

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

Acute Upper Airway Obstruction

Antoine Eskander, M.D., John R. de Almeida, M.D., and Jonathan C. Irish, M.D.

From the Department of Otolaryngology–Head and Neck Surgery/Surgical Oncology (A.E., J.R.A., J.C.I.) and the Institute for Health Policy Management and Evaluation (A.E., J.R.A.), University of Toronto, Sunnybrook Health Sciences Centre and Michael Garron Hospital (A.E.), the Institute for Clinical Evaluative Sciences (A.E.), and Princess Margaret Cancer Centre (J.R.A., J.C.I.) — all in Toronto. Address reprint requests to Dr. Irish at the Department of Otolaryngology–Head and Neck Surgery, University Health Network, Princess Margaret Cancer Centre, 200 Elizabeth St., 8NU-882, Toronto, ON M5G 2C4, Canada, or at jonathan.irish@uhn.ca.

N Engl J Med 2019;381:1940-9.
DOI: 10.1056/NEJMra1811697

Copyright © 2019 Massachusetts Medical Society.

ACUTE UPPER AIRWAY OBSTRUCTION IS A LIFE-THREATENING EMERGENCY and requires immediate assessment and intervention with little margin for error, making it a constant challenge for clinicians.^{1,2} Substantial advances have been made in preventive medicine, our understanding of the pathophysiology of acute upper airway obstruction, and surgical and anesthetic technologies, but management of the obstructed airway remains one of the most challenging emergencies in clinical medicine. Arguably the largest effect on acute upper airway obstruction in the modern era has occurred through preventive medicine with the widespread implementation of *Haemophilus influenzae* type B vaccination, a development that has resulted in marked declines in the incidence of and the mortality related to epiglottitis and pneumonia.³⁻⁵ Epiglottitis, once a very common occurrence, is now exceedingly rare. Similarly, the public health campaign to widely implement and use epinephrine autoinjectors in persons with anaphylaxis has successfully decreased the need for airway intervention in many cases.

Treating acute upper airway obstruction in the clinical setting is complicated and requires managing a sometimes chaotic environment, understanding the potentially multiple causes of disease (infectious, inflammatory, traumatic, mechanical, and iatrogenic), and having the technical ability to quickly secure a challenging airway. Each patient with acute upper airway obstruction is distinct. The treating clinician must consider several factors, including the patient's age, coexisting conditions, ability to remain in the supine position, level and severity of obstruction, stability of the cervical spine, ability to ventilate, and level of anxiety.

The historical algorithm for the management of acute upper airway obstruction was a stepped series of interventions that started with the administration of high-flow oxygen and the use of conservative measures and extended to bag-mask ventilation, intubation, and the surgical opening of the airway. This algorithm has been modified as our understanding of the pathophysiological underpinnings of the most common causes of acute upper airway obstruction has increased and the anesthetic and surgical technologies used have advanced. Here we review recent advances that have affected the management of acute upper airway obstruction.

ANATOMY

A brief overview of the anatomy of the upper airway is required for a thorough understanding of advances in the causes of obstruction and in emerging technologies for treatment (Fig. 1). Acute upper airway obstruction can occur at any one of several anatomical levels. In infants, who have historically been considered to be obligate nasal breathers, nasal or nasopharyngeal masses or obstructions can lead to airway distress, although in most cases not to airway obstruction, since open-mouth breathing can compensate for this level of obstruction.

Obstruction at the level of the oropharynx (soft palate, palatine tonsils, posterior pharyngeal wall, and tongue base), larynx (including the supraglottic, glottic, and

