Manual of Otolaryngology

Diagnosis and Therapy

Second Edition

Marshall Strome, James H. Kelly, Marvin P. Fried
To our medical students and residents, whose intellectual curiosity and stimulation have earned them a place as special contributors to this text.

M. S., J. H. K., M. P. F.

To Deena, Scott, and Randy - no man could have hoped for more.

M. S.

To Alexander, Jamie, and Erin - with love.

J. H. K.

To Rita, who has been with me all the way.

M. P. F.
Preface

Our goal in the preparation of the second edition of the Manual of Otolaryngology was similar in many respects to that of the first edition: a concise, timely reference detailing the essentials of otolaryngology and head and neck surgery for students of the discipline. Pertinent new information, including artwork, has been incorporated and an entire new chapter on AIDS has been added. Unlike the contributors to the first edition, the primary contributors of this edition were, with one notable exception, either the editors or the full-time otolaryngology faculty of two Harvard institutions: the Brigham and Women's and Beth Israel Hospitals.

Throughout the revision process we had the support of many people, and though unnamed, they have our deepest appreciation. Our families deserve special recognition for their constant caring, counsel, and forbearance. We are more than fortunate that the Brigham Surgical Group Foundation continues to provide us with support not only for this effort but for research, postgraduate courses, and clinical activities.

Over the past 5 years we have had exposure to ever-increasing numbers of medical students, many of whom have chosen to pursue otolaryngology as a career. They have enriched, and continue to enrich, our academic lives. We hope this manual will meet their needs, as it is with them in mind that it is published.

M. S.
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Chapter 1. Emergencies

Severe Acute Emergencies

I. Supraglottitis (epiglottitis) is an acute infection involving the supraglottic larynx above the vocal cords to the tip of the epiglottis, including the aryepiglottic folds and the arytenoids. Swelling caused by infection in this critical part of the airway can lead to asphyxia and potentially death. Most cases in children are caused by *Haemophilus influenzae*, type B, whereas in adults a wider range of bacteria are causative (*Streptococcus pneumoniae*, beta-hemolytic streptococcus, and *Staphylococcus aureus*). In an adult, a viral etiology must also be considered. Early recognition and prompt intervention are critical to successful therapy.

A. Signs and symptoms. Supraglottitis in childhood is often characterized by abrupt onset and a relentless clinical course. The typical initial symptom is fever (temperature > 102°F), followed in a relatively short period of time by pain in the hypopharynx, often compromising the ability to swallow, and finally by respiratory embarrassment. The sequence of events from onset to a state of respiratory collapse may be as short as 4 hours. In other cases, the initial symptom complex may span 2-3 days. Differences in the clinical course are due to varied host resistance, airway size, and the virulence of the organism.

B. Diagnosis

1. The classic symptomatic triad of fever, dysphagia, and rapidly progressive respiratory embarrassment strongly suggests the diagnosis of supraglottitis. In most cases, the symptoms are sufficiently classic to proceed directly to the emergency measure of securing the airway. Supraglottitis in the older child or adult, however, may be more difficult to diagnose and is not necessarily as rapidly progressive relative to airway obstruction as it is in the young child. In the absence of definite oropharyngeal pathology, the symptoms of neck pain, aggravated by swallowing or speaking, combined with low-grade fever and a muffled voice should alert the physician to the probability of supraglottitis.

2. Lateral x ray of the neck. In cases in which the diagnosis is in doubt and the patient's condition stable, a lateral x ray should be obtained. X-ray findings of a swollen epiglottis and dilated hypopharynx establish the diagnosis.

3. Indirect laryngoscopy. The larger adult airway makes laryngeal occlusion unlikely during indirect laryngoscopy. This is not true, however, in the pediatric population. This examination should not be performed in the presence of hypoxia. The preferable current technique is use of the fiberoptic laryngoscope inserted transnasally. Placement of the tip of the laryngoscope well above the epiglottis (in the upper oropharynx) should not produce laryngospasm and allows excellent visualization of the supraglottis and possibly the level of the vocal cords. For those adults not requiring emergency measures, this technique allows for frequent evaluations of the airway.
4. Points of caution in the diagnosis of pediatric patients

a. A tongue blade examination or indirect laryngoscopy should not be performed in hypoxic children with suspected supraglottitis. The intense pain and respiratory distress that are usually present make such an exercise difficult for the patient and may provoke acute respiratory arrest from laryngeal spasm, induced by tactile stimulus of the epiglottis.

b. Children with supraglottitis may appear to have meningitis. They usually have high fever, a toxic appearance and, at times, nuchal rigidity. To position such a patient for lumbar puncture would further compromise the airway. One can make the differentiation clinically on two grounds: (1) patients with epiglottitis have respiratory signs and symptoms, which are not found in most patients with meningitis, and (2) patients with supraglottitis severe enough to cause nuchal rigidity also resist neck motion in virtually any direction - side-to-side, flexion, and extension.

C. Management. In most cases, the severity of respiratory distress requires stabilization of the airway by endotracheal intubation or tracheotomy. A decision as to the extent of airway compromise must be made, however. In some patients, especially adults, the compromise may be mild, requiring at most hospitalization for airway monitoring and administration of antibiotics, at times combined with steroids. In contrast to the pediatric population, intubation or tracheotomy is less often required in adults.

1. Stabilization of the airway. Once the diagnosis has been established, acute pediatric management usually proceeds to securing the airway. In major centres, nasotracheal intubation is the preferred method because it usually carries a lower complication rate than tracheotomy. With an endotracheal tube, however, unexpected extubation requires that an expert endoscopist be present for reintubation, if warranted. Ancillary services essential for managing small endotracheal tubes in children must also be available; close observation after intubation is as critical as the intubation process. From the time the diagnosis of supraglottitis is suspected until the airway is secured, a physician should be in attendance.

a. Nasotracheal intubation. Procedural techniques for intubation follow:

(1) General anesthesia by mask (nitrous oxide and oxygen) induction is given and subsequently enhanced with halothane. Muscle relaxants are ill-advised in this setting.

(2) Invasive procedures, even the institution of an intravenous line, should await intubation when possible.

(3) Oral intubation is performed after a satisfactory level of anesthesia is obtained. The endotracheal tube size should be at least one size smaller than that normally used for a patient of similar size.

(4) The change to nasotracheal intubation can be effected once the airway has been secured by oral intubation, cleared of all secretions, and the patient fully oxygenated. The nasotracheal tube usually remains in position 24-72 hours, depending on the clinical course. Patients seldom require reintubation if the tube is left in place for at least 24 hours.
(5) **Extubation.** performed 24-72 hours after intubation, can be performed in the ICU. Endoscopy is no longer considered essential prior to extubation.

(5) **Tracheotomy.** If expertise - medical, nursing, or both - is not available to maintain adequately a nasal endotracheal tube, a tracheostomy (see sec. V.F.) is preferable. Whenever possible, the procedure should be performed in the operating room with an endotracheal tube in place.

2. **Humidification.** During the period of mechanical ventilation, a humidified environment, with 30% inspired oxygen, is beneficial. In addition, humidification of the area around the tube helps prevent the development of thick local secretions, which could obstruct the small tubes in children. Suction and, when necessary, saline irrigation should be used both to remove secretions and check tubal patency.

3. **Antibiotic therapy.** Once the airway has been secured, antibiotic therapy should be initiated. Blood cultures should be obtained before beginning therapy to identify the bacterial organisms and their sensitivities. Such identification is of particular import in adults. Antigen studies on the serum will rapidly confirm the presence of *Haemophilus influenzae*, type B, in many patients.

   a. **Choice of drug**

   (1) **Combination drug therapy** is the initial treatment of choice. In our experience, chloramphenicol and ampicillin can be administered simultaneously without adverse effect and usually with a prompt clinical response. When the specific sensitivities become available, the antibiotic of choice is administered and other antibiotics discontinued.

   (2) **Chloramphenicol.** In many parts of the country, *Haemophilus influenzae* is often resistant to ampicillin; consequently, the recommended antibiotic regimen is chloramphenicol, 100 mg/kg/day, in four divided doses administered intravenously. For very young patients (under the age of 18 months), the dose should be lowered to 50-75 mg/kg/day. When serum levels of chloramphenicol are known, they can be used to adjust the dosage.

   (3) **Ampicillin.** If the pathogenic organism is sensitive to ampicillin, a dose of 200 mg/kg/day, in four divided doses administered intravenously, can be used.

   (4) **Second- and third-generation cephalosporins** (eg, cefuroxime, cefotaxime, ceftazidime) are excellent alternatives, particularly when ampicillin resistance is suspected or found.

   b. **Duration of antibiotic therapy.** In most instances, 7 days of antibiotic therapy is sufficient and further therapy is not required. During the last 2-3 days of hospitalization, the patient is usually ambulatory and on a regular diet.

   c. **Use of steroids.** Although steroids remain a controversial issue, they may be of value in certain patients in whom respiratory collapse is not imminent, ie, in the adult patient and some children.
Dexamethasone is administered in the following dosage: 5 mg for the first 10 lb and 1 mg/10 lb thereafter up to a total of 20 mg as an initial dose. Half the initial dose is repeated in 6 hours and no more given.

D. Prognosis. If the steps of management previously outlined are observed, patients should recover without adverse sequelae. The prognosis for recovery to normal is excellent. If any question exists about the adequacy of the airway by the managing physician, early intubation must be performed. Failure to do so may result in respiratory arrest. Complications of intubation in this setting are infrequent.

II. Croup syndromes. Croup is an ill-defined cluster of diseases, characterized primarily by inspiratory stridor but sometimes by expiratory stridor as well. Anatomically, croup affects the subglottis, in contrast to supraglottitis, and displays a characteristic biannual epidemic character, usually peaking in the beginning of the winter season or late in the fall.

A. Viral laryngotracheitis. Viral croup, the most common of the croup syndromes, usually affects patients in the first 2 years of life. Bronchitis is specifically omitted from the name because distal inflammatory disease rarely occurs concomitantly. The disease typically occurs as a community-wide epidemic.

1. Signs and symptoms. Affected children appear to be ill but are not toxic, have a croupy cough, hoarse voice, and stridor that can be both inspiratory and expiratory. There is no significant dysphagia, throat pain, drooling, or preferred position. An elevated temperature is common but is not marked or spiking in character.

2. Diagnosis. The diagnosis of viral laryngotracheitis is most often clinical. The question of additional information from radiography has not been fully explored, although there appears to be no correlation between the x-ray findings and the degree of hypoxemia. Anteroposterior x rays of the neck, however, often help confirm the diagnosis when the characteristic subglottic tapered narrowing is visualized. Lateral soft tissue views assist only in eliminating other etiologic considerations (eg, supraglottitis, foreign body). Endoscopy is considered only as part of the management protocol for advanced disease.

3. Management

a. Outpatient therapy can be considered for most patients if the respiratory rate is less than 40 times/minute and if the patient is able to maintain oral hydration. Temporary relief may be obtained by using the bathroom shower to produce instant humidification. The use of a cool-mist vaporizer in the immediate environment is an important part of the therapy, particularly in the winter season with the associated dryness secondary to heating systems.

b. Inpatient therapy is advised for children with respiratory rates greater than 40 and for those who are unable to drink adequate amounts of fluid.

