading dilators (26 and 28 F). The 45-cm-long exchange catheter is passed through the tracheostomy tube to be replaced; its cuff is deflated, and the tube is removed over the exchange catheter. The replacement tracheostomy



**Figure 50-8**   Weinmann Tracheostomy Exchange Set. (Courtesy of Cook Critical Care, Bloomington, IN.)

tube and its loading dilator are placed over the AEC, which is connected to the Rapi-Fit adapter, permitting ventilation as required. The loading catheter and tracheostomy catheter are advanced using the AEC as a guide. If stomal dilatation is required, the set is equipped with Ciglia Blue Rhino dilators (32 to 38 F).[360] The only description of this device advocates jet ventilation and high-flow oxygen insufflation. AEC insertion must be atraumatic, confirmed with a carbon dioxide ($CO_2$) tracing, and the depth verified to ensure it is not beyond the carina. Only the amount of oxygen ($O_2$) required to prevent significant oxygen desaturation should be supplied.

## VII. JET VENTILATION THROUGH STYLETS

The preceding sections stressed the importance of being able to supplement oxygenation during a tube exchange. In most circumstances, a patient's oxygen content can be adequately sustained with insufflation, obviating the need for high-pressure jet ventilation. If oxygen requirements are high before a tube exchange, the equipment should be immediately available to provide jet ventilation. This equipment consists of a manually cycled, Venturi-type jet ventilator with a Luer-Lok adapter and an in-line pressure-reducing valve (see Fig. 50-8).[361] The objective of jet ventilation is to correct life-threatening hypoxemia—not to normalize arterial blood gases. Although the achievement of normal $PaCO_2$ may be attainable, the risks quite likely exceed the benefits.[343] Barotrauma has occurred through such misguided objectives, and it has been fatal in some cases.

As an example of the risks, the Chief Coroner of Ontario, Canada, investigated the death of a man who had intermaxillary fixation after orthognathic surgery. He was awake and comfortable at the conclusion of surgery. He was extubated over an AEC, through which oxygen was insufflated at 5 L/min. On arrival in the PACU, he was distressed and complained of back pain and difficulty breathing. Shortly thereafter, he had a cardiac arrest. Needle thoracentesis revealed a tension pneumothorax, and although the initial resuscitation restored a pulse, he developed hypoxic encephalopathy and died. It is unclear whether the insertion was traumatic (although the autopsy did not reveal a laceration or perforation at the distal tip location), whether the flows were at some point much higher, or whether jet ventilation had been employed at any time. The specific device, its size, and details regarding its insertion and fixation are unknown, but it is advisable to obtain a capnographic tracing before applying oxygen insufflation or jet ventilation. The lowest possible flows and driving pressures should be used.

### A. In Vitro Studies

Transtracheal jet ventilation by means of an intravenous catheter or intratracheal ventilation using a stylet or tube exchanger has been advocated in the management of the "cannot intubate, cannot ventilate" patient.[5,362] The inspiratory volume depends on the driving pressure, injection

time, respiratory compliance and resistance, and resistance of the tube exchanger. The latter is determined by the catheter's ID and length. The expiratory volume depends on exhalation time, elastic recoil of the lungs, and airways resistance.[361,363] Mismatch between inspiratory and expiratory volumes can have serious consequences.

In vitro studies using jet stylets have been conducted to determine flow, pressure, and entrainment characteristics. Using an in vitro model, with three sizes of Sheridan T.T.X. catheters, Dworkin and colleagues measured the inspiratory and expiratory flows resulting from a 50-psi injection as the simulated upper airway resistance, lung compliance, gas flow rate, and injection times were varied.[363] The upper airway resistance was determined by the effective tracheal diameter, which they defined as a computed difference between the OD of the T.T.X. and the tracheal diameter. They simulated upper airway obstruction by using various sizes of ETT adapters (11- to 3.5-mm ID) in the proximal airway. The gas flows through the large, medium, and small tube exchangers, when connected to a pressure source of 50 psi, were 63, 33, and 12 L/min, respectively. In their model, if the difference between the tracheal and T.T.X. diameters resulted in an effective tracheal diameter that was greater than 4 to 4.5 mm, air trapping did not occur. Because increased upper airway resistance and reduced effective tracheal diameter resulted in larger tidal volumes, they concluded that jet ventilation through a long catheter that was positioned close to the carina caused little Venturi effect or air entrainment. Placement of the catheter close to the carina may ensure a higher oxygen concentration by reducing room air entrainment, but it also increases the risk of distal catheter migration and barotrauma.

In another in vitro model, calculations based on oxygen dilution and direct measurement using a pneumotachograph revealed that air entrainment accounted for 0% to 31% of the inspired volume.[364] The largest T.T.X. and lung compliance resulted in the greatest entrainment. Gaughan and colleagues used a high driving pressure (50 psi), long inspiratory time (1 second), and brief expiratory time (1 second).[364] Even within a low-compliance system, the large T.T.X. was associated with excessive tidal volumes.