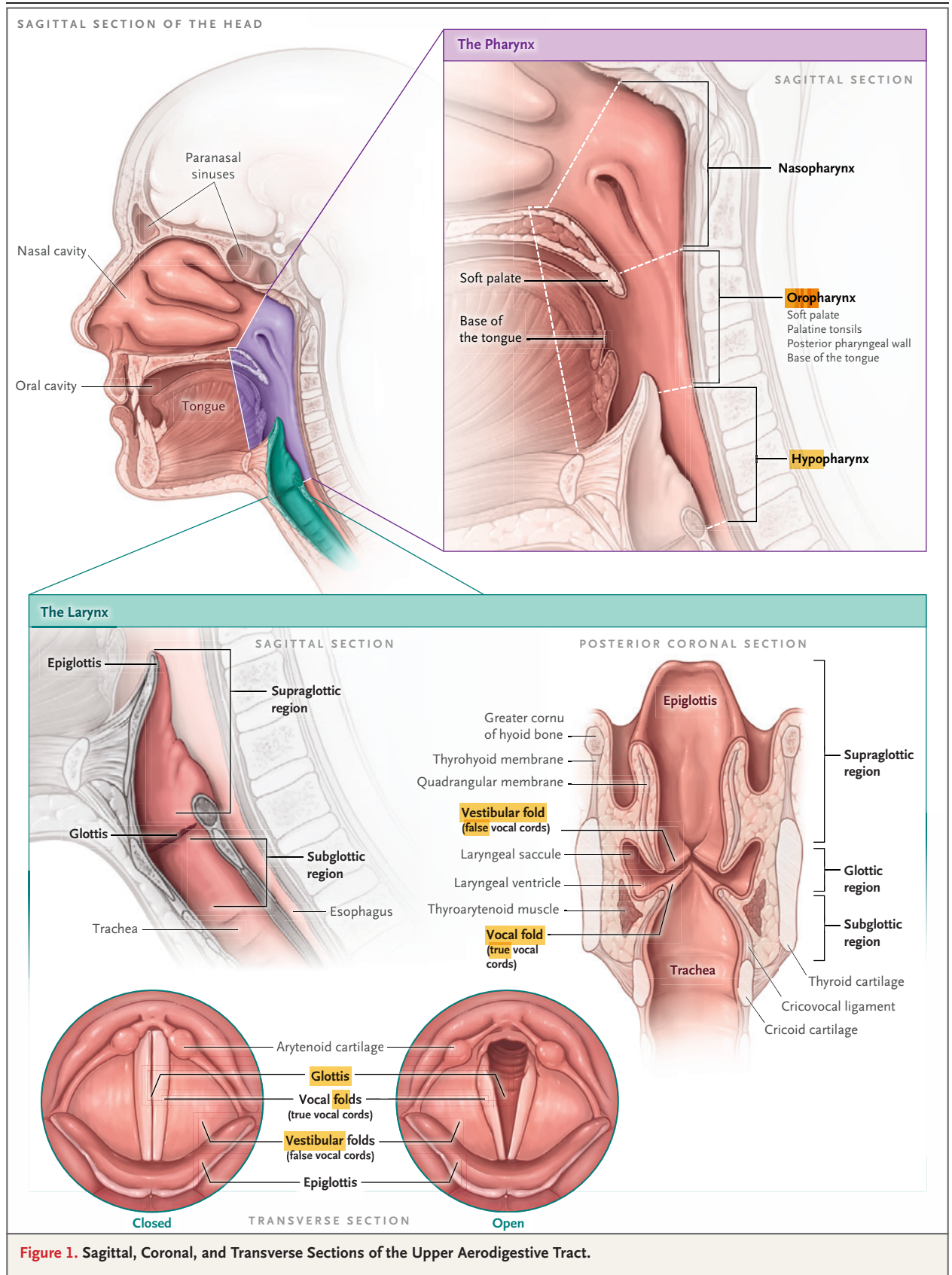


Figure 1. Sagittal, Coronal, and Transverse Sections of the Upper Aerodigestive Tract.

subglottic subsites), and hypopharynx may lead to acute upper airway obstruction. Historically, the most common site for airway obstruction was the supraglottic larynx, which includes the epiglottis, the aryepiglottic folds, the arytenoids, and the ventricular folds (or “false cords”). Obstruction can occur at the level of the glottis, or “true” vocal cords. With adduction, the vocal cords allow for phonation and, more important, when effortful and strong, produce an effective cough, an important airway-protection maneuver. The posterior aspect of the glottis carries the largest cross-sectional area (Fig. 1) and is therefore responsible for the majority of airflow, which explains why lesions in the anterior glottis have less effect on the airway and greater effect on voice, with the opposite effects applying to lesions on the posterior glottis.⁶ Distal to the glottis is the subglottis, which is the narrowest portion of the airway in neonates, followed by the cervical trachea.