(1) Intravenous fluids and appropriate humidification of the inspired air permits a number of these patients to recover uneventfully. Despite these measures, however, some will still experience substantial respiratory distress.
(2) Medication

(a) Temporary resolution can be obtained by the use of racemic epinephrine nebulized with positive pressure, using a face mask. The dose for this agent is 0.25-0.50 mL mixed with 2 mL of saline. If administered by an experienced respiratory therapist, this agent typically produces striking improvement that lasts for varying periods of time.

If racemic epinephrine is used, hospital admission is mandatory because of the risk of rebound airway obstruction in the hours following therapy. Racemic epinephrine can be administered as frequently as q30-60 min, provided tachycardia does not contraindicate its use. The frequency of treatments required by the patient is a rough indication of how the clinical course is proceeding. A patient whose interval of therapy decreases from 4 hours to 1 hour is a candidate for more aggressive therapy.

(b) The use of steroids in this disease is an unresolved question. Although there is evidence that the use of steroids reduces edema in the supraglottic space when the edema is produced by trauma, the data for viral laryngotracheitis are more confusing. Several double-blind studies using low-dose steroids have not demonstrated efficacy, although two studies have demonstrated some efficacy if high-dose steroids are used.

If steroids are elected, they should be given early in the course of illness and in high doses: dexamethasone, 0.5-1.5 mg/kg, as a single-injection IM or IV, up to a total of 20 mg in one administration.

(c) Antibiotic therapy in viral laryngotracheitis is not indicated initially. If the course becomes protracted, however, consideration must be given to the probability of a secondary bacterial infection, and on that basis appropriate antibiotic therapy should be started. In this instance, ampicillin, 100 mg/kg, parenterally administered, is the initial drug of choice.

(3) Intubation and tracheotomy. Should intravenous fluids, humidification, and antibiotics fail to reverse the clinical status of the patient, the only alternatives remaining are intubation or tracheotomy to mechanically establish an airway. With careful, advanced planning, patients requiring airway assistance can be managed systematically and without confusion. Endoscopy should be performed in the operating room, initially defining the magnitude of inflammation, detecting the presence of any associated anomalies (eg, subglottic stenosis), and removing inspissated secretions.

(a) Intubation. The area of narrowing in the airway occurs within a rigid space, the cricoid ring; therefore, it is imperative to use the smallest possible endotracheal tube consistent with good ventilation. In children, this typically means an endotracheal tube that is 0.5-1.0 mm smaller in outside diameter than would normally be used for that age or size child (Table 1-1). If a smaller than normal tube is not used, the risk of tracheal stenosis in the weeks following recovery is substantially increased. If intubation is the initial procedure chosen to establish the airway mechanically, a reevaluation is mandatory at 48 hours. If extubation is not feasible, tracheotomy becomes an important consideration.

(b) Tracheotomy. The initial choice between an endotracheal tube and a tracheotomy in croup is based on many of the same issues as for supraglottitis (see sec. I.C.). In croup,
however, tracheotomy does offer the distinct advantage of not placing a foreign body through
the relatively immobile inflamed subglottis and should be considered early for slow resolution.

B. Bacterial laryngotracheitis is less common than viral, typically being caused by
Staphylococcus aureus, Streptococcus pyogenes, Streptococcus pneumoniae, or Haemophilus
influenzae. Clinically, the illness may be indistinguishable from viral croup but should be
suspected when there is a persistent fever, elevation of white blood count, or lack of
resolution with normal therapeutic measures. Tracheoscopy and cultures from the area identify
the offending organism in most instances and guide appropriate antibiotic therapy.
Occasionally, obstructing subglottic mucous casts or thick secretions necessitate their removal.

Table 1-1. Size of bronchoscopes and tracheotomy tubes in children

<table>
<thead>
<tr>
<th>Age</th>
<th>Bronchosopes (mm x cm)</th>
<th>T tubes Routine</th>
<th>T tubes Respirator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature</td>
<td>3 x 20</td>
<td>00</td>
<td>0</td>
</tr>
<tr>
<td>Newborn - 3 mo</td>
<td>3.0-3.5 x 20-25</td>
<td>00 or 0</td>
<td>0 or 1</td>
</tr>
<tr>
<td>3-6 mo</td>
<td>3.5 x 25</td>
<td>0 or 1</td>
<td>1</td>
</tr>
<tr>
<td>6-12 mo</td>
<td>3.5-4.0 x 30</td>
<td>0 or 1</td>
<td>1 or 2</td>
</tr>
<tr>
<td>1-2 yr</td>
<td>3.5-4.0 x 30 4 x</td>
<td>1 or 2</td>
<td>1 or 2</td>
</tr>
<tr>
<td>3 yr</td>
<td>30</td>
<td>1 or 2</td>
<td>2 or 3</td>
</tr>
<tr>
<td>4 yr</td>
<td>4-5 x 30-35</td>
<td>2</td>
<td>2 or 3</td>
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<tr>
<td>5-7 yr</td>
<td>5 x 35</td>
<td>2 or 3</td>
<td>3 or 4</td>
</tr>
<tr>
<td>8-12 yr</td>
<td>5-7 x 35-40</td>
<td>3 or 4</td>
<td>4 or 5</td>
</tr>
</tbody>
</table>

C. Spasmodic croup is poorly defined entity consisting of croup symptoms, rapid in
onset, usually occurring at night with a very short clinical course. An environmental change
often effects resolution; steam and cool night air seem to be equally beneficial. Continuous
symptoms for more than 24 hours would ordinarily rule out this diagnosis.

D. Recurrent croup suggests the possibility of a congenital anatomic abnormality,
most often subglottic stenosis.

III. Foreign bodies. Cough and recurrent pneumonia, stridor, asthma, and respiratory
arrest have all been seen as the result of aspirated foreign material. The symptoms relate
primarily to the site of impaction. The hypopharynx is the area most frequently involved.
Denture wearers or intoxicated patients are prone to aspiration of foreign bodies. Chicken and
fish bones are the foreign bodies most often identified.

A. Symptoms

1. Hypopharynx. Most frequent among the presenting symptoms are pain on
swallowing and persistent localized throat discomfort. The patient can readily demarcate the
involved area. Hoarseness is not a frequent occurrence, yet the voice may have a "hot potato"
quality, sounding more muffled with larger foreign bodies.

2. Larynx. Impacted foreign bodies in the larynx often produce significant partial or
total airway obstruction. The Heimlich sign, bringing the hand to the throat, is almost
universally given. If complete obstruction occurs, the patient is unable to phonate, rapidly becomes pale, then cyanotic, showing increasing anxiety with subsequent agitation and then coma. Partial obstruction initially causes a cough, followed by hoarseness and stridor. Stridor may take several days to develop with smaller foreign bodies.

3. **Trachea.** Tracheal foreign bodies cause symptoms similar to those noted in the larynx. A brief but noteworthy coughing prodrome, followed by a quiet interlude, should suggest aspiration of a foreign body. Those that partially obstruct and change position may produce intermittent stridor, an audible thud or, if obstructing, ultimately cyanosis.

4. **Bronchus.** Objects usually lodge in the more distal air passages in children. The initial episode may go unnoticed. In 80% of cases, coughing, choking, or wheezing occurs alone or in combination. Not infrequently, however, the foreign body initially goes undetected. Therefore, unexplained recurrent pneumonia or localized asthma in childhood should always raise the suspicion of a foreign body.

**B. Diagnostic technique**

1. **Indirect laryngoscopy** is especially beneficial in the evaluation of hypopharyngeal foreign bodies, which are most often located in the tongue base or piriform sinus. This technique is similarly beneficial in detecting laryngeal foreign bodies. It is the procedure of choice if there is a suspicion of a radiolucent aspirate (eg, apple cores). In uncooperative patients, fiberoptic laryngoscopy via the transnasal route should be routine, not only for the hypopharynx and larynx, but also for the upper trachea.

b. **Radiography** can prove beneficial in the evaluation of radiopaque objects in the hypopharynx, larynx, trachea, and bronchi. Anteroposterior (AP) and lateral views of the neck, in addition to a lateral chest film with the arms held posteriorly, are necessary to assess the tracheobronchial tree. Fluoroscopy is mandatory, because 34% of radiolucent foreign bodies are not detected on routine x-ray studies within the first 24 hours. If the foreign body is not radiopaque, a region of obstructive atelectasis or emphysema can be noted fluoroscopically and augmented by changes in respiration.

**C. Points of caution**

1. **Total obstruction** with sudden aphonia while eating is an emergency situation, best handled by a sudden scapular blow. If this blow does not dislodge the foreign body, it should be followed immediately by the Heimlich maneuver. If both are unsuccessful, an emergency cricothyrotomy is indicated.

2. **Injudicious manipulation of a foreign body** in the trachea may cause subglottic impaction with acute obstruction. In the presence of an adequate airway, the patient should not be disturbed, and an endoscopy should be performed in a controlled manner.
D. Management

1. Hypopharynx. Removal of hypopharyngeal foreign bodies can be effected by having a cooperative patient hold the tongue, freeing the examiner's hand for a mirror-and-instrument extraction. If this fails, removal may require general anesthesia.

2. Larynx. Controlled laryngoscopy under general anesthesia is the most efficacious method of removal.

3. Tracheobronchial tree. Tracheobronchial foreign bodies should be removed endoscopically in the controlled operating room environment. Usually, the airway is secure in that only a portion of the respiratory tract is obstructed. Ill-conceived attempts at removal often cause further impaction of the object. A "safety zone" of time usually exists, allowing appropriate personnel, facility, and instruments to be prepared. A duplicate of the foreign object should be obtained, whenever possible, enabling the endoscopist to select the most appropriate instrumentation for removal. Topical vasoconstrictors, Fogarty catheters, preoperative steroids, and magnification are helpful adjuncts in selected instances.

IV. Neck infections compromising the airway

A. Submandibular space. (See Chap 4, V.A.4.) Ludwig's angina is infectious involvement of the entire submandibular and sublingual space. Dental or periodontal infections are the most common sources of submandibular abscesses. It is not uncommon, however, for a submandibular gland infection to initiate a submandibular abscess. Initially, cellulitis may be limited to the sublingual space, and the abscess may be drained through the floor of the mouth.

1. Anatomy. The submandibular region is composed of two anatomic spaces: (1) the sublingual space, occupying the region above the mylohyoid muscle, and (2) the submaxillary space, lying external to the mylohyoid. These two spaces are in direct continuity posteriorly.

2. Signs and symptoms. If the tongue is elevated, but the neck is only minimally involved, respiratory compromise is not present. If infection penetrates through the mylohyoid, the disease process progresses rapidly. The submaxillary space becomes hard, and swallowing elicits pain. The temperature is elevated and, as the posterior tongue swells, respiration becomes labored.

3. Diagnosis. Dental and mandibular films are warranted to rule out a foreign body. A lateral x ray is most beneficial in assessing the degree of swelling in the tongue base.