Prolonging expiratory time reduces the minute ventilation by reducing the respiratory rate. This technique still exposes the lungs to potentially injurious tidal volumes. An alternative approach is to reduce the driving pressure. Gaughan and coworkers assessed the tidal volumes and air entrainment in a model lung with a range of compliance sets ventilated by high- and low-flow regulators through 14- and 16-gauge intravenous catheters.[365] Their high-flow regulator at steady state produced flow rates of 320 L/min at 100 psi, whereas the low-flow regulator produced flows up to 15 L/min at 9 to 5 psi. Intravenous catheters, because of their short length, offer considerably less resistance to flow. Their proximity to the upper airway also results in greater air entrainment (15% to 74%). Both high- and low-flow regulators allowed adequate minute ventilation in the setting of normal tracheal and bronchial diameters and normal compliance. The investigators recommended that during transtracheal jet ventilation, when low-flow regulators were used, an inspiratory-to-expiratory (I:E) ratio of 1:1 should be used because it yields the greatest minute ventilation. Although this observation is undoubtedly true, it remains to be determined whether such high minute volumes are clinically necessary or safe.[343,361]

## B. In Vivo Studies

Chang and colleagues provided intraoperative jet ventilation using a 3.5-mm chest tube as a jet catheter.[332] They ventilated with 15 psi at 10 to 16 breaths/min and continued until spontaneous ventilation was deemed adequate. The patient recovered but had a left pneumothorax that was attributed to catheter migration and unilateral ventilation. The investigators mentioned that they had encountered three cases of pneumothoraces and one pneumoperitoneum in approximately 600 similar procedures. They drew attention to the importance of catheter placement and advised that even brief airway obstruction can result in barotrauma. However, they failed to mention that vocal fold apposition occurring during recovery might promote this complication. In a subsequent paper, the same investigators stated that the jet stylet had been used for the ventilation of six patients, resulting in normocarbia and adequate ventilation.[331] Baraka described a patient with a poor laryngeal view in whom an ETT exchange was facilitated using a CAEC.[366] This was advanced until resistance was encountered. Jet ventilation at 50 psi was commenced before withdrawal of the ETT or deflation of its cuff. The result was right-chest expansion but incomplete deflation, but within three breaths, asystole was observed. A needle thoracotomy confirmed the diagnosis of tension pneumothorax, and sinus rhythm was restored.[366]

Egol and colleagues described pneumothoraces and a pneumoperitoneum in three patients using a variety of delivery devices and driving pressures.[333] They included an 18-F suction catheter at 50 psi, a nasogastric sump tube at 20 psi (inspiratory time at 30%), and a fiberoptic laryngoscope at 40 psi. They attributed the barotrauma observed to incorrect catheter placement, ventilation during phonation, and possible direct mucosal penetration from jet injection. They examined the relationship between the number of distal side holes in the tube exchanger and the pressure at the catheter tip. At any given driving pressure, the more side holes, the lower the pressure at the catheter tip. They recommended vigilance regarding the location of the catheter tip, including avoidance of direct mucosal contact and insertion into orifices where exhalation might be restricted. They advocated securing the catheter to minimize migration, use of catheters with multiple side holes, use of small-diameter jet catheters to minimize the resistance to exhalation, and use of the minimal effective driving pressure. They encouraged the development and use of an effective pressure sensor and pressure-cutoff device.

The ETVC has an end hole and eight distal side holes (see Fig. 50-7). Its use to provide jet ventilation during general anesthesia with muscle relaxation on 45 occasions was described.[338] Its attachment to a handheld jet ventilator with a pressure-reducing valve is shown in Figure 50-9. Between 1991 and 1993, Irish and coworkers
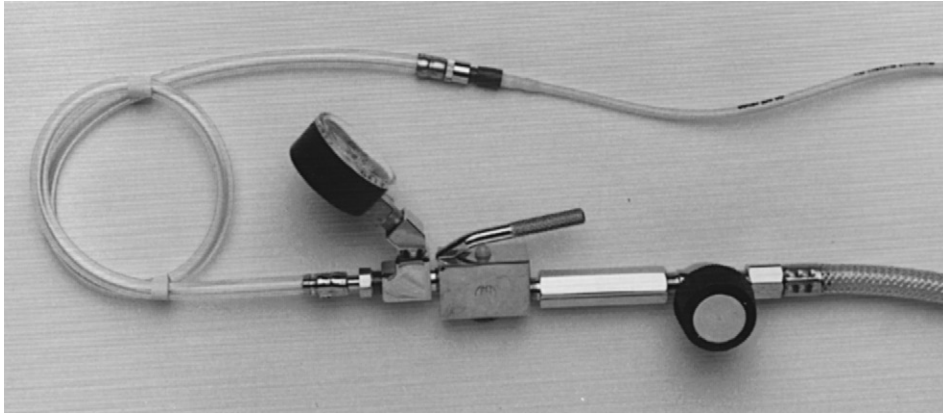
**Figure 50-9**   An Endotracheal Ventilation Catheter (ETVC) is connected to a handheld jet injector. The Rapi-Fit adapter or the Sheridan JETTX exchanger can be similarly attached. A pressure-reducing valve enables the operator to select a driving pressure that yields adequate chest expansion while minimizing the risk of barotrauma.