PATHOPHYSIOLOGICAL FEATURES

Much of our current understanding of the pathophysiological features of acute upper airway obstruction can be discerned from our understanding of the neurophysiological modulation and pathophysiological features of obstructive sleep apnea. Apnea, whether acute or chronic, causes an increase in respiratory effort. Phrenic and sympathetic nerve activation from airway obstruction are largely driven by chemoreceptor afferents and glutamatergic neurotransmission in the nucleus tractus solitarius.⁷ In acute upper airway obstruction, severe hypoxemia can lead to cardiac arrest. Chronic obstructive sleep apnea is associated with major cardiac events, hypertension, arrhythmias, congestive heart failure, and stroke.^{8,9}

PHYSICAL EXAMINATION

One of the most important advances in airway management has been the development of physical examination grading scales to help predict a difficult airway. Some scales rely only on the visibility of the vocal cords on laryngoscopy (e.g., the Cormack–Lehane grading scale, on which grades range from 1 to 4, with higher grades indicating poorer visibility), whereas others use prelaryngoscopic factors (e.g., the Wilson

score, in which a scale of 0 to 10 is used to indicate the likelihood of difficulty with intubation, with higher scores indicating greater difficulty).¹⁰⁻¹² The factors used in assessment include increased weight, decreased cervical spine mobility, decreased jaw mobility, retrognathia, and prominent incisors, all of which are associated with increased difficulty with intubation. Other aspects of physical examination that can be used to assess the likelihood of a difficult intubation include the hyomental–thyromental distance, with shorter distances indicating greater difficulty, and the Mallampati score, which is used to assess the visibility of oropharyngeal structures with the mouth opened maximally. For the latter, a scale of 1 to 4 is used, with higher numbers associated with poorer visibility. Findings from a recent systematic review suggest that the best predictor is the inability to bite the upper lip with the lower teeth.¹³ However, no finding on physical examination and no specific risk factor consistently rule out a potentially difficult intubation. In short, one should always be prepared to manage a difficult airway.¹³

ADVANCES IN TREATMENT ACCORDING TO CAUSE

An anatomical approach to classifying the causes of acute upper airway obstruction is the most practical (Table 1). Congenital causes are more common in children, and neoplastic causes are more common in adults, particularly in patients with a substantial history of smoking or alcohol abuse. Although chronic causes of airway obstruction are not the focus of this article, all chronic causes can reach a point at which there is patient decompensation, and what was once a chronic problem is now acute. Thus, it is important to obtain a detailed history, even in patients with an acute presentation, to delineate the cause. Infection and inflammation are the most common causes of acute upper airway obstruction.

CROUP

The parainfluenza viral infection of the supraglottis known as croup occurs in 3% of children 6 months to 3 years of age.¹⁴ Children with croup have a barking cough, inspiratory stridor, hoarseness, and respiratory distress, symptoms that typically have an abrupt onset at night.¹⁵ The mainstay of treatment is oral or inhaled

glucocorticoids. Oral dexamethasone should be administered in a single dose of 0.6 mg per kilogram of body weight. Nebulized dexamethasone should be administered in a dose of 160 μ g, with a fill volume of 3 ml and the oxygen flow set to 5 to 6 liters per minute. A Cochrane review of 43 randomized clinical trials involving 4565 children reported that glucocorticoids reduced symptoms at 2 hours and were associated with shorter hospital stays and lower rates of return to the hospital than placebo or other pharmacologic treatments.¹⁶ Racemic epinephrine and humidified cold air can also be useful adjuncts.

EPIGLOTTITIS AND SUPRAGLOTTITIS

Vaccination against *H. influenzae* type B has resulted in a lower incidence of epiglottitis and lower mortality associated with epiglottitis and pneumonia in children.³⁻⁵ Supraglottitis, like epiglottitis, is also rare in adults. It is typically caused by infectious processes but may also result from injuries from trauma or inhalation or from the ingestion of caustic substances. Patients with an aggressive disease course are typically male, have dyspnea or stridor, and present with edema of the epiglottis or aryepiglottic folds. Elevated C-reactive protein levels and hyperglycemia are also typical. These patients often have a history of recurrent episodes.¹⁷ The most common presenting symptoms are sore throat (79%) and dysphagia (71%). Stridor (3.6%) and dyspnea (6.7%) are less common but are associated with increased need for airway intervention.¹⁷