4. Management. Therapy is directed primarily toward maintaining an airway and controlling infection. Appropriate antibiotics are given, especially for staphylococci and streptococci. Anaerobes must be considered. Antibiotics alone are the regimen of choice when respiratory obstruction is not present. High-dose penicillin intravenously or clindamycin intravenously (or both) is preferred. If respiratory obstruction occurs because of the posterior spread of infection of the tongue base or supraglottic larynx, a tracheotomy (see sec V.F.) must be performed. Surgical management necessitates opening the tense space from the submental region into and including the tongue base.
B. Retropharyngeal space. (See Chap 4, V.A.2.) Infections in the retropharyngeal space are most common in infants and children and arise from infection involving the nasopharynx, adenoids, and posterior nasal chambers, as well as the sinuses. Foreign bodies that penetrate the posterior pharyngeal wall can similarly be etiologic. Tuberculosis has produced these abscesses in adults.

1. Anatomy. The retropharyngeal space lies behind the hypopharynx and esophagus, extending from the base of the skull to the first thoracic vertebrae. Infections in this space can spread posteriorly to the prevertebral space that extends the entire length of the vertebrae or laterally into the mediastinum.

2. Signs and symptoms. The difficulty in diagnosing high abscesses in a child arise when lymphoid tissue, normally present in the nasopharynx, is enlarged. If an abscess is present, the patient's neck is usually held rigid and tilted away from the side of involvement. Pain is present, especially on swallowing. The child is febrile, the voice is muffled, and respiratory difficulty occurs relatively early in the course of the disease. Cellulitis secondary to adenitis occurs more frequently than a true abscess, and the symptomatology is less fulminant.

3. Diagnosis. Palpation can help delineate the true nature of a retropharyngeal swelling. When an abscess is suspected, palpation should be performed with the patient's head in a dependent position in the event of rupture. An abscess can be suggested by a lateral soft tissue film of the neck, with edema and secondary widening of the retropharynx being detailed and confirmed by fluoroscopy or computed tomography (CT) scan.

4. Management. Therapy includes antibiotics alone for cellulitis or in combination with surgical drainage for abscess formation. The surgical approach, either transoral or external, depends on the extent of the infection. An abscess extending below the level of the hyoid bone should have external drainage. Smaller superior collections respond to the transoral approach.

C. Pharyngomaxillary (parapharyngeal) space. (See Chap 4, V.A.3.) Infections in the pharyngomaxillary area are common and arise from disease in the tonsils, adenoids, teeth and adnexa, parotid glands, and lymph nodes that drain the nose and pharynx. The posterior compartment may become contaminated via infection in the middle ear and mastoid (Bezold's abscess). Although not directly obstructing the airway, this most common of deep facial space infections can spread to contiguous areas, impinging on the airway in the pharynx or neck.

1. Anatomy. The pharyngomaxillary space is shaped like an inverted pyramid; the base is the base of the skull, and the apex is the hyoid bone. This space is divided by the styloid process into anterior and posterior compartments.

2. Signs and symptoms

   a. Anterior compartment involvement causes trismus (internal pterygoid muscle irritation) and swelling of the lateral pharyngeal wall.
b. Posterior compartment infections cause less trismus than do anterior; however, infection here juxtaposes the great vessels of the neck with the potential for thrombosis or hemorrhage.

c. Common to both anterior and posterior compartments are symptoms of fever, nuchal rigidity, odynophagia, parotid swelling, and pharyngalgia.

3. Diagnosis. A CT scan should define the extent of involvement. Lateral x rays of the neck may show concomitant posterior pharyngeal swelling. If vascular obstruction is suspected, orbital plethysmography can prove beneficial. Angiography may become essential if surgery is a consideration.

4. Management. Antibiotics, primarily cefoxitin in combination with gentamycin, must be administered intravenously in high doses. Surgical drainage when necessary is through an external incision in the submandibular region, going deep to the submaxillary gland and cephalad along the carotid sheath. Once recognized, surgery should be an early consideration.

V. Trauma causing respiratory embarrassment is seen with injury occurring in the area extending from the anterior oropharynx to the cervical trachea. Trauma can range from displaced dentures occluding the airway to tracheal separation. With severe trauma, there may be more than one region contributing to airway obstruction.

A. Points of caution

1. Maintenance of the airway must be the primary management consideration.

2. Motion of the neck must be restricted until injury of the cervical spine is excluded.

B. Midface and mandible

1. Initial considerations. Obstruction of the airway can occur with mid- or lower-facial injury secondary to hemorrhage, edema, and posterior displacement of fracture segments. Foreign materials (eg, denture fragments) are frequent causes of obstruction. No specific diagnostic respiratory obstructive patterns are manifested by these injuries and, therefore, the physician must remain alerted to the frequently associated airway compromise. Proper management must first include the airway and then the fracture. The diagnosis should be established using clinical information, and appropriate measures should be taken to secure the airway. Time should not be wasted in first obtaining x rays or ancillary studies (eg, blood gases).

2. Management

a. The oral cavity and pharynx must be cleared of debris, clot, and foreign body, either manually or by suction.

b. After cleaning, if the airway is still obstructed, simple repositioning of retrodisplaced segments should be performed. An oral or nasopharyngeal airway can maintain air flow until definitive fixation of the fractured segments is performed.
c. An emergency tracheostomy is rarely necessary for these injuries, but should be considered when swelling of the tongue base obstructs the airway.

C. Larynx. Injury to the larynx can occur from either blunt or penetrating trauma. A penetrating injury, associated with an open wound and air emanating from the site of impact, demands consideration of a laryngeal injury. Blunt trauma is more difficult to evaluate because the symptoms and signs may be slow to evolve.

1. Signs and symptoms. Subcutaneous emphysema, progressive airway obstruction, dysphonia, focal neck pain, loss of the thyroid cartilage prominence, and overt intralaryngeal mucosal lacerations can all be associated with significant laryngeal trauma. Dislocation of the cricoarytenoid joint must be considered, but in the acute stage, edema may mask this relatively subtle finding.

2. Diagnosis
   a. Indirect laryngoscopy should be attempted initially, but may be difficult to perform in patients with multiple injuries.
   b. Direct laryngoscopy. Flexible fiberoptic laryngoscopy can frequently clarify the nature and magnitude of the injury. When doubt exists, direct rigid laryngoscopy should be performed in the operating room with the airway secure. Disruption of normal anatomy and the presence of mucosal lacerations, foreign material, and cartilage injury should be documented. Cord mobility must be assessed and the arytenoids palpated. In massive trauma, injury to the trachea or esophagus often coexists; thus, endoscopic examination of these structures is indicated.
   c. Radiographs. Plain films are of little value in assessing acute injuries, except for delineating the presence of subcutaneous emphysema. CT scans can aid in assessing the extent of laryngotracheal injury. Chest films assist in the evaluation of a pneumomediastinum or pneumothorax.

3. Management
   a. Tracheotomy (see F.) is mandatory in the presence of an unstable airway secondary to laryngeal injury. Cricothyrotomy should be avoided if at all possible. Laryngeal fragments can easily be disrupted and infection spread if a cricothyrotomy is performed.
   b. Acute laryngeal injuries with tissue disruption should be surgically explored and repaired at the earliest possible time. Mucosal lacerations must be meticulously repaired and cartilaginous fragments realigned and stabilized when necessary with nonreactive suture material or miniplates. Endolaryngeal stenting may be required to give intraluminal support.

Specific attention must be directed to the cricoid cartilage, since undiagnosed injuries at this site, once scarred, are very difficult to reconstruct. Unless there has been an obvious transection of the recurrent laryngeal nerve, considerations regarding function should wait. Vocal cord weakness often subside spontaneously if the nerve has been contused, but should
call attention to potential injury in the tracheoesophageal groove (i.e., great cervical vessels). Antibiotics (penicillin) should be used in open wounds.

**D. Trachea.** Although disruption of the airway with a laryngotracheal separation usually involves an open wound, rapid flexion-extension in sudden deceleration can produce the same injury.

1. **Signs and symptoms**
   a. Upper tracheal injury has diagnostic characteristics similar to those of the larynx.
   b. Subcutaneous emphysema should suggest a tracheal or esophageal disruption, although not signifying the magnitude.

2. **Management**
   a. The esophagus must be examined for laceration, and the mediastinum should be evaluated for associated injury.
   b. The mandatory tracheotomy should be placed as far from the site of injury as possible.
   c. Tracheal separations must be repaired as soon as possible.
   d. Debridement of obviously necrotic tracheal tissue may require a supraglottic laryngeal release to allow primary tracheal approximation.

**E. Soft tissue of the neck**

1. **Points of caution**
   a. With blunt neck trauma, the primary consideration must be maintenance of an adequate airway.
   b. The cervical spine deserves serious thought in the initial evaluation.

2. **Signs and symptoms.** Open or penetrating wounds of the neck are frequently associated with significant vascular involvement.
   a. An expanding mass within the neck raises the possibility of vascular injury.
   b. A patent airway can be jeopardized by progressive soft tissue swelling.
   c. Bleeding by mouth can indicate significant midneck vascular (carotid) disruption.

3. **Diagnosis.** When time allows, vascular radiographic studies are invaluable in the specific delineation of the site of injury. Cervical spine injuries must also be assessed by x-ray.
Table 1-2. Indications for surgical exploration of neck wounds

I. Vascular injury
A. Immediate indications
1. Hemorrhage from neck wound.
2. Active transoral bleeding without visible source.
3. Chyle leak.
4. Expanding cervical hematoma.
5. Airway obstruction.
6. Widened superior mediastinum.
7. Absence of pulses.
8. Progressive CNS deficit from hypoperfusion.
B. Delayed indications
1. Major vessel thrombosis.
2. Occult hemorrhage.
3. Arteriovenous fistula.
4. Aneurysm or pseudoaneurysm.
5. Vertebral artery injury.
6. Thoracic inlet injury.
II. Neurologic injury
A. Jugular foramen syndrome (deficit of cervical sympathetic, fourth, fifth, sixth, and seventh nerves).
B. Submandibular space injury with deficit to seventh and lingual nerves.
C. Horner's syndrome.
D. Brachial plexus deficit.
E. Hemiplegia.
F. Diminished visual acuity or contraction of visual fields.
III. Respiratory and digestive tract injury
A. Immediate indications
1. Crepitus.
2. Stridor.
3. Aphonia or dysphonia.
4. Dysphagia.
5. Hyoid, thyroid, and cricoid cartilage painful to palpation.
7. Positive endoscopy.
8. Positive contrast study.
B. Late indications.
1. Neck infection.

4. Management

a. Pressure tamponade should be performed initially. Indiscriminate clamping or ligation may cause associated nerve injury.
b. Transoral bleeding can be controlled by pharyngeal packing; however, a tracheotomy (see F.) is mandatory prior to placement of the pack. Vaginal packing with a long instrument (eg, a Kelly clamp) can be used.

c. Lower neck trauma can be associated with injury to the intrathoracic vasculature, and these injuries require an emergency thoracotomy.

d. Not all open neck wounds require exploration. If significant structural injury is suspected (Table 1-2), exploration is performed.

