Introduction

Otolaryngologists are frequently asked to evaluate patients who are endotracheally intubated or have voice or airway complaints after being intubated. In many situations this occurs when tracheotomy is deemed necessary for prolonged airway management. This may be after the patient has been intubated for one or more weeks. Any physician being asked to evaluate a patient who is or has been intubated should be aware of both the acute and long term complications that may be encountered, as well as understand their diagnosis, prevention and management.

Background

Endotracheal intubation is recorded as early as 1000 AD. In 1878, Macewen first described orotracheal intubation for the administration of anesthesia using a tube fashioned of brass. It was not until 1889 that Annandale designed a tube constructed of rubber. Later, Guedel and Waters added an inflatable cuff. In 1964, the first tube constructed of polyvinylchloride (PVC) with an integrated inflatable cuff was marketed. High-volume, low-pressure cuffs were introduced in 1970. Polyvinylchloride is superior in that it softens slightly at body temperature as compared to room temperature, while rubber becomes somewhat more rigid and has a greater propensity for inducing mucosal injury. Tubes constructed of PVC with low-pressure cuffs are the most commonly used today.

The indications for intubation are varied. Generally, it is indicated for the relief of obstruction, for ventilatory assistance, to aid in respiratory toilet and to prevent aspiration. It has supplanted tracheotomy as the method of choice for short-term management of the airway. However, in cases where the need for airway support is expected to surpass one to two weeks, tracheotomy should be considered early. Notable exceptions to this are in neonates and burn victims, in whom the duration of intubation is often extended.
Evaluation

The evaluation of a patient with suspected intubation injury begins with a thorough history. The reason for intubation and co-existing illnesses should be noted. The date of initial intubation, failed attempts at extubation and total length of intubation are very important. The mode of intubation should be noted (i.e. fiberoptic, blind or with direct laryngoscopy), as should any details regarding difficulty in securing the airway.

Examination of the intubated patient should begin by noticing the level of consciousness. Is there excessive movement due to inadequate sedation? Is ventilator movement transmitted to the tube? The route of intubation should be appreciated (oro- or naso-tracheal). The size of the tube and the presence of a nasogastric tube are important. In patients who have been extubated this information may not be readily available, but should be sought by careful review of the medical record.

Examination of the larynx is often challenging in the intubated patient. Flexible fiberoptic laryngoscopy is often attempted but is inadequate and much information may be missed, specifically in the posterior glottis where injury is most likely to occur. In the extubated patient who is able to cooperate with examination, flexible fiberoptic laryngoscopy may provide valuable information concerning motion of the true vocal folds. Videostrobolaryngoscopy (VSL) is also useful in this regard if the patient is able to tolerate the procedure. Alessi et al. found VSL to be quite valuable in the documentation of intubation trauma and were successful in performing the examination at the bedside in critically ill patients.

Direct laryngoscopy is the method of choice for complete evaluation of intubation injuries. Benjamin advocates direct laryngoscopy in adult patients after 7 days of intubation, in children after 1-2 weeks and in infants after failed attempts at extubation. He feels that after complete examination, a rational decision can be made regarding the safety of continued intubation or whether to proceed with tracheotomy. Others feel that tracheotomy is indicated at 7 days, with endoscopic evaluation reserved for those in whom decannulation is unsuccessful. Benjamin argues that with this approach, there is the possibility of performing tracheotomy when the laryngeal injury is mild and could have been avoided. During direct laryngoscopy, the endotracheal tube may be removed allowing complete examination of the posterior glottis and subglottic region. Examination with rigid fiberoptic telescopes, both 0° and 30°, is indispensable in identifying injury within the posterior glottis. Palpation of the arytenoid cartilages may reveal fixation of the cricoarytenoid joint. Many injuries may be treated during the diagnostic procedure.