LUDWIG'S ANGINA

Patients with Ludwig's angina have bilateral infection of the sublingual and submandibular spaces that is characterized by submental and submandibular induration, cellulitis, and a swollen and tender floor of mouth, all of which result in a posteriorly displaced tongue. This displacement ultimately leads to obstruction at the oropharyngeal and supraglottic levels and can be life-threatening. The most common causes of Ludwig's angina are dental infections, followed by sialadenitis, peritonsillar abscess, abscess involving the parapharyngeal space, traumatic injuries to the oral cavity, and mandibular fractures. Conservative management with intravenous antibiotics is associated with a risk of airway compromise that is nearly 10 times as high as that

Table 1. Causes of Acute Upper Airway Obstruction According to Subsite.*

Supraglottic
Croup
Supraglottitis, epiglottitis, or neck abscess
Ludwig's angina
Angioedema
Tumor
Foreign body
Glottic
Iatrogenic (bilateral vocal cord paralysis)
Tumor
Foreign body
Subglottic or tracheal
Foreign body
Subglottic stenosis
Tumor (extrinsic or intrinsic)

* Inhalation and traumatic (penetrating or blunt) injuries can affect the airway at any level and are therefore not included.

in patients who receive early surgical drainage (26.3% vs. 2.9%).¹⁸ Immunocompromised patients are at a higher risk for airway compromise, other complications (e.g., sepsis and pneumonia), longer hospital stays, and death.¹⁹ Overall, the mortality associated with Ludwig's angina is approximately 8%.²⁰ Therefore, the standard of care includes securing the airway followed by a formal incision and drainage of the sublingual and submandibular spaces.

ANGIOEDEMA

The condition angioedema is characterized as recurrent, nonpitting, nonpruritic swelling of the deep layers of the skin and mucosal tissues. It may be hereditary, acquired, drug induced, or idiopathic.²¹ Hereditary angioedema, an autosomal dominant condition, results from deficiency of the C1 esterase inhibitor, which may reflect inadequate levels (type I) or function (type II). Deficiency of the C1 esterase inhibitor changes activation of the complement and contact systems and also affects the regulation of coagulation and fibrinolysis, although to a lesser extent.²¹ Table 2 summarizes the differences among hereditary angioedema, acquired angioedema, and angioedema that is associated with angiotensin-

Table 2. Subtypes of Angioedema and Related Laboratory Abnormalities.*

Diagnosis	C4 Level	C1 Inhibitor Function	C1 Inhibitor Level	C1q Level
Hereditary angioedema				
Type I	Low	Low	Low	Normal
Type II	Low	Low	Normal	Normal
Acquired angioedema	Low	Low	Normal or low	Low
ACE-inhibitor-associated angioedema	Normal	Normal	Normal	Normal

* Adapted from Banerji et al.²¹ ACE denotes angiotensin-converting enzyme, and C1q a protein that is part of the classic complement pathway.

converting-enzyme (ACE) inhibitors. ACE inhibitors are thought to cause angioedema through an effect on the kallikrein-kinin system that reduces the catabolism of bradykinin and increases its activity, leading to the inflammatory effect seen in angioedema.

A recent change in guidelines recommends that patients carry on-demand treatment for hereditary angioedema at all times and consider short-term prophylaxis before undergoing procedures that can induce an attack.²² The revised guidelines recommend consideration of on-demand treatment (with self-dosing of a C1 esterase inhibitor) for all attacks, treatment of attacks affecting the upper airway, early treatment, and initiation of treatment with a C1 inhibitor — either ecallantide (a kallikrein inhibitor) or icatibant (a bradykinin receptor antagonist).²² Ecallantide should be administered subcutaneously by a health care professional in three 10-mg (1-ml) injections; if the attack persists, an additional dose of 30 mg may be administered within a 24-hour period. The recommended dose for icatibant is 30 mg injected subcutaneously in the abdominal area; additional doses may be administered at intervals of at least 6 hours if the response is inadequate or if symptoms recur, but no more than 3 doses may be administered in any 24-hour period. The consensus guidelines recommend screening children from families with a history of hereditary angioedema and all offspring of an affected patient.²² Because the diagnosis is often delayed,^{23,24} a low threshold for screening in the emergency department is recommended, particularly in patients with a family history of angioedema.²⁵

In patients who present to the emergency department with angioedema, the likelihood of the

need for intubation or tracheostomy is increased if the anterior tongue, base of the tongue, or larynx is involved and if drooling or stridor occurred within 4 hours after symptom onset.^{26,27} In such cases, fiberoptic intubation is the favored approach.^{28,29}