used this device with a driving pressure of 50 psi in 25 anesthetized and paralyzed patients undergoing percutaneous dilatational tracheostomies.[367] They observed barotrauma in one patient. Arterial blood gases in 12 consecutive, critically ill patients revealed a pH of 7.37 ± 0.09, $Pa_{CO_2}$ of 45.5 ± 10.8, and $Pa_{O_2}$ of 256 ± 126 (mean ± SD). In a subsequent report, a patient ventilated for 90 minutes at only 20 psi developed a pneumothorax.[343] Chan and Manninen also described use of the ETVC to provide jet ventilation.[368] After performing a fiberoptic intubation in a patient with an unstable cervical spine, they discovered that the cuff of the ETT had been damaged. They inserted a flexible bronchoscope through the other nostril and advanced it through the vocal cords anterior to the original ETT. They then passed an ETVC through the original ETT and provided three breaths of jet ventilation at 50 psi. The patient developed a pneumothorax. Unfortunately, they used a high driving pressure through an exchange catheter that might have been too deeply inserted in the setting of near-complete glottic occlusion (i.e., partial cuff deflation, 6-mm ETT, and an FOB passing through the vocal cords).

These cases reinforce the general principles previously stated.[343,369] The need for jet ventilation should always be weighed against its possible risks. It should be immediately available and used when there is evidence of deteriorating oxygenation. If a 15-mm connector is available, capnography should be used to confirm intratracheal placement. An in-line pressure-reducing valve should be used, and ventilation should begin with the lowest pressure capable of producing adequate chest expansion. The duration of inspiration should be minimized while the duration of exhalation is determined by observing the return of the thoracic diameter to its position before inspiratory. The depth of catheter insertion should be far enough from the carina that distal migration does not occur but not so proximal that jet ventilation results in the catheter's ejection from the glottis. Multiple distal side holes can reduce catheter whip and the distal catheter pressure during jet ventilation. Every effort must be taken to minimize expiratory resistance.

## VIII. CONCLUSIONS

Successful airway management does not end with tracheal intubation, any more than a safe flight is only concerned only with the take-off. Although respiratory complications are more common at extubation than during intubation, most are relatively minor and do not require reintubation. However, the need for reintubation cannot always be predicted. Reintubation may prove to be difficult and dangerous in a variety of circumstances. The ASA Task Force and the Canadian Airway Focus Group have recommended that each anesthesiologist have a preformulated strategy for extubation of the difficult airway. A risk stratification scheme can be used to identify patients for whom special extubation precautions seem to be warranted. Although many strategies are available, their benefits have not been subjected to rigorous evaluation. A reversible extubation can be performed with a tube exchanger. Use of a stylet does not guarantee that reintubation will succeed. The probability of a successful tube exchange may be enhanced if it can be performed under visual control using direct or indirect laryngoscopy. Carefully used, however, it should enhance patient safety by providing oxygen insufflation and jet ventilation while other avenues are explored.

## IX. CLINICAL PEARLS

- Careful planning of tracheal extubation or tube exchange is as vital as the planning required for intubation. Airway complications are as common after tube removal as during insertion.

- Extubation carries the risk of reintubation and the risk of failed reintubation.

- Reintubation may be required because of an airway obstruction or because of failure of oxygenation, ventilation, clearance of secretions, or airway protection.

- Anticipating a successful extubation is an inexact science. Any emergent reintubation is likely to be more complex due to urgent conditions and physiologic instability.

- Reintubation may fail because of inadequate access to the airway (e.g., halo fixation, maxillomandibular fixation), preexisting anatomic features (e.g., retrognathism, prominent incisors), inadequate preparation, lack of expertise or insufficient information (e.g., emergencies), a rapidly deteriorating clinical state, or blood, secretions, or swelling obscuring the visual field.

- Many threatening circumstances can be anticipated and managed preemptively with an extubation strategy.

- Extubation strategies include deep extubation, bronchoscopic examination under anesthesia through a supraglottic airway (SGA), substitution of an endotracheal tube with an SGA, and extubation over a tube exchanger.

- The safest extubation strategy may be a surgical airway.

- A supraglottic device or tube exchanger should be left in place until it is likely that reintubation will not be required. Premature withdrawal is a common mistake.

- A reintubation strategy may include the judicious administration of oxygen by insufflation or jet ventilation; advancement of an endotracheal tube over the tube exchanger, preferably with tongue retraction; or indirect laryngoscopy.

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