Lindholm proposed a classification system based upon the findings at direct laryngoscopy. Grade I injury is characterized by edema and hyperemia without macroscopic ulceration. Grade II injury has continuous superficial ulceration of less than 1/3 of the circumference of the airway. Grade III injury exhibits continuous deep ulceration of less than 1/3 of the circumference of the airway, or continuous superficial ulceration of over 1/3 of the circumference of the airway. Grade IV injury is that with deep ulceration of the mucus membrane with exposed cartilage, or continuous deep ulceration of more than 1/3 of the circumference of the airway. This system is not universally used, but may aid in describing the severity of injury between clinicians.
Acute Complications of Intubation

While the prolonged presence of a translaryngeal tube for airway protection may result in characteristic changes, direct laryngoscopy and tracheal intubation may have acute complications as well. A myriad of non-specific and unfortunate complications have been reported after attempted intubation of patients with difficult anatomy or performed by inexperienced clinicians. These include reports of a swallowed endotracheal tube after inadvertent esophageal intubation, gastric perforation resulting from unrecognized esophageal intubation and ventilation, and the aspiration of the bulb from a laryngoscope. Difficult intubation may result in laceration to the mucosa of the oropharynx, hypopharynx, larynx, trachea or esophagus, with the pyriform sinus and region posterior to the cricopharyngeus muscle being the most at risk. Perforation of the mucosa with passage of the tube into the soft tissues of the neck may occur, leading to subcutaneous emphysema and soft-tissue infection.

The endolarynx is especially vulnerable as a result of its delicate anatomy. The true vocal folds may be damaged by the use of excessive force with resultant hematoma formation, laceration or avulsion. Such injuries may lead to scarring and the formation of granulomas in atypical locations. The management of these injuries requires careful evaluation with individualized treatment.

Dislocation of the arytenoid cartilage is also a rare complication of intubation. Sataloff reports 26 cases of which 50% were the result of intubation. The most common symptom is hoarseness, followed by breathlessness, dysphagia, sore throat, dyspnea, vocal fatigue and the loss of voice control. The diagnosis is may usually be made by the detection of decreased vocal fold mobility and gross arytenoid dislocation at indirect laryngoscopy or flexible fiberoptic laryngoscopy. The most consistent physical examination finding is an unequal level between the vocal folds. This is most easily recognized with VSL, an integral part of the evaluation. Laryngeal electromyography (EMG) and computed tomography (CT) may aid in diagnosis.

The treatment of choice for arytenoid dislocation is closed reduction. This is best performed at the time of diagnosis, as early treatment improves the likelihood of the restoration of a normal voice. Prolonged dislocation may lead to ankylosis of the cricoarytenoid joint with the inability to reduce the dislocation. However, there is no clear time period as to when this occurs and reductions as remote as one year from the initial injury have resulted in dramatic improvement in voice quality. Sataloff recommends local anesthesia and sedation for the procedure as this allows assessment of the voice and vocal fold mobility intraoperatively. Some patients experience spontaneous reduction of the dislocation and require no intervention. In cases where closed reduction has failed, medialization techniques, open reduction and arytenoidectomy have all been applied.

Sequelae of Prolonged Intubation

The presence of an endotracheal tube in the larynx causes characteristic pathology. The incidence of laryngeal complications after prolonged intubation is between 4% and 13%. Benjamin has described several stages of injury and the resultant chronic laryngeal changes that may be seen after prolonged intubation. The earliest of these changes are non-specific hyperemia
and edema due to mucosal irritation. Edema is often marked in the mucosa of the laryngeal ventricle, causing a prolapse or protrusion of the mucosa. Edema within the submucosa in the subglottis at the level of the cricoid may increase slowly leading to delayed airway obstruction hours after the removal of the endotracheal tube. Occasionally, true vocal cord edema persists long after extubation as Reinke’s edema with vocal dysfunction.

As the tube remains in place, ulceration occurs with varying degrees of granulation tissue formation. The most susceptible site to irritation from the endotracheal tube is in the posterior larynx, especially the mucosa overlying the vocal processes of the arytenoids. This granulation tissue formation begins within 48 hours. Benjamin describes "tongues of granulation” extending from the vocal processes bilaterally anterior to the endotracheal tube. They may become quite large and prolapse into the glottis at extubation causing airway obstruction with the need for immediate re-intubation. Benjamin states that if the patient undergoes tracheotomy or extubation is accomplished at this stage, removal of granulation tissue is unnecessary as it will likely resolve. On the other hand, Deeb et al. advocate removal of obstructing granulation tissue at the time of tracheotomy in patients who fail extubation in order to prevent long term complications.