BILATERAL PARESIS AND PARALYSIS OF THE VOCAL CORDS

In paresis and paralysis of the vocal cords, the vocal cords fail to adequately abduct. Bilateral vocal-cord paresis and paralysis can result from tumor infiltration of the glottic larynx or both recurrent laryngeal nerves, prolonged intubation or placement of a nasogastric tube, and infectious and pathologic conditions affecting the brain stem. In some instances, vocal-cord paralysis can also result from complications during thoracic and anterior neck surgery. Patients can often abide a narrowed airway for some time, but further diminution of the airway caliber, with inflammatory insults such as a viral infection, may lead to airway compromise. For this reason, a tracheostomy should be considered while the assessment for recovery is pending. If recovery is not achieved, surgical procedures can be used to improve the airway (leading to tracheostomy decannulation), but these interventions, including arytenoidectomy, with or without arytenoid abduction, often affect voice quality.

SUBGLOTTIC AND GLOTTIC STENOSIS

Cases of subglottic and glottic stenosis are rare and challenging; they most often result from prolonged or traumatic intubation but may also be congenital or idiopathic. Patients with granulomatous polyangiitis and relapsing polychondritis may also present with subglottic steno-

sis.^{30,31} Mucosal inflammation and localized fibrosis are the main pathophysiological features of the disease. Diagnosis is confirmed by means of laryngoscopy and bronchoscopy. Standard treatment involves dilation of the airway, commonly performed with the adjunctive use of lasers to radially incise the stenosis or of pressure-controlled balloons to dilate the stenosis. Some surgeons consider injection of glucocorticoids or the topical application of mitomycin. A sample dose of triamcinolone acetonide (40 mg per milliliter) should be injected submucosally in a circumferential pattern with the use of a 25-gauge butterfly needle controlled with micro-laryngoscopic alligator forceps. Usually no more than 0.1 ml can be applied per quadrant. Mitomycin should be applied at the site of stenosis for 2 to 4 minutes with two lightly soaked cotton pledgets at a dose of 0.4 mg per milliliter. Neither the surgical technique used nor the grade of stenosis alters surgical intervals; however, mitomycin application is associated with extension of the periods between subsequent endoscopic treatments.³² Although in-office glucocorticoid injections have been studied, long-term outcomes have not yet been reported.^{33,34}

An international, prospective, pragmatic trial protocol published in 2018 compares three standard surgical approaches to idiopathic subglottic stenosis at multiple international institutions.³⁵ The primary end point is the time to recurrent operative procedure. Patients are being followed for up to 36 months, and data on a number of secondary end points related to quality of life are being collected. In patients with recalcitrant stenosis who are unlikely to have a response to conservative surgical dilation, definitive cricotracheal resection and reconstruction of the stenotic segment can be considered when there is no active airway inflammation (e.g., active subglottic granulomatosis polyangiitis).

NEOPLASMS INTRINSIC OR EXTRINSIC TO THE UPPER AERODIGESTIVE TRACT

Neoplasms can lead to acute upper airway obstruction. The most common intrinsic neoplasms associated with airway obstruction are glottic and supraglottic cancers, most often squamous-cell carcinomas. They are often caused by smoking and alcohol abuse, which act synergistically. Patients who require a tracheostomy before management of their laryngeal cancer have worse

outcomes than those who have the procedure after management.^{36,37} Benign tumors of the thyroid, particularly when they extend substernally, can lead to substantial subglottic and tracheal deviation and compression. In rare instances, thyroid cancers invade the airway, leading to both an extrinsic compression effect and an intrinsic narrowing related to intraluminal disease.

PSYCHOGENIC UPPER AIRWAY OBSTRUCTION

Psychogenic presentations of upper airway obstruction are rare. Most patients have paradoxical vocal-fold motion disorder, a conversion disorder in which the vocal cords adduct on inspiration and abduct on expiration, leading to inspiratory stridor. Patient presentation is similar to that of patients with epiglottitis.³⁸ The condition can also be induced by exercise and is associated with laryngopharyngeal reflux, allergic and sinus problems, and obstructive sleep apnea.^{38,39} The diagnosis is often confused with severe asthma. It is made by means of fiberoptic laryngoscopy when no other abnormalities of the upper aerodigestive tract are noted. Respiratory retraining and laryngeal control therapy are the mainstays of treatment, which is performed by a speech–language pathologist. The use of counseling and psychiatric assessment is in decline.