F. Tracheotomy: potential considerations

1. Indications

The need to establish an airway with a tracheotomy is due to airway obstruction or to ventilatory failure. Either can be acute or slowly progressive. The adage that when a tracheotomy is considered, it should be performed remains valid. It is far better to place a tracheotomy in a controlled operating room environment under local or general anesthesia than under duress with an anxious, obstructed patient and flustered personnel. The following outlines the indications for a tracheotomy.

a. Obstruction

(1) Trauma

(a) Partial or complete obstruction of airway.

(b) Blunt injury with edema of the endolaryngeal or tracheal structures.

(c) Blood or foreign material in airway.

(d) Maxillofacial injury with aspiration and local edema.

(e) Collapse of airway and soft tissue support.

(2) Foreign body

(3) Inflammation

(a) Secondary to infection (eg, supraglottitis).

(b) Angioedema.

(c) Allergic response.

(d) Deep neck space abscess.

(e) Secondary to caustic or thermal injury.
(4) Congenital lesions
(a) At birth (eg, subglottic stenosis).
(b) Aggravated by superimposed infection.
(c) Progression of abnormality (eg, laryngeal cyst, Pierre Robin syndrome).

(5) Neoplasia
(a) Hemangioma, lymphangioma.
(b) Squamous cell carcinoma.

(6) Paralysis or paresis of the vocal cord
(a) Bilateral.
(b) Associated neuromuscular dysfunction.

b. Ventilatory failure

(1) Retained secretions
(a) Depression of cough, postsurgical, pneumonitis, cystic fibrosis.
(b) Aspiration of gastric contents.

(2) Inefficient respirations due to depressed ventilatory stimulus of coma, central nervous system (CNS) disease, chest wall fracture or paralysis, chronic lung disease, head trauma.

2. Method highlights. In most situations, placement of an endotracheal tube should be the primary mode of airway management when simpler measures fail. Circumstances arise when intubation is not feasible (eg, massive injury to the lower midface, laryngeal fracture, or an acutely obstructing foreign body). If an open path to the airway is present, it should be cannulated with an endotracheal tube as a temporary measure. Especially in children, morbidity and mortality increase when a tracheotomy is performed in a struggling patient without initial airway control via an endotracheal tube or bronchoscope.

Cricothyrotomy should be used only as a temporary measure, until a controlled tracheotomy can be performed. A vertical incision is usually used for ease and rapidity in an emergency tracheotomy. Sedation is contraindicated in acute obstruction, because respiratory collapse or total obstruction may ensure. Silk stay sutures placed through the tracheal wall and brought out through the wound may be invaluable should the tracheotomy tube become dislodged. Tracheotomy tape should be tied with the patient's head in flexion; otherwise, the tape will be too loose. After a tracheotomy, a chest x ray should be obtained to assess tube placement and to rule out a pneumothorax.
3. Complications. An emergency tracheotomy is hazardous under the best conditions. Prior control of the airway greatly diminishes the incidence of complications. The following complications are divided into immediate and delayed: those occurring within 24 to 48 hours after the operation, and those occurring after 48 hours.

a. Immediate complications

(1) Hemorrhage may occur during the procedure from a prominent anterior jugular vein, an anomalous artery, or the thyroid gland.

(2) Pneumothorax and pneumomediastinum occur in more than one-third of emergency procedures, usually in patients with acute respiratory failure, as well as in those tracheotomies performed without an indwelling endolaryngeal tube. Avoidance is effected by minimum dissection of the pretracheal fascia and by controlled positive pressure. Often a pneumomediastinum needs no further therapy.

(3) Immediate apnea occurs in patients with hypercarbic respiratory stimulation in whom the hypoxic drive has been eliminated. On insertion of an airway, blood PCO₂ is diminished, and the respiratory stimulus is decreased or abolished. Controlled respiration may be needed in this situation.

(4) Subcutaneous emphysema sometimes results from tight incisional closure, aggravated by mechanical ventilation.

(5) Atelectasis, usually of the left lobe, occurs when a long tube is in the right mainstem bronchus. This condition is relieved by appropriate repositioning of the tracheotomy tube.

(6) A poorly positioned or dislodged tube occurs when the trachea is not cannulated under direct vision or the tube is not a proper length. Suturing the flanges to the neck helps avoid accidental removal. This is a frequent cause of death in the initial postoperative period.

(7) Aerophagia may occur in children under the age of 3 and leads to gastric dilatation and cardiac arrhythmias. A nasogastric tube provides relief.

(8) Tracheoesophageal fistula rarely occurs as an immediate problem unless the tube is forced posteriorly and transects the trachea and esophageal wall.

(9) Recurrent laryngeal nerve paralysis occurs when the surgical dissection is off the midline, into the tracheoesophageal groove.

b. Delayed complications

(1) Hemorrhage may occur secondary to local granulation at the stoma. It can result from tracheal trauma from too vigorous suctioning, as well as dryness with associated crusting. More seriously, it may be due to erosion of a prominent vessel (eg, the innominate artery) by the tracheostomy tube. The incidence of such can be decreased by placement of the tube at the second tracheal ring.
(2) **Laryngeal stenosis** is secondary to a tube placed into the larynx or the cricothyroid membrane and not lowered to the trachea.

(3) **Tracheal stenosis** occurs at the level of either the stoma or the cuff and is usually due to erosion (by motion, infection, and pressure).

(4) **Tracheitis sicca** most often occurs during the winter, when drying of the tracheal mucosa results in crusting. Adequate humidification is a must.

(5) **Displaced tracheotomy tube** is discussed in a(6).

(6) **Tracheoesophageal fistula**, occurring as a delayed complication, is secondary to erosion by the tube or cuff, usually in the presence of a firm nasogastric tube. It can be a fatal complication in debilitated patients.

(7) **Dysphagia and aspiration** are usually secondary to loss of laryngeal competence and to irritation of the esophagus by the tracheotomy tube.

(8) **Delayed decannulation** is most often noted in children who rely on the open airway and who may require a gradual decannulation procedure (ie, progressively smaller tubes). If airway obstruction is present with plugged, fenestrated tracheotomy tube in place, the plug must be removed and the cervical airway reassessed.

VI. Acute angioneurotic edema (see Chap 4, V.B.2.).

A. **Allergic angioneurotic edema** of any region of the larynx may occur as a response to a wide range of provocabors, the most common of which are foods, inhalants, and drugs. A family history of asthma, hay fever, eczema, or angioneurotic edema may be used to differentiate hereditary and nonhereditary types. Aspirin is a common precursor; however, sensitivities to chicle, bee stings, house dust, cosmetics, and beef have also been reported. Iodine ingestion, as well as tetanus antitoxin and vaccines, induce the same responses.

1. **Signs and symptoms.** Laryngeal edema may occur after administration of penicillin, either orally or parenterally, and can be delayed 1-4 hours after exposure. Acute swelling is an anaphylactic reaction that can involve the epiglottis, arytenoids, ventricular bands, true vocal cords, or subglottis.

2. **Diagnosis** may be difficult. A family allergy history is invaluable. On indirect laryngoscopy, pale boggy edema of the entire larynx or a portion thereof is noted.

3. **Treatment.** In the early stages, subcutaneous epinephrine (0.3 mL) can rapidly reverse the swelling. Supplemental use of an antihistamine (diphenhydramine, 25-50 mg IV) or corticosteroid (eg, dexamethasone, 5-10 mg IV) may be of value. If response to medical therapy does not occur, an airway must be established. The airway is best accomplished by endotracheal intubation. Cases of failure of intubation alone to control the airway have been reported, so the patient's airway must be carefully monitored, even after the endotracheal tube has been placed.
**B. Hereditary angioneurotic edema (HAE)** is a genetic disease of autosomal dominant inheritance in which there is a deficiency of the inhibitor of the first component of complement. The attacks begin in childhood, occur frequently, and diminish in the middle-age years. Mortality has been reported to vary from 5-50%.

1. **Signs and symptoms.** HAE differs from other forms of angioneurotic edema because of the presence of associated gastrointestinal symptoms (intermittent abdominal pain, nausea, vomiting, diarrhea), peripheral edema, poor response to standard epinephrine, steroid and antibiotic therapy, and more pronounced laryngeal involvement. Attacks are frequently brought on by trauma, stress, or anxiety, and have been aggravated by pregnancy and menstruation.

2. **Diagnosis** is aided by the laboratory measurement of C1 esterase, which is abnormally low. Also, C4 and C2 levels (other complement system components) may be depressed. Testing reveals no specific allergic substance. The same pale, swollen larynx found in allergic angioneurotic edema is seen on indirect laryngoscopy in complement-initiated angioneurotic edema. Once an attack begins, laryngeal edema may be slow to progress and may not reach maximum proportions for 24-48 hours, in contrast to the more rapidly progressing allergic variety.

3. **Management**

   a. **Acute** therapy, if the airway is endangered, often requires a tracheotomy. The trauma of intubation may aggravate the local laryngeal edema. As noted in A.3., steroids, antihistamines, and epinephrine may not prove beneficial. In those patients with known HAE, close observation of the airway is mandatory. Purified C1 esterase inhibitor is available and effective in the acute situation.

   b. **Maintenance** measures are currently directed toward inhibiting plasma so that less C1 esterase is produced. Epsilon aminocaproic acid (EACA) is such an antiplasma substance, as is the analog of EACA, tranexamic acid. These two agents have been used in both short- and long-term therapy of patients with hereditary angioneurotic edema. Androgens, such as danazol or Stanazol, have also been successful therapeutically when used as a preventive measure.

**VII. Acute airway problems of the newborn and infant.** Respiratory distress in the newborn can occur from mechanical airway obstruction or from depression of physiologic respiration. Often the differentiation is difficult; however, slow, shallow respiratory efforts in a cyanotic child should suggest central, metabolic, or cardiovascular disturbance. What follows is a brief outline of some significant head and neck lesions causing mechanical airway obstruction.

**A. Choanal atresia** (see Chap 4, V.D.'). At birth and for the first 2-3 weeks of life, the child is an obligate nasal breather. Nasal airway obstruction, irrespective of etiology, can occlude the only functional air passage. This is a true emergency and must be dealt with rapidly. Choanal atresia can be either bilateral or unilateral. Bilateral obstruction causes symptoms from birth, while unilateral atresia may go unnoticed until the first upper respiratory tract infection, at which time the child becomes acutely symptomatic. The atresia
is most often bony (90%), and the remainder are membranous, presumably secondary to a persistent buccopharyngeal membrane.

1. Signs and symptoms. The neonate shows signs of nasal obstruction relieved with crying. A mucoid cast of the nasal chambers may be present. Persistent unilateral mucoid rhinorrhea should raise the suspicion of either atresia or a foreign body.

2. The diagnosis of choanal atresia is confirmed by CT scan. Other associated skull base malformations are delineated as well. The standard advocated approach - passing a nasal catheter - can be misleading and is not recommended. The CT scan has replaced older contrast studies.