In some instances, the granulation tissue resolves incompletely or matures into chronic laryngeal scarring. With incomplete healing and persistent perichondritis, an intubation granuloma may form. This is a localized, rounded mass that protrudes from the site of ulceration, most commonly at the vocal process and medial surface of the arytenoid. At laryngoscopy it is noted as a globular, yellow-red pedunculated mass. Granulomas arising in other sites are often the result of a mucosal laceration. Patients with intubation granuloma present weeks to months after extubation with voice changes, globus or rarely, airway obstructive symptoms. Benjamin states that the treatment of choice is removal with the carbon dioxide laser. Inadequate removal may result in recurrence, while overly aggressive removal may result in exposure of perichondrium or cartilage. Removal with microsurgical techniques is discouraged as it results in bleeding which may obscure the attachment of the granuloma making accurate resection difficult.

Scarring may occur on the free edge of the vocal fold, most commonly near the vocal process of the arytenoid. This may mature into a healed fibrous nodule covered with intact mucosa. Removal of the nodule rarely results in improvement of the voice.

Upon removal of the endotracheal tube, large tongues of granulation tissue may fall towards the midline contacting one another. Adherence and healing in this configuration may result in an interarytenoid adhesion. The vocal folds may be tethered to one another limiting abduction and mimicking vocal fold paralysis. The glottic airway is partially obstructed as a result of the poor abduction. The adhesion may be lysed in the acute stages before it becomes fibrotic. After fibrosis occurs, it can be difficult to distinguish the adhesion from posterior glottic stenosis or bulky scar tissue within the posterior glottis. The formation of an interarytenoid adhesion is more likely in patients with vocal cord paralysis or patients with decreased movement of the vocal cords as a result of a depressed level of consciousness. The adhesion is easily divided with microsurgical scissors or the carbon dioxide laser with excellent results.

As stated previously, the site most susceptible to injury from prolonged intubation is the posterior glottis. Pressure necrosis and ulceration occur over the medial surface of the arytenoid
and within the cricoid in the area of greatest tube diameter. Superficial ulceration is an early finding, occurring 4-6 hours after intubation. As the irritation from the endotracheal tube persists, the mucosal ulcerations deepen and are invaded by bacteria from the respiratory tree. At 48 hours, the inflammatory reaction has reached the perichondrium. Prolonged intubation results in progression of these changes. If the tube is removed early when the ulceration is superficial, healing and re-mucosalization are rapid. Deep ulceration will frequently be appreciated as "ulcerated troughs". These can be seen only after removal of the endotracheal tube and appear as wide, deep erosions through the perichondrium and into the cartilage on the medial aspect of the arytenoid and cricoid cartilages. The cricoarytenoid joint is often exposed and may become inflamed leading to chronic fibrosis and ankylosis with accompanying dysphonia. Weeks to months after extubation, these ulcerated troughs may heal and be noticed as "healed furrows". A cursory examination may miss these findings. Direct laryngoscopy and examination with a 30° telescope is usually necessary to adequately assess the posterior glottis. Intervention at this stage is difficult and there is no effective treatment. Ulceration crossing the midline of the posterior glottis with no median strip of intact mucosa signifies a high risk for the formation of posterior glottic stenosis upon scar maturation.

Posterior glottic stenosis is a common occurrence after prolonged intubation in both adults and children. The posterior glottis is the area most affected by translaryngeal intubation. Deep ulceration commonly occurs with chondritis of the arytenoid and cricoid cartilages. After intubation, these ulcerations heal with marked scarring. In severe cases, this scarring matures into a thick fibrous band between the arytenoid cartilages. Abduction of the true vocal cords may be limited and may be misdiagnosed as bilateral abductor paralysis. Such findings have prompted the label pseudolaryngeal paralysis. These patients usually have airway complaints ranging from dyspnea on exertion to near complete obstruction. The voice often is near normal.