INHALATION INJURY

Approximately 15% of patients with burn injuries have inhalation injury, which, along with the total body-surface area of the burn, is an important independent predictor of mortality.^{40,41} In patients with recent injury from smoke inhalation, intubation should be initiated if there are extensive facial or neck burns, a decreased level of consciousness, or airway obstruction. In those with clinical signs and symptoms of inhalation injury, such as dysphonia, dysphagia, singed nasal hairs, sooty sputum, stridor, cyanosis, or neurologic symptoms, endoscopic assessment by an otolaryngologist is warranted.⁴² Intubation is also called for if erythema, edema, or sooty exudate is detected. These patients are at a substantial risk for decline in the first 24 hours after inhalation injury.⁴²

Patients with inhalation injuries tend to have long-term laryngological complications. Consequent to local inflammation and edema, patients may have granulation tissue, scarring, webbing, vocal-cord immobility, stenosis, and laryngeal

hyperfunction. The subglottis is particularly sensitive, and patients with inhalation injuries have an earlier onset and more severe stenosis than those whose subglottic stenosis is due to other causes.^{43,44} The earlier onset and greater severity associated with subglottic stenosis in these patients have led to calls for earlier use of tracheostomy.⁴⁵

Traumatic Airway Injury

Traumatic injury to the airway can be open (penetrating) or closed (blunt). A study examining the

Emerging Technologies and Simulation

U.S. Nationwide Emergency Department Sample from 2009 through 2011 showed that the majority of injuries (91.4%) were closed. The majority of patients had multiple injuries (76.2%), were admitted to the hospital (71.9%), and had an overall mortality of 3.8%. The rate of fiberoptic examination was poor (1.9%), although this approach probably had a low rate of severe injury given the low frequency of both intubation (2.6%) and tracheostomy (0.1%). In a cohort study of changes in airway management from 1995 through 2011, researchers in Alberta, Canada reported that increased use of computed tomography in the later years of the study was associated with a higher rate of intubation (27% in 1995 vs. 64% in 2011). It should be noted that patients in this cohort had more severe injury than those in most other studies that have examined the management of difficult airways associated with trauma and that 56% of the patients in this cohort underwent surgical intervention.

Classic signs and symptoms of a major injury after open or closed neck trauma include a sucking or bubbling neck wound, pharyngeal bleeding, a large or expanding hematoma, subcutaneous emphysema, dysphagia, dysphonia, and stridor. Vascular, pharyngeal, and laryngotracheal injuries are managed simultaneously and require the expertise of a multidisciplinary team, preferably at a specialized trauma center.

Algorithm for Management of a Difficult Airway

Guidelines for the management of unanticipated difficult intubation from the Difficult Airway Society⁴⁶ include a flowchart that escalates from laryngoscopy to supraglottic insertion of an air source and has a large opening through which

Several recent advances in airway instrumentation have provided new tools to help manage the challenging airway. Nasal high-flow oxygen therapy (Optiflow, Fisher and Paykel Healthcare) is an alternative to the standard face mask or nasal prongs used in patients who are in airway distress. This approach provides warmed, humidified oxygen at high flow rate (1 to 60 liters per minute, with 60 liters being the maximum), with a linear relationship between airflow and airway pressures as well as lung impedance.⁴⁷ Unlike a face mask or nasal prongs, nasal high-flow therapy also provides some positive end-expiratory pressure that may decrease the work of breathing, especially in patients with acute upper airway obstruction. This therapy has also been described as being more comfortable than a face mask or nasal prongs. However, since very few studies have evaluated the specific indications for nasal high-flow therapy, recommendations regarding its use remain unclear.

In patients requiring a definitive airway for mechanical ventilation, endotracheal intubation should be considered, barring any contraindications. On the basis of a recent systematic review and meta-analysis, the first-pass success rate for emergency airway intubation is 84.1% (95% confidence interval, 80.1 to 87.4%).⁴⁸ However, over the past decade, the use of video intubation technology has increased, and there has been a subsequent decrease in the use of direct laryngoscopy.⁵³ Video laryngoscopes (e.g., the GlideScope [Verathon] and C-MAC [Storz]) provide indirect visualization of the glottis, with a camera and lighting placed at the tip of the scope to facilitate