3. Therapy

   a. Acute. A McGovern nipple (a baby bottle nipple with an open tip) or a conventional oral airway can be placed to maintain a patent airway until the nasal conduit can be established or until mouth breathing is acquired.

   b. Delayed management. Repair should be performed, in most instances, when the physical condition affords the administration of general anesthesia. Associated anomalies must be excluded. With the CHARGE syndrome (Coloboma, Heart disease, Atresia choanae, Retarded growth, Genital hypoplasia, and Ear anomalies) initial tracheotomy is the management. If corrected early, choanal atresia associated with CHARGE almost uniformly restenoses. Even in the child who rapidly acquires mouth breathing skills, an unrepaired bilateral atresia can alter the growth and development of the face. Surgery can be performed using microtechniques transnasally, and in selected cases the carbon dioxide laser may prove beneficial. Thick bony plates can require a transpalatal approach, and it remains the authors' procedure of choice. Stenting with plastic sheeting or tubing may be necessary for a period of 4-6 weeks to ensure mucosalization.

   c. Differential diagnosis. Other abnormalities causing nasal airway obstruction include encephalocele, meningocele, and adenoidal hypertrophy.

B. Congenital lesions of the mouth and pharynx rarely produce an acute airway emergency, but may precipitate respiratory distress by a combination of a lack of pharyngeal support and ineffective bolus transport with a predilection to aspiration. Such lesions include:

   Cleft lip or palate (or both).
   Pierre Robin syndrome (micrognathia, cleft palate, microglossia, and glossoptosis) (see Chap 4 V.D.1.).
   Oral hemangioma or lymphangioma.
   Treacher Collins syndrome (mandibulofacial dysostosis).

C. Laryngeal abnormalities. Although congenital lesions of the larynx represent the most common anomalies causing respiratory distress in the newborn, acute obstruction is rarely the cause.
1. Laryngomalacia (or exaggerated infantile larynx) is the most common of these lesions. There is a lack of rigidity of the soft collapsible epiglottis, supraglottic structures, or both that easily occludes the airway during deep inspiration. Fortunately, this condition usually resolves by 18-24 months of age.

   a. Signs and symptoms. The stridor produced is usually high pitched, but the cry is clear and strong. Stridor is aggravated in the supine position.

   b. Diagnosis is made by direct laryngeal examination. The epiglottis appears narrow, curled, and elongated.

   c. Therapy. Bronchoscopy should be considered in severe cases because of the possibility of associated anomalies in the tracheobronchial tree. In spite of the potential for total airway obstruction, tracheotomy is rarely indicated. Micro-laser techniques can have a major therapeutic impact in this disorder.

2. Atresia and web

   a. Atresia. Although rare, complete congenital obstruction of the larynx can occur, causing immediate airway distress in the newborn. The infant violently makes attempts at respiration, but to no avail. Direct laryngoscopy with possible intubation should be attempted. Often the atresia plate is cartilaginous, not allowing the endotracheal tube to pass. Tracheotomy must then be performed.

   b. Webs. Laryngeal web formation occurs because of arrest of laryngeal development at the tenth fetal week. Total arrest causes atresia; webs of the larynx represent "partial" atresia. Most webs (75%) occur at the level of the vocal cords, with the remaining being equally divided between supraglottic and subglottic. Webs in the subglottis are frequently associated with cricoid abnormalities.

      (1) Diagnosis. Infants with laryngeal webs have alterations in cry and in respiration. The magnitude of the symptoms varies with the extent of the web.

      (2) Therapy. Often a thin membrane involves the anterior half of the glottis and can be incised endoscopically. Thicker webs may require repeated excision (eg, with the carbon dioxide laser), dilatation, and stenting. Insertion of a metal or plastic laryngeal keel at the anterior commissure may be necessary to prevent reformation after thyrotomy has been performed.

3. Congenital subglottic stenosis may occur as an isolated abnormality and, after laryngomalacia, is the second most common laryngeal anomaly. The potential for significant airway obstruction is definitely aggravated with the first upper respiratory tract infection. Subglottic stenosis can be due to failure of development of the endolarynx, the cricoid cartilage, or both.

   a. Signs and symptoms. Stridor may occur from birth, but can often be mild or absent until an infection intervenes. It is at this time that acute obstruction occurs.
b. **Diagnosis** should be suspect in all children with congenital stridor and in those infants with recurrent episodes of "croup". The diagnosis is made by direct inspection prior to intubation so that iatrogenic subglottic stenosis is not a consideration.

c. **Therapy.** Significant obstruction requires a tracheotomy. Resolution can be augmented by repeated dilatation, endolaryngeal excision via the laser, or open cricothyrotomy and insertion of autogenous cartilage or bone, depending on the degree of stenosis.

4. **Laryngeal paralysis.** Paralysis of the vocal cords in infants can be unilateral or bilateral, the latter being more common. Both types may be due to birth trauma with cervical injury, causing a transient state that usually clears in 4-6 weeks. Bilateral paralysis is frequently associated with a central nervous system disorder. It may be due to injury of the nucleus ambiguus (by hemorrhage or anoxia) or associated with meningomyelocele, Arnold-Chiari malformation, or cerebral agenesis.

Cases of hereditary bilateral abductor vocal cord paralysis have been reported in both normal and mentally retarded patients. Hypoxia, because of laryngeal obstruction, has been suggested as a possible cause of the associated retardation. Unilateral leftsided cord paralysis is more common than right because of coincident cardiovascular anomalies. It is compounded by the longer course of the recurrent laryngeal nerve on the left. Esophageal or paraesophageal abnormalities can impinge on the nerve, causing subsequent cord weakness.

a. **Signs and symptoms.** Unilateral lesions may be associated with very few symptoms, which include a weak cry and hoarseness. Stridor or respiratory compromise rarely occurs. Patients with bilateral cord paralysis may have a normal cry because of the symmetry and apposition of the vocal cords. Respiratory obstruction is not uncommon, especially in association with upper respiratory tract infection.

b. **Diagnosis** is made by direct laryngoscopy without anesthesia using the pediatric fiberoptic laryngoscope. Preoperative ultrasound can further define the condition.

c. **Management.** When obstruction is not a problem, the patient should be followed expectantly. With obstruction, tracheotomy may be necessary to establish an airway.

5. **Neoplasms.** The most common neoplasms in infants and children are papillomas and subglottic hemangiomas.

a. **Papillomas** are the most frequent laryngeal tumors of childhood and can be present at birth. They tend to recur and involve any region of the respiratory tract. The etiology is viral. Seeding to the distal respiratory tract has been noted primarily after surgical manipulation or tracheotomy. Spontaneous remission has been noted and can occur at any time. The lesions are benign, but malignant transformation has been associated with radiation therapy.

  (1) **Signs and symptoms** include persistent hoarseness, a croupy cough and, occasionally, progressive airway obstruction.

  (2) **Diagnosis** is best made by direct examination and biopsy.
(3) **Management.** Current therapy is directed toward maintenance of an adequate airway by periodic tumor removal and avoidance of a tracheotomy. The carbon dioxide laser is now the established treatment. In selected cases, microexcision and cryosurgery still have their proponents. Once a tracheotomy is placed, papilloma formation may occur at or near the stoma. Approximately 6% of children with severe respiratory difficulty require a tracheotomy.

b. **Hemangiomas.** Subglottic hemangioma may occur in isolation or in association with cutaneous lesions. There is a female preponderance by a ratio of 2:1.

(1) **Signs and symptoms.** These are often congenital lesions and may appear as recurrent episodes of "croup". Growth of the tumor usually ceases by 6 months of age, often followed by spontaneous regression. The symptoms are aggravated by crying, which causes engorgement of the tumor.

(2) **Diagnosis** can be made by radiography and confirmed by direct laryngoscopy. Biopsy remains controversial, but probably should be performed to rule out another tumor. Experience has shown subsequent hemorrhage to be rare.

(3) **Therapy.** If the lesion is small, the child should be followed expectantly, and often further intervention is not warranted. With progressive obstructive symptoms, CO$_2$ laser surgery must be considered as the initial treatment. With severe respiratory obstruction, tracheotomy below the level of the tumor may be necessary.

c. **Other laryngeal neoplasms** include lymphangioma (cystic hygroma) and congenital cysts (either supraglottic or subglottic).

**Epistaxis**

The warming and humidifying functions of the nose require that it have a good blood supply. The nose is supplied by both the internal and external carotid systems. The anterior and posterior ethmoid arteries arise from the internal carotid by way of the ophthalmic artery and supply the superior part of the nose. The greater palatine and sphenopalatine arteries derive from the external carotid by way of the internal maxillary artery. The superior labial is a branch of the facial artery, also from the external carotid. Numerous anastomoses communicate these two systems. A large concentration of vessels known as Kiesselbach's plexus, is found in Little's area on the anterior septum. Over 90% of the patients who seek attention for epistaxis have bleeding from this region. Bleeding tends to be anterior in the young and posterior in the old. It is important that an attempt be made to identify the bleeding point, even if packing is necessary, so rational decisions can be made about the location of packing and specific artery ligation, if this becomes necessary.

I. **Differential diagnosis**

A. **Nasal trauma** is a common cause of epistaxis. Nasal fractures usually involve tears of the mucosa and bleeding. If an ethmoid artery is torn, there may be very brisk intermittent bleeding arising high in the nasal vault.
B. **Drying of the nasal mucosa** in cold, dry weather or increased stress (eg, sneezing, nose blowing, or exercise) may be responsible for discrete bleeding from a single vessel.

C. **Hypertension** results in thickening of the arterial wall and decreased vasoconstriction, but is not per se a cause of epistaxis.

D. A **septal perforation** often crusts and bleeds at its edges. Most perforations result from overvigorous cautery, but Wegener's granulomatosis and midline granuloma must be considered, as must cocaine use.

E. **Foreign bodies** in the nose are frequently the cause of epistaxis in children. Nasal obstruction and purulent discharge often accompany the bleeding.

F. **Other causes.** A generalized ooze or persistent or recurrent bleeding requires that one consider less common causes, such as:

1. **Hematologic disorders**
   - Anticoagulant use
   - Antiplatelet drugs
   - Clotting deficiencies
   - Leukemia
   - Thrombocytopenia
   - Thromboasthenia
   - Renal failure
   - Hepatic failure

2. **Nasal tumor**
3. **Sinus tumor**
4. **Nasopharyngeal tumor**
5. **Hereditary hemorrhagic telangiectasia**

II. **Physical examination.** The first person to examine the patient with epistaxis has the best chance of clearly identifying the bleeding point. The approach must be careful and systematic.

A. **Equipment.** Head mirror and light source (or head light), nasal speculum, bayonet forceps, suction with Fraser tip, 4-5% cocaine solution, and cotton or gauze pledgets.

B. **Preparation.** The doctor and patient should be gowned. The patient should blow his or her nose to clean it of clots and then sit upright.

C. **Method of examination**

1. **Patients presenting with bleeding**
a. Observe whether the bleeding is anterior or posterior and which is the predominant nasal chamber. The bleeding may appear to be bilateral if it is very posterior in origin or if there is a septal perforation.

b. If the site of the bleeding is obscure, the nasal mucosa should be constricted, anesthetized, and cleared of blood by packing the nose with cottonoid strips that have been impregnated with 4-5% cocaine solution. An alternative to cocaine is a mixture of 4% lidocaine (Xylocaine) and 1:100,000 epinephrine. The strips should be left in place for at least 5 minutes. In the pediatric patient, a parent can assist, if necessary, by placing several drops of either solution in each nostril.

c. When the packing is removed, the nose should be inspected again. The bleeding should have slowed or stopped, and it is usually possible to identify the bleeding point. Attention should be directed to the septum, the turbinate, and the meati between the turbinates as possible bleeding sites. An extreme septal deflection may make visualization impossible.

d. Rigid fiberoptic nasal endoscopes, although not universally available, do allow for exact localization of a bleeding site and help direct specific local treatment or packing. This new technique clearly can minimize the amount of manipulation required.