Posterior glottic stenosis is often labeled laryngeal stenosis. Benjamin states that this term is imprecise, without regard for the nature, timing and degree of the injury. The diagnosis of posterior glottic stenosis should be suspected in patients with a likely history, and physical examination that reveals limited abduction of the vocal folds on inspiration with incomplete glottic closure on phonation. Definitive diagnosis requires direct laryngoscopy with microscopic examination of the posterior glottis using 0° and 30° telescopes. The transverse fibrotic scar appears as a firm, thick web in the posterior glottis between the arytenoid cartilages. The anterior margin may be sharp, localized or blunt. The web may extend from above the interarytenoid area through the level of the glottis and into the subglottic region. If during the assessment of an acute injury a central, intact strip of mucosa is identified, posterior glottic stenosis is unlikely to occur. Subglottic stenosis, which is more easily recognized, may be coexistent with posterior glottic stenosis. Treatment of the subglottic stenosis is unlikely to be successful without addressing the scarring in the posterior glottis.

Posterior glottic stenosis may be treated endoscopically or through open procedures. The scarring may divided in the midline at endoscopy using a number 11 scalpel blade on a long handle or the carbon dioxide laser. With the knife, the web is divided superiorly from the subglottic region, applying enough pressure that the posterior lamina of the cricoid cartilage is reached. Quite frequently the tissues fall apart leaving a "V" shaped defect in the posterior commissure. The patient may experience a limited but dramatic relief of the airway obstruction.
Unfortunately, recurrence of the stenosis is common and repeated division may be necessary. Montgomery advocates laryngofissure with excision of the scar in the posterior glottis and coverage with a superiorly based mucosal flap. In severe cases, a cartilage or bone spreader graft in the posterior lamina of the cricoid cartilage may be necessary to attain any relief of the stenosis. Cases with severe stenosis, fibrosis and fixation of the cricoarytenoid joints are exceedingly difficult to manage and often refractory to treatment.

Prolonged intubation is the most common cause of subglottic stenosis in both adults and children. By definition, subglottic stenosis is a narrowing of the subglottic space above the inferior margin of the cricoid cartilage and below the level of the glottis. The size of the subglottis is variable with age, thus absolute values for subglottic size are difficult to define. A subglottic airway less than 4 mm in diameter in a full-term infant or less than 3 mm in a premature infant will likely cause symptomatic obstruction and should be considered abnormal. The loose tissue covering the cricoid cartilage makes this area specifically vulnerable to mechanical trauma during intubation and edematous swelling may cause critical airway obstruction. Patients with abnormal cricoid cartilages and congenital subglottic stenoses are more likely to have subglottic complications from intubation. The entire spectrum of diagnosis, evaluation and treatment of subglottic stenosis was the subject of a separate recent Grand Rounds presentation and will not be reiterated here. It is mentioned only to remind the reader that it is a frequent complication and should be considered in any post-intubation patient with airway complaints.

Complete stenosis of the larynx may occur within the glottis or subglottis. While prolonged intubation is often the inciting event, repeated attempts at dilation or injudicious attempts at laser excision of laryngeal scarring worsen the injury. Intervention is usually unsuccessful.

True vocal cord paralysis may occur as a result of endotracheal intubation. The paralysis is most commonly unilateral, but bilateral paralysis with airway obstruction has been reported. Brandwein et al. examined the course of the anterior branch of the recurrent laryngeal nerve and discovered it to be vulnerable to compression between the inflated cuff of the endotracheal tube, the lateral projection of the abducted arytenoid, and the thyroid cartilage. Injury to the recurrent laryngeal nerve most commonly results in the cord lying in the paramedian position, although some variability in cord position has been noted. Other etiologies of limitation of vocal fold mobility i.e. posterior glottic stenosis, should be excluded as they may mimic vocal fold paralysis. Laryngeal EMG may aid in predicting recovery which should be expected within 6 months.