- of chronic angioedema. *Allergy Asthma Proc* 2009;30:11-6.
22. Maurer M, Magerl M, Ansotegui I, et al. The international WAO/EAACI guideline for the management of hereditary angioedema — the 2017 revision and update. *Allergy* 2018;73:1575-96.
23. Bork K, Davis-Lorton M. Overview of hereditary angioedema caused by C1-inhibitor deficiency: assessment and clinical management. *Eur Ann Allergy Clin Immunol* 2013;45:7-16.
24. Lunn ML, Santos CB, Craig TJ. Is there a need for clinical guidelines in the United States for the diagnosis of hereditary angioedema and the screening of family members of affected patients? *Ann Allergy Asthma Immunol* 2010;104:211-4.
25. Hirose T, Kimbara F, Shinozaki M, et al. Screening for hereditary angioedema (HAE) at 13 emergency centers in Osaka, Japan: a prospective observational study. *Medicine (Baltimore)* 2017;96(6):e6109.
26. McCormick M, Folbe AJ, Lin HS, Hooten J, Yoo GH, Krouse JH. Site involvement as a predictor of airway intervention in angioedema. *Laryngoscope* 2011;121:262-6.
27. Kieu MC, Bangiyev JN, Thottam PJ, Levy PD. Predictors of airway intervention in angiotensin-converting enzyme inhibitor-induced angioedema. *Otolaryngol Head Neck Surg* 2015;153:544-50.
28. Driver BE, McGill JW. Emergency department airway management of severe angioedema: a video review of 45 intubations. *Ann Emerg Med* 2017;69:635-9.
29. LoVerde D, Files DC, Krishnaswamy G. Angioedema. *Crit Care Med* 2017;45:725-35.
30. Ugan Y, Doğru A, Aynalı G, Şahin M, Tunç SE. A clinical threat in patients with granulomatosis polyangiitis in remission: subglottic stenosis. *Eur J Rheumatol* 2018;5:69-71.
31. Childs LF, Rickert S, Wengerman OC, Lebovics R, Blitzer A. Laryngeal manifestations of relapsing polychondritis and a novel treatment option. *J Voice* 2012;26:587-9.
32. Feinstein AJ, Goel A, Raghavan G, et al. Endoscopic management of subglottic stenosis. *JAMA Otolaryngol Head Neck Surg* 2017;143:500-5.
33. Hoffman MR, Coughlin AR, Dailey SH. Serial office-based steroid injections for treatment of idiopathic subglottic stenosis. *Laryngoscope* 2017;127:2475-81.
34. Franco RA Jr, Husain I, Reder L, Padle P. Awake serial intralesional steroid injections without surgery as a novel targeted treatment for idiopathic subglottic stenosis. *Laryngoscope* 2018;128:610-7.
35. Gelbard A, Shyr Y, Berry L, et al. Treatment options in idiopathic subglottic stenosis: protocol for a prospective international multicentre pragmatic trial. *BMJ Open* 2018;8(4):e022243.
36. Semdaie D, Haroun F, Casiraghi O, et al. Laser debulking or tracheotomy in airway management prior to total laryngectomy for T4a laryngeal cancer. *Eur Arch Otorhinolaryngol* 2018;275:1869-75.
37. Du E, Smith RV, Ow TJ, Tassler AB, Schiff BA. Tumor debulking in the management of laryngeal cancer airway obstruction. *Otolaryngol Head Neck Surg* 2016;155:805-7.
38. Matrka L. Paradoxical vocal fold movement disorder. *Otolaryngol Clin North Am* 2014;47:135-46.
39. Chiang T, Marciniow AM, deSilva BW, Ence BN, Lindsey SE, Forrest LA. Exercise-induced paradoxical vocal fold motion disorder: diagnosis and management. *Laryngoscope* 2013;123:727-31.
40. Hogg G, Goswamy J, Khwaja S, Khwaja N. Laryngeal trauma following an inhalation injury: a review and case report. *J Voice* 2017;31(3):388.e27-388.e31.
41. Osler T, Glance LG, Hosmer DW. Simplified estimates of the probability of death after burn injuries: extending and updating the Baux score. *J Trauma* 2010;68:690-7.
42. Kot Baixauli P, Morales Sarabia JE, Rovira Soriano L, De Andrés Ibáñez J. Proposal for an algorithm for the management of the patient's airway after smoke inhalation. *Rev Esp Anestesiol Reanim* 2018;65:170-2.