2. Patients in whom bleeding has subsided. If the bleeding subsides and the site of bleeding cannot be determined, the patient should be observed for several hours for recurrence.

III. Management. The bleeding should be controlled with the least possible manipulation and trauma. This is especially important when a clotting disorder is present, because manipulation has the potential to cause more bleeding. Even a successful pack causes additional trauma on removal.

A. Packing

1. Pediatric patients. The bleeding usually subsides when pressure is applied to the caudal end of the nose for several minutes with the head upright. If pressure is ineffective, silver nitrate application is preferred to electric cautery initially, because it is less likely to cause necrosis. Any cautery, however, should be judicious. Cautery can usually be performed quickly after topical anesthesia has been applied (see B.1.). Both sides of the septum should not be aggressively cauterized simultaneously, recognizing that this could result in a septal perforation. If packing is necessary, general anesthesia may be required.

2. Adult patients

   a. Light to moderately heavy bleeding

      (1) Manual pressure. As with the child, bleeding in the adult can usually be controlled with alar pressure for several minutes.

      (2) Cautery. If manual pressure is not effective, cautery is indicated. Before cautery, the interior of the nose should be anesthetized by cocaine (or lidocaine and epinephrine)
packing. If the bleeding remains too brisk to cauterize effectively, a cotton-tipped applicator that has been dipped in 1:1000 epinephrine solution should be held against the bleeding site. A ring of surrounding mucosa can then be lightly cauterized, the applicator removed, and the bleeding site cauterized. A small piece of Surgicel or Oxycel should then be placed over the cauterized area.

**Postcautery instructions to the patient should include:**

No manipulation  
No nose blowing  
Open the mouth if you sneeze  
No straining, lifting, or strenuous activity for 1 week  
Elevation of the head of the bed for 1 week  
No hot food or drink for 1 week  
No smoking or alcohol for 1 week  
No aspirin for 1 week  
A cold-mist humidifier at the bedside  
Lubricating drops for the nose 3 times a day for 1 week (petrolatum or normal saline).

**b. Bleeding too brisk for effective cauterity** can also be controlled temporarily by injecting lidocaine with 1:100.000 epinephrine around the bleeding site. Similarly, lidocaine with 1:100.000 epinephrine can be injected into the pterygopalatine fossa.

c. **Bleeding from beneath the inferior turbinate** is best managed by wedging a small piece of Gelfoam, Oxycel, or Surgicel beneath the turbinate. If this is ineffective, it is also possible to compress the bleeding site by fracturing the turbinate toward the lateral nasal wall. Alternatively, the turbinate can be fractured medially to expose the bleeding site for cautery. **Fracturing of the turbinate, however, should not be performed unless other measures fail,** because the nasal mucosa may be lacerated, adding an additional bleeding site.

d. **General oozing from the septum** can usually be stopped by placing oxidized cellulose soaked in epinephrine over the bleeding site. Topical thrombine or Avitene applied to the bleeding site may also be beneficial.

e. **Persistent bleeding from septal perforations** can be controlled by placing a piece of Silastic on either side of the septum and sewing them together through the perforation. Avitene can be pushed into the perforated area between the two Silastic sheets.

**B. Anterior packing.** If cautery is unsuccessful, tamponade by packing may be necessary.

1. **Anesthesia.** Topical anesthesia with cocaine or lidocaine and epinephrine reduces the discomfort. Local infiltration, as for reduction of a nasal fracture, is also helpful (see Nasal Fractures, sec. III.C.). Nevertheless, packing is frequently unpleasant for both the patient and physician.

2. **Method.** Ideally, after the anesthesia has taken effect, usually 10-15 minutes, 0.5-in iodoform gauze, well lubricated with bacitracin ointment, or petrolatum-impregnated gauze
is carefully layered in the nose with bayonet forceps. The gauze should be packed firmly down toward the floor of the nose as it is layered. It should be manipulated under the inferior turbinate and layered on both sides of the middle turbinate. The packing should be firm, but not deforming, and should not extrude into the nasopharynx. A drip pad of 2-in square gauze can be taped over the nostril. Care should be taken so the nasal vestibule does not swell, thereby causing alar necrosis. The packing should be left in place for 3-5 days. After that interval, it should be cautiously removed. General oozing may follow pack removal and last several minutes, but usually stops without further manipulation.

3. Alternate methods

a. Finger cot packs can be used to pack the nasal chamber. These are made by cutting the fingers from a surgeon's glove, filling each with gauze, and tying them closed with heavy sutures. They can be lubricated with bacitracin ointment and introduced with a bayonet. To prevent aspiration of a finger cot pack, all the sutures should be tied over a dental roll placed at the anterior nares.

b. Epistaxis balloons are also useful. Some are filled with air, others with normal saline.

c. Expandable sponge (Merocel) can be placed intranasally and affords compression to the bleeding site with minimal concern for pressure necrosis. These packs are also easily removable. Some are designed with hollow central tubes to maintain a patent nasal airway.

4. General instructions for the patient. In addition to the instructions after cautery listed in 2.a(2), the patient should:

- Have an antibiotic (ampicillin or erythromycin), because the sinuses are obstructed.
- Have some medicine to dull the pain.
- Expect a headache.
- Expect epiphora and sometimes blood from the lacrimal punctum.
- Expect nasal drainage and obstruction for 1-2 weeks after the pack is removed.
- Have careful follow-up after pack removal for lysing of synechiae as they form.

5. Hospitalization. An anterior pack can cause increased respiratory effort. In the elderly or critically ill, there is a risk of respiratory decompensation. Complete nasal obstruction, as with bilateral anterior packs or with most anteroposterior packs, is dangerous even in the young. Hospitalization of patients with anterior or posterior packs and careful observation are mandatory. Serial blood gas determination and oxygen may be necessary. The patient with a pack should not be sedated.

C. Anteroposterior packing. When the bleeding is posterior, an anterior pack may not provide sufficient tamponade. An anteroposterior pack is the conventional treatment in this circumstance.

1. Anesthesia should include intranasal topical anesthesia, infiltrative anesthesia, and limited intravenous sedation only in the most uncooperative patients.
2. Method

a. First, the posterior pack is prepared. A vaginal tampon, a piece of lamb's wool, or three 4-in-square pieces of gauze may be used. The posterior pack is meant to fill the nasopharynx. Three long umbilical tapes or three heavy silk sutures are tied around the middle of the posterior packing material.

b. A soft rubber catheter is introduced into the front of the nose, preferably in the side that is bleeding, until the end is visualized in the pharynx. A hemostat grasps the pharyngeal end of the catheter, bringing it out through the mouth. Two of the tapes or sutures are tied to the pharyngeal end protruding from the mouth. Pulling the nasal end then draws the posterior pack into the nasopharynx. Usually, it must be guided into the nasopharynx with a finger. Then the two tapes or sutures at the anterior nares are detached from the catheter and held with a clamp. The third tape or suture is left trailing from the mouth.

c. The posterior pack provides a buttress for the placement of a firm anterior pack in the posterior third of the involved nasal chamber. The anterior pack is layered as described (see sec. B.2.).

d. When the anterior pack is properly positioned, the two umbilical tapes or sutures attached to the posterior pack are tied over a dental roll or a piece of foam rubber placed at the anterior nares, preventing alar necrosis. The tape or suture trailing from the mouth is either taped to the cheek or cut sufficiently short so that it just protrudes into the oropharynx.

3. Alternate method. Control of posterior bleeding can also be effected by introducing a Foley catheter through the nose, inflating the balloon when it is in the nasopharynx, and then drawing it up into the posterior choanae.


a. Daily monitoring of hematocrit and arterial blood gases is essential.

b. Oxygen by mask is administered as needed.

c. An antibiotic is required (ampicillin).

d. Pain medication must be titrated carefully to avoid undue respiratory depression.

e. Anteroposterior packing is always uncomfortable. Especially in the elderly, sedation reduces discomfort and cardiac stress must be balanced by a concern for hypotension from blood loss and respiratory depression from nasal obstruction and associated pharyngeal and palatal edema.

f. The packing is usually removed in 5 days. The patient is prepared beforehand for repacking in the operating room, in case bleeding recurs. The tapes or sutures tied over the dental roll are cut and secured with a clamp. The anterior pack is carefully removed. Next, the tape or suture in the mouth is secured, the two tapes or sutures at the anterior nares
released, and the tape or suture in the mouth pulled to deliver the posterior pack. There is usually a small general ooze after removal. If there is brisk bleeding, repacking should be performed in the operating room. However, bleeding following removal of an initially successful anteroposterior pack usually indicates the need for appropriate vessel ligation.

5. **Follow-up after discharge.** Careful follow-up after discharge is indicated to lyse synechiae as they appear.

D. **Arterial ligation.** If bleeding is not controlled with an anteroposterior pack, if there is rebleeding after the removal of packing, or if the patient is unable to tolerate the morbidity of the packing, or if the patient is unable to tolerate the morbidity of the packing, the arteries supplying the nose can be ligated. This procedure is especially indicated in elderly individuals and those patients with chronic obstructive lung disease who cannot tolerate hypoxia induced by nasal obstruction. Ligation may involve one or more of the following arteries.

1. **The internal maxillary artery** and its branches are ligated in the pterygomaxillary fossa via a transantral approach. Ligation first requires a Caldwell-Luc procedure and then access to the pterygomaxillary fossa via the posterior antral wall.

2. In poor-risk patients, ligation of the **external carotid** using local anesthesia is often the surgical procedure of choice.

3. The **anterior and posterior ethmoid arteries** are ligated as they pass from the orbit to the ethmoid labyrinth.

E. **Embolization.** Use of angiography and embolization of the arteries is also a means of controlling troublesome recurrent epistaxis. This procedure should be considered in those patients who would be poor surgical candidates after routine measures have been tried. The very real possibility of inadvertent intracranial embolization and the cerebrovascular sequelae must be weighed against the benefits of this procedure and should be performed only by those skilled in this technique.

F. For hereditary hemorrhagic telangiectasia, a **septal dermoplasty** may be required. The mucosa of the septum and floor of the nose is removed surgically, and a skin graft is placed. Progesterone has also been shown to be effective.

**Nasal Fractures**

**Nasal fractures** are frequent. The prominent position of the nose makes it susceptible to isolated injury from birth onward.

I. **Anatomy.** The upper third of the nose has bony support; the lower two-thirds, cartilaginous support. Superiorly, the nasal bones sit laterally on the nasal process of the maxilla and are joined above by the nasal process of the frontal bone. The paired upper and lower lateral cartilages provide support for the lower part of the nose. The nasal septum is cartilaginous (quadrilateral cartilage) anteriorly and bony (perpendicular plate of the ethmoid and vomer) superiorly and posteriorly.
II. Diagnosis

A. A detailed history is necessary. Included should be information about the date and time of the trauma, type of injury, epistaxis, prior nasal injuries or surgery, possible CSF rhinorrhea, and nasal obstruction. One must consider more extensive facial injuries (e.g., ethmoid, orbital, maxillary, and nasolacrimal apparatus). The patient should be asked about loss of consciousness as a consequence of intracranial injury.