Ductal retention cysts may result from obstruction of the submucous glands by an in-dwelling endotracheal tube. They are most common in infants, occurring posteriorly in the subglottis. Small cysts may be incidental findings, while larger cysts can cause airway obstruction. Tracheotomy may be necessary and laser excision is the treatment of choice.

Factors Contributing to Intubation Trauma

Multiple risk factors for developing complications after intubation have been identified. While absolute correlations between coexistent illnesses and particular types of injury have not been
made, certain situations should raise the suspicion for intubation trauma and the sequelae of prolonged intubation. Physical trauma incurred during the act of intubation is usually the result of abnormal anatomy and difficult laryngoscopy, multiple intubations or lack of skill of the operator. Abnormal larynges are more prone to injury, as in acute laryngotracheobronchitis where the inflammatory response already present within the larynx makes the mucosa more susceptible to pressure necrosis.

Several characteristics of the tube itself may predispose to greater injury. Diameter of the tube is important in determining the extent of injury. Some authors suggest that the upper limit of inside diameter of the endotracheal tube be 8.0 mm in males and 7.0 mm in females. More appropriately however, the size tube used should be individualized. Santos et al. demonstrated that patients who had prolonged intubation with a tube greater than 7.5 mm had an increased incidence of vocal fold immobility after extubation and thus recommend that prolonged intubation with a tube larger than this be avoided. Infants and children less than 8 years old should have an uncuffed tube that allows for an air leak with 20 cm of water positive pressure. Even with an appropriately sized endotracheal tube, excessive motion of the tube may induce repeated trauma. This may occur as a result of patient movement, motion transmitted from the ventilator or manipulation during suctioning. Fortunately, high-volume, low-pressure cuffs have replaced the high-pressure cuffs used in the past. The cuff may still induce excessive trauma if it is inflated too high within the larynx. Rubber tubes, which were commonly used in the past, are excessively irritating and are rarely used today.

Gastroesophageal reflux and aspiration are common in critically ill patients. This repetitive bathing of the laryngeal structures with gastric acid causes a chemical irritation that adds to the local injury from the endotracheal tube. It may be beneficial to use H2-blockers to minimize reflux in critically ill and intubated patients.

The presence of a nasogastric tube increases the likelihood of reflux. Nasogastric tubes may also cause irritation and ulceration in the post-cricoid region and have been reported to cause cricoid chondritis. Friedman et al. noticed several patients with cricoid chondritis secondary to nasogastric tubes. They found localized odynophagia and pain radiating to the ear to be attributable to mucosal ulceration. These patients had the nasogastric tube for longer than three days, often positioned in the midline. They thus recommend that patients with nasogastric tubes who experience pain, have the tube for longer than 3 days or who are unconscious receive anteroposterior neck radiographs. If the nasogastric tube is positioned in the midline, it should be re-positioned laterally. If symptoms persist, laryngoscopy should be performed to rule out cricoid chondritis. Early chondritis should be treated with parenteral antibiotics. Injury due to nasogastric tubes may occur with or without endotracheal tubes, and the concurrent presence of both may magnify the insult.

Chronic disease may predispose the patient to intubation trauma as well. Medical conditions associated with decreased tissue perfusion increase the likelihood of tissue necrosis and ulceration. These conditions include congestive heart failure, liver failure, hypoxemia and anemia. Gaynor and Greenberg noted a very high incidence of severe complications in patients with insulin dependent diabetes mellitus and they recommend early tracheotomy in these patients if the length of intubation is expected to exceed 4 days. Immunosuppressed patients are more
prone to bacterial infection of mucosal ulcerations and should be monitored closely for the development of chronic chondritis and its sequelae. In patients with any of these co-existing illnesses, early tracheotomy should be considered.

**Controversies**

Several areas surrounding intubation trauma to the larynx have given rise to differing opinions. The best method for evaluating laryngeal injury is still unclear. While recent articles such as that by Alessi et al. propose bedside VSL for evaluation of the larynx, Benjamin feels that direct laryngoscopy is mandatory for complete examination. Most authors agree that flexible fiberoptic laryngoscopy is totally inadequate for the examination of the intubated larynx.