43. Wan J, Zhang G, Qiu Y, Wen C, Fu T. Heat dissipation by blood circulation and airway tissue heat absorption in a canine model of inhalation thermal injury. *Burns* 2016;42:548-55.
44. Casper JK, Clark WR, Kelley RT, Colton RH. Laryngeal and phonatory status after burn/inhalation injury: a long term follow-up study. *J Burn Care Rehabil* 2002;23:235-43.
45. Reid A, Ha JF. Inhalation injury and the larynx: a review. *Burns* 2019 45:1266-74.
46. Sethi RKV, Khatib D, Kligerman M, Kozin ED, Gray ST, Naunheim MR. Laryngeal fracture presentation and management in United States emergency rooms. *Laryngoscope* 2019;129:2341-6.
47. Randall DR, Rudmik L, Ball CG, Bosch JD. Airway management changes associated with rising radiologic incidence of external laryngotracheal injury. *Can J Surg* 2018;61:121-7.
48. Jain U, McCunn M, Smith CE, Pittet JF. Management of the traumatized airway. *Anesthesiology* 2016;124:199-206.
49. Frerk C, Mitchell VS, McNarry AF, et al. Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. *Br J Anaesth* 2015;115:827-48.
50. Parke RL, Bloch A, McGuinness SP. Effect of very-high-flow nasal therapy on airway pressure and end-expiratory lung impedance in healthy volunteers. *Respir Care* 2015;60:1397-403.
51. Spoletini G, Alotaibi M, Blasi F, Hill NS. Heated humidified high-flow nasal oxygen in adults: mechanisms of action and clinical implications. *Chest* 2015;148:253-61.
52. Park L, Zeng I, Brainard A. Systematic review and meta-analysis of first-pass success rates in emergency department intubation: creating a benchmark for emergency airway care. *Emerg Med Australas* 2017;29:40-7.
53. Lee JK, Kang H, Choi HJ. Changes in the first-pass success rate with the GlideScope video laryngoscope and direct laryngoscope: a ten-year observational study in two academic emergency departments. *Clin Exp Emerg Med* 2016;3:213-8.
54. Yousef GT, Abdalgalil DA, Ibrahim TH. Orotracheal intubation of morbidly obese patients, comparison of GlideScope video laryngoscope and the LMA CTrach with direct laryngoscopy. *Anesth Essays Res* 2012;6:174-9.
55. Belze O, Lepage E, Bazin Y, et al. GlideScope versus Airtraq DL for double-lumen tracheal tube insertion in patients with a predicted or known difficult airway: a randomised study. *Eur J Anaesthesiol* 2017;34:456-63.
56. Sakles JC, Mosier J, Patanwala AE, Dicken J. Improvement in GlideScope Video Laryngoscopy performance over a seven-year period in an academic emergency department. *Intern Emerg Med* 2014;9:789-94.
57. Cook TM, Boniface NJ, Seller C, et al. Universal videolaryngoscopy: a structured approach to conversion to videolaryngoscopy for all intubations in an anaesthetic and intensive care department. *Br J Anaesth* 2018;120:173-80.
58. Cook TM, Kelly FE. A national survey of videolaryngoscopy in the United Kingdom. *Br J Anaesth* 2017;118:593-600.
59. Wu TS, Tseng WC, Lai HC, Huang YH, Wu ZF. Sugammadex and laryngospasm. *J Clin Anesth* 2019;56:52.
60. Greenaway S, Shah S, Dancy M. Sugammadex and laryngospasm. *Anaesthesia* 2017;72:412-3.
61. McGuire B, Dalton AJ. Did sugammadex cause, or reveal, laryngospasm? A reply. *Anaesthesia* 2016;71:1112-3.
62. Cole RR, Aguilar EA III. Cricothyroidotomy versus tracheotomy: an otolaryngologist's perspective. *Laryngoscope* 1988;98:131-5.
63. Terlinden N, Van Boven M, Hamoir M, Schmitz S. An innovative approach to tracheotomy in patients with major obstruction of the upper airway. *Am J Otolaryngol* 2014;35:445-8.
64. Soleimanpour H, Shams Vahdati S, Mahmoodpour A, et al. Modified cricothyroidotomy in skill laboratory. *J Cardiovasc Thorac Res* 2012;4:73-6.

Copyright © 2019 Massachusetts Medical Society.