B. Examination and palpation of the external nose can disclose deviation or depression, movement, degree of edema, ecchymoses, localized tenderness, crepitation, and subcutaneous emphysema. Examination of the inside of the nose may reveal blood or mucosal lacerations. The lining of the nose is closely adherent to the bony or cartilaginous framework; there will seldom be a fracture without blood, ecchymoses, or a mucosal laceration. This examination is easier if the mucosa is shrunk by packing the nose with 4-5% cocaine solution (or 4% lidocaine and 1:100,000 epinephrine) on cottonoid strips.

C. X rays are frequently obtained when there is a question of a nasal fracture. They are seldom helpful because nasal bony injury often heal by fibrosis, leaving persistent radiologic evidence of an earlier fracture. Moreover, cartilaginous injuries cannot be visualized by x ray. The history and examination are the best methods of ascertaining the extent of the injury, although x rays may be helpful in delineating associated bony injuries.

D. Reexamination. If there is uncertainty, the patient should be asked to return in 2 or 3 days for reexamination, as the associated swelling subsides. This is especially important in children, recognizing that they can be difficult to examine, and an undiscovered fracture may result in distortion of the nose with growth. It may be necessary to examine the child under general anesthesia.

III. Management

A. Decision to reduce the fracture depends on an evaluation of both appearance and function. A boxer or football player is likely to have little concern about a slight deviation. An aesthetically perfect initial reduction may not be permanent due to contracture during healing. Most people cannot tolerate nasal obstruction or the loss of smell. For them a reduction is indicated.

B. Timing of reduction. Injuries seen shortly after trauma (1-3 hours) and without associated swelling may be reduced immediately. If the nose is very swollen, reduction should be performed 5-7 days after the injury. In children, an earlier reduction (2-3 days) may be necessary since healing is rapid. If there is a delay of more than 2 weeks in the adult, the
injury should be given 6 months to heal and then correction should be performed using standard rhinoplasty techniques.

C. Anesthesia. In some cases, sedation before beginning the anesthetic infiltration is helpful. In the anxious adult, 5 mg of diazepam given slowly intravenously is usually sufficient. In children, general anesthesia may be necessary. The techniques for internal and external local anesthesia follow.

1. Internal. The nose should always be packed for topical anesthesia; 4-5% cocaine solution on cottonoid strips (well wrung out) is introduced into each nostril and left in place for 5-10 minutes. It is sometimes helpful to repeat this procedure, since the anesthesia from the first packing can allow more complete packing the second time. The smallest possible amount of cocaine should be used for adequate anesthesia in order to prevent its adverse effects. Although 4 mL of 5% solution (200 mg) is often considered the maximum amount tolerated, dosage varies greatly among patients. The initial symptoms of cocaine toxicity involve the central nervous system. These symptoms include excitation and anxiousness, followed by an increasing respiratory rate and later respiratory collapse. The treatment of cocaine toxicity is by the intravenous administration of diazepam, 5-15 mg, or a short-acting barbiturate. A mixture of 4% lidocaine and 1:100.000 epinephrine can be substituted for the cocaine.

a. Two percent lidocaine with 1:100.000 epinephrine should be injected at the infraorbital foramen on each side. The foramen is palpable slightly medial to the midline of the lower orbital rim. Then, via an external and intranasal approach, the anesthetic should be infiltrated along each side of the nose from just above the medial canthus to the upper lip. Finally, one should infiltrate across the dorsum of the nose at the level of the medial canthi and across the upper lip. The anesthetic should be given 5-10 minutes to take effect. An initial wheal of local anesthetic should be injected to test the patient's sensitivity to epinephrine. Occasionally, an initial tachycardia will occur and subside.

b. If there is great nasal discomfort, one can additionally inject anesthetic through the greater palatine foramen and pterygopalatine fossa. The greater palatine foramen is on the lateral hard palate, just anterior to the junction with the soft palate. It is just medial to the anterior aspect of the third molar and can be palpated as a slight depression anterior to the hamulus. The distal 2.0 cm of the needle should be bent and advanced into the canal. If the needle is advanced too far, it can enter the orbit. An injection into the pterygopalatine fossa often slows or temporarily controls epistaxis.

2. External

a. Two percent lidocaine with 1:100.000 epinephrine should be injected at the infraorbital foramen on each side. The foramen is palpable slightly medial to the midline of the lower orbital rim. Then, via an external and intranasal approach, the anesthetic should be infiltrated along each side of the nose from just above the medial canthus to the upper lip. Finally, one should infiltrate across the dorsum of the nose at the level of the medial canthi and across the upper lip. The anesthetic should be given 5-10 minutes to take effect. An initial wheal of local anesthetic should be injected to test the patient's sensitivity to epinephrine. Occasionally, an initial tachycardia will occur and subside.

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D. Method

1. External reduction. The patient should bring recent photographs for comparison. The reduction is guided primarily by palpation externally and by intranasal inspection. If the nasal pyramid has shifted, firm pressure and intranasal elevation on that side with restore a more normal configuration. Depressed fragments should be elevated. A scalpel handle or nasal elevator is useful. If the entire nasal pyramid is displaced, an Asch forceps should be used to distract the fracture sites by placing one arm of the forceps in each nasal passage, pulling
the nose away from the face, and then shifting it into an anatomic position. A large Kelly forceps with rubber tubing around each jaw can be used in place of an Asch forceps.

2. Septal reduction. After nasal reduction the septum often returns to the midline, but its position must be assessed. A light nasal pack should be placed to assist in support and to prevent hematoma formation.

3. Suturing of open wounds. A nasal fracture is often an open fracture since the mucosa is frequently torn. If the external skin is torn as well, it should be carefully sutured without sacrificing tissue. The excellent blood supply in the nose usually facilitates the healing of even severely traumatized tissue, but if there is an open injury, a broad-spectrum antibiotic should be considered.

4. Splinting and packing. The nasal pyramid is usually stable after reduction. There is little normal motion or distraction by muscles. An external splint is often applied, however, to protect the nose and to remind the patient to be cautious. It can be formed of soft metal or plastic. Tape is placed underneath the splint for padding and support. If the fragments collapse, internal support from packing the nasal chambers usually maintains their positions (see Epistaxis, sec. III.B.).

5. Postreduction instructions to the patient

a. After the injury and again after the reduction, the patient should be instructed to:

   Sleep with the head elevated.
   Apply ice for the first 12 hours.
   Use a humidifier at night.
   Sneeze through the mouth.
   Refrain from blowing the nose.
   Use lubricating drops (normal saline every 2-4 hours as needed).
   Refrain from vigorous exercise.

b. The patient should always be cautioned that it will take several weeks for the obstruction to decrease and that they can expect some infraorbital discoloration. Even an ideal reduction can be altered by scar contracture and may require a subsequent cosmetic procedure.

6. Antibiotic prophylaxis. If the nasal mucosa is opened, an antibiotic (eg, ampicillin) should be given to diminish the possibility of secondary infection.

7. Patient follow-up. There should be careful follow-up to prevent the formation of synechiae, which are lysed as they appear. Initial follow-up examination should be 1-2 weeks after the injury, with repeat visits in 1-2 months.

Oropharyngeal Conduit

I. Peritonsillar abscess, also called quinsy, is a loculation of pus in the potential space surrounding the tonsil. It develops as an infection in a peripheral tonsillar crypt that penetrates the capsule, entering the connective tissue space between the capsule and the
superior constrictor muscle. Another pathological mechanism can be an inflammation of the minor salivary glands (Weber's glands) located primarily at the superior tonsillar pole. The abscess occurs due to lack of drainage and location of pus. In this case, the tonsil may not be the primary cause of the infection. This may account for cases of peritonsillar abscesses in patients who have had their tonsils removed. The infectious process generally remains localized to the peritonsillar area, but may break through the superior constrictor, gaining access to the deep spaces of the neck.

Peritonsillar abscess may follow an untreated or inadequately treated episode of acute follicular tonsillitis, occurring most often in adolescents or patients in their early twenties and affecting both sexes equally. The abscesses may be bilateral (7% of patients). Approximately 5% have had a prior episode. In many instances, a history of recurrent tonsillitis is not obtained. Approximately 70% of the abscesses are located in the superior pole of the tonsil, 19% are in the midtonsillar area, and 10% are either in combined areas or in the lower pole.

A. Signs and symptoms. A 3- to 7-day history of an extremely severe sore throat with associated dysphagia, odynophagia, trismus, and decreased oral intake is frequent. Speech is best characterized as being muffled of "hot potato" in quality. Usually, the patient's temperature at presentation is between 99 and 100°F. Except in the very young, airway obstruction is infrequent.

B. Examination reveals moderate to severe trismus, sometimes compromising an adequate intraoral examination. Copious mucus may cover intraoral structures. The oropharynx is inflamed, and the infected tonsil is swollen and usually displaced inferomedially, with similar displacement of the soft palate. Palpation of the area sometimes reveals fluctuance.

C. Diagnostic studies should include a complete blood count (CBC) with differential, Monospot testing, Gram staining, and aerobic and anaerobic cultures of aspirated material. Patients previously treated with antibiotics commonly have sterile cultures. If careful anaerobic cultures are performed, a significant percentage of these negative cultures will show anaerobic organisms. In untreated cases, group A beta-hemolytic streptococci have been isolated in approximately 25-50% of patients. Gram-negative organisms have also been isolated from peritonsillar abscesses and may play a major role in a chronic abscess.

D. Differential diagnosis

1. Acute follicular tonsillitis with peritonsillar cellulitis clinically may appear exactly as a peritonsillar abscess. The failure to aspirate pus differentiates the two. The therapy is similar.

2. Infectious mononucleosis appears as diffuse tonsillitis, usually without peritonsillar swelling. Increased adenopathy, a positive Monospot test, and atypical lymphocytes in a peripheral smear are often present. Petechiae are frequently seen on the soft palate.

3. Traumatic aneurysms of the internal carotid artery have no associated signs of infection. A pulsatile unilateral swelling, however, may appear in the region of the peritonsillar space.
4. **Parapharyngeal space infections** appear as an acute infectious process but often have significant associated upper cervical swelling and tenderness. The lateral pharyngeal wall is more edematous than the area around the tonsil. This process is also unilateral.

5. **Parapharyngeal or tonsillar tumors** present with local pain and unilateral swelling, with either an intact or a necrotic mucosa. There are, however, no signs of acute infection (fever or elevated white blood cell count).

6. **Diphtheria** presents with diffuse pharyngitis after first involving the posterior pharyngeal wall. The tonsils are often covered with a dirty gray pseudomembrane (see Chap 4, III.B.), which bleeds easily on manipulation.

E. **Management**

1. **Aspiration.** Cooperative adults who are not severely ill and are not developing complications can be managed with aspiration of the peritonsillar abscess and antibiotic therapy. Aspiration may be performed with an 18-gauge spinal needle inserted above the tonsil at the level of the junction of the uvula and the soft palate. Topical anesthesia (eg, benzocaine or lidocaine) can be used. Infiltrative anesthesia is usually ineffective due to the alkaline pH caused by the abscess. After aspiration the patient is placed on Augmentin 500 mg q8h because of the likelihood of a mixed bacterial flora and followed closed until the episode resolves. Should there be reaccumulation of pus or evidence of progression, such as increased trismus, further difficulty in handling secretions, or inadequate oral intake, the patient should be hospitalized, incision and drainage performed, and intravenous antibiotics administered. A negative aspirate may signify either disease of limited extent (eg, peritonsillar cellulitis) or a failure to enter the abscess cavity.

2. **Incision and drainage.** When an abscess is present, incision and drainage in a hospital setting are indicated in patients not responding to aspiration, in children in whom aspiration under local anesthesia would prove difficult, or in patients reaccumulating pus after adequate aspiration and antibiotic therapy. In an adult, after adequate topical anesthesia, a small incision is made through the mucosa at the level above the tonsil, near the junction of the uvula and soft palate. The coagulation status of the patient must be known because of potential bleeding. Using a blunt hemostat, the incision is spread until pus is encountered and is sufficiently opened to provide adequate drainage. This procedure should be performed with the patient in a sitting position. A similar procedure can be performed under general anesthesia if the patient is not a candidate for local drainage. In this situation, the patient is intubated and placed in the Trendelenburg position to prevent aspiration of pus. Acute tonsillectomy is usually performed if drainage requires general anesthesia.

3. **Acute tonsillectomy.** Tonsillectomy should be considered when the adequacy of incision and drainage is questioned on the basis of the clinical response. Inferior pole abscesses tend to reaccumulate following aspiration and are additionally difficult to evacuate with incision and drainage. The procedure is performed with general anesthesia, the patient having been intubated to prevent aspiration. There is controversy as to whether the uninvolved tonsil should be removed at the time of abscess tonsillectomy.
4. **Antibiotic therapy.** Unless the results of Gram staining indicate otherwise, the patient should be treated from the outset with penicillin (1,000,000 units IV q4-6h) or a first-generation cephalosporin. If the patient is allergic to penicillin, cephalosporin should be avoided, and clindamycin (600 mg IV q8h) should be used. Patients undergoing quinsy tonsillectomy should receive intravenous antibiotics 12-24 hours prior to the procedure, ensuring adequate blood levels and preventing bacteremia. Antibiotics should be continued for 10-14 days after surgical therapy.

5. **Instruction.** If the patient undergoes an acute tonsillectomy, he or she should receive routine instruction as to soft foods (approximately 1 week), pain medication (acetaminophen or codeine, 30-60 mg q4h), rest for 5-7 days, and observation for bleeding, which can occur 3 to 10 days postoperatively (see Chap 4, V.A.). Bleeding must be reported immediately.

**F. Complications**

1. **Sepsis** can result from regional and distant seeding from the abscess. Septicemia occurs from progressive pharyngeal cellulitis or spread to the pterygomaxillary space (see Chap 4, IV.A.3.).

2. **Laryngeal obstruction**

3. **Dissection to deep fascial spaces**

4. **Pneumonia**

5. **Posttonsillectomy haemorrhage.** The exact incidence of hemorrhage following tonsillectomy is unknown, but is though to be between 1 and 5%. It may reflect a secondary infection in the tonsillar bed and, as such, the recommendation for postoperative antibiotics. The most common times for posttonsillectomy hemorrhage are during the first 24 hours and 7-10 days following the procedure, when the eschar separates from the bed. It must be emphasized, however, that hemorrhage can occur at any time after tonsillectomy until the tonsillar bed is well healed.

a. **Management.** Any patient with posttonsillectomy hemorrhage should be thoroughly examined without delay. Unless the bleeding is immediately life-threatening, however, the examination should be deferred until an otolaryngologist is present, especially in a marginally tolerant patient and certainly in a child. This is essential if exacerbation of the bleeding and airway compromise are to be avoided.

(1) **Routine examination and management of light to moderate bleeding**

(a) **Equipment needed.** Proper lighting, suction equipment with a tonsil suction tip, and instruments to handle a potential crisis are key. These instruments include tongue blades, long forceps and hemostats, suture material (eg, 2-0 or 3-0 chromic catgut on a taper needle), and a tonsillar gauze sponges. Intubation equipment should always be available.
(b) Anesthesia. Topical anesthesia, such as 4% lidocaine, can be used to facilitate the examination.

(c) Technique. In the cooperative patient, the bleeding site can usually be identified after careful removal of the clot by forceps or suction. Most often the bleeding is slow and a large clot covers the tonsillar bed. Patients bleeding slowly or intermittently frequently swallow most of the blood and may not have outward signs of significant hemorrhage. Occasionally, no active bleeding is observed at the time of initial examination, but a clot is almost always present, denoting the specific site of the hemorrhage. The bleeding can usually be controlled by cauterization using silver nitrate. If silver nitrate is not effective, a solution of epinephrine and tannic acid, placed on a tonsil sponge and held for 2 minutes with pressure, will control most hemorrhages. More active arterial hemorrhages, however, may require either electrocautery or suture ligation of the involved bleeding vessel; both procedures are best done in the operating room.

(2) Management of severe hemorrhage. The most important consideration in managing severe posttonsillectomy hemorrhage is ensuring that the airway is secure. The vast majority of patients dying from posttonsillectomy bleeding do so not from blood loss but from aspiration and secondary obstruction. With a severe hemorrhage, control of the airway must take precedence over any manipulation of the tonsillar fossa. Intubation or tracheotomy is performed to secure the airway, and a firm pack is placed at the site to control blood loss until an operating room is readied. Most children with significant hemorrhaging need immediate operative management. When time permits, a hematocrit, blood typing, and cross-matching should be performed.

II. Traumatic hemorrhage. Posttraumatic oropharyngeal bleeding occurs most often in young children who fall on objects they have put in their mouths.

A. Management of light to moderate bleeding. Bleeding from small vessels usually subsides spontaneously, but if it does not, the bleeding can be controlled by simple ligatures. Before ligation, the area should always be carefully inspected for the presence of a residual foreign body.

B. Management of severe bleeding. Because of the proximity of several large vessels to the oropharyngeal area, including the internal carotid artery, traumatic injury may result in heavy bleeding. With severe bleeding, the airway must be protected; if immediate control of the hemorrhage is not possible, intubation or tracheotomy should precede aggressive packing of the pharynx. Whenever possible, the patient should be stabilized with fluid replacement prior to definitive operative control of the bleeding site.

C. Follow-up for possible complications. The most dreaded complication of "pencil injury" is thrombosis of the internal carotid artery and its intracranial branches. Neurologic deficits and even death have been reported following what first appeared to be minor trauma to the palate or tonsillar region, secondary to this injury. For this reason, some physicians recommend hospitalization of a patient with injuries caused by sharp objects for a period of 48-72 hours of close observation. If neurologic deficits become apparent if the injury is in the immediate area of the carotid artery, arteriography should be performed.
III. Caustic ingestion followed by corrosive esophagitis remains one of the more serious and controversial problems facing physicians who deal with the aerodigestive tract. Because of the controversies in management, the section that follows should be used only as a general guide and not as a definitive course of treatment. Caustic ingestion followed by corrosive esophagitis occurs most frequently in children under the age of 10 years. Safety caps and public education have done much to reduce the incidence in children, yet these measures have not served the adult population similarly. A history of attempted suicide and alcoholism are frequent associations in the adult group.

A. Types of corrosive esophagitis injuries. Corrosive esophagitis is a burn of variable depth within the esophageal wall. Ingestion of acids produces an injury that differs from that produced by bases. Caustics such as sodium hydroxide and potassium hydroxide produce liquefaction necrosis and may penetrate deeply, causing full-thickness burns, with acute perforation. Acids produce coagulation necrosis. This type of burn is generally not as deep as that produced by an equivalent concentration of base. Acid ingestions are, however, more prone to secondary perforation and stricture formation. Chlorates (bleaches) constitute a third category of caustics with the potential to cause a burn resulting in stricture. They generally cause much milder injury than do strong alkalis and acids. Ammonia usually produces burns that are equivalent in severity to those of the chlorates. The aforementioned are the most frequently ingested caustics.

B. Stages of injury. There are three stages in the esophageal reaction to a caustic injury.

1. The acute or inflammatory phase begins immediately and lasts for approximately 2 weeks.

2. The intermediate phase follows the acute phase and lasts for 8 weeks. During this phase, cicatrix with organization takes place. Eighty percent of esophageal strictures are evidenced within 8 weeks after caustic ingestion.

3. The third phase of injury is that of chronic obstruction secondary to stricture.

C. Signs and symptoms. The history may indicate the ingestion of a caustic. Suspected cases should be treated until more definitive clinical data are obtained. If possible, the known or suspected caustic agent and container revealing the chemical composition should be brought to the hospital for examination.

D. Examination. Normal findings on physical examination, including inspection of oropharyngeal mucosa, do not rule out serious esophageal damage, thus the need to initially treat and further evaluate all suspected cases. Patients with significant caustic ingestion often present with burns of the lips, oral cavity, hands, and "dribble" burns of clothing and skin. They may be drooling and refuse anything by mouth. More severe cases may manifest signs or symptoms of an acute complication such as laryngeal involvement, mediastinal perforation, cardiovascular collapse, and gastric damage.

E. Diagnosis. In suspected cases of corrosive esophagitis, esophagoscopy should be performed between 24 and 48 hours when the patient's general condition is stable. The
esophagoscope must not be passed further than the upper limit of the first burn for fear of perforation. The visualized area of burn should be described, and the presence or absence of circumferential lesions noted. As an initial procedure, a barium swallow is not indicated. Esophagoscopy should not be performed after 48 hours because of the risk of perforation.

F. Treatment

1. Initial measures

   a. Neutralizing substances. There is good reason to consider keeping the patient without oral intake and to avoid the following measures inasmuch as there is usually a considerable time lapse prior to obtaining management by a physician. The induction of vomiting should be avoided. Gastric lavage is controversial. If the patient will accept liquids by mouth, various neutralizing agents for alkali caustics have been suggested, including milk, water, citrus fruit juices diluted with vinegar, egg whites, butter, and olive or mineral oil. For acids, substances to consider include milk, water, milk of magnesia (1 tsp in 1 cup of water), aluminum hydroxide gel followed by egg whites, butter, and mineral oil. For bleaches, warm water or milk may be used. In instances of severe burns, when there is a danger of perforation, oral intake must be withheld.

   b. Nasogastric intubation. If severe burns are identified on esophagoscopy, passage of a nasogastric tube risks perforation and is somewhat controversial. Swallowing a heavy gauze string, weighted or unweighted, has been recommended to provide localization of the lumen for further dilatation, should it prove necessary.

   c. Antibiotics. Penicillin (1,000,000 units q4-6h IV) is considered by most to be the initial antibiotic of choice. It should be started immediately in a suspected burn. If esophagoscope reveals burns, antibiotics - either IV or PO - should be continued until mucosal healing is complete.

   d. Steroids. If burns are seen on esophagoscopy, steroids (eg, prednisone 