The role and timing of tracheotomy is still the subject of some debate. Most agree that patients intubated for 7 days, thought should be given to tracheotomy if extubation is not imminent, and lengths of intubation greater than 10 days should be avoided. Deeb et al. advocate direct laryngoscopy for all patients who fail extubation. If significant injury is present, they feel early removal of granulation tissue and tracheotomy is indicated. Gaynor and Greenberg advocate tracheotomy in patients with diabetes mellitus when the length of intubation is expected to exceed 4 days. There are several exceptions to these guidelines. In burn victims the time to tracheotomy is extended, as the site may bathe the burn wounds with contaminated secretions. In infants the time to tracheotomy may be extended to several weeks and in neonates with good nursing, there is almost no length of intubation that is considered unsafe. The exact explanation for this is unknown, but the immaturity and pliability of the laryngeal cartilages may play a significant role in reducing pressure necrosis of the mucosa.

The selection of an endotracheal tube size is also important. While most laryngologists would agree that the smaller the tube the better, more specific guidelines are needed. Santos et al. found that patients intubated with tubes larger than 7.5 mm inner diameter had a greater incidence of delayed vocal fold mobility, with no cases occurring in patients intubated with size 7.5 or smaller tubes. They recommend avoiding 8.0 tubes in all patients. Most authors agree that women should not be intubated with tubes larger than 7.0 mm inner diameter.

The role of gastroesophageal reflux in exacerbating laryngeal injury is not clearly known. Furthermore, the best methods for preventing or minimizing its effects need to be further investigated. Clearly, the presence of a nasogastric tube increases the likelihood of reflux and worsening the laryngeal injury. All patients with a nasogastric tube should be placed on H₂-blocker therapy and this recommendation may be carried over to endotracheally intubated patients as well.

**Case Study**

A 56 year old woman presents to clinic 5 months after being hospitalized for acute exacerbation of congestive heart failure (CHF). She now complains of hoarseness and slowly increasing dyspnea on exertion. Her internist feels her CHF is under good control at this time. She states that she was in the intensive care unit for approximately 2 weeks, requiring ventilator support for much of this time. She is unsure of the details of her care during this period. Review of the
medical record reveals that she was intubated for 11 days. No record is found of the size tube used, the use of a nasogastric tube or difficulties with intubation. She was discharged 16 days after admission. She states that she was mildly hoarse at that time, but her symptoms have gradually increased.

Examination in clinic reveals no abnormalities of the ears, nose, oral cavity or oropharynx. At indirect laryngoscopy, the true vocal folds have decreased abduction bilaterally with moderate erythema. No other specific abnormalities are noted. These findings are confirmed at VSL.

The patient was scheduled for direct laryngoscopy. Examination with the endotracheal tube in place is unremarkable. With the endotracheal tube removed under apneic technique, a dense, fibrotic scar is noted extending between the arytenoid cartilages in the posterior glottis. The scar narrows the glottic airway and tethers the arytenoid cartilages to one another. The subglottis is normal. The scar is divided in the midline using the carbon dioxide laser. The arytenoid cartilages fall laterally after division.

On follow-up visit, the patient states that her shortness of breath has improved, as has her hoarseness, although not to pre-intubation levels. She is satisfied with the results. She returns for follow-up in 4 months, again with some increase in her symptoms. Examination reveals that once again abduction of the arytenoid cartilages is limited. After discussion with the patient, she desires repeat direct laryngoscopy and division of the scar band that is likely to have recurred.

Conclusion

Intubation injury to the larynx is relatively common and all types of injury have been reported. In patients intubated for prolonged periods certain types of injury can be expected. The physician asked to evaluate a patient for intubation injury should have a clear idea of the type injury that may be encountered as well as a thorough knowledge of the best methods of prevention and intervention. In many cases, the injury will resolve without incident, while in others the injury is irreversible. Frequently, the process can be corrected with good results if the proper treatment is instituted.

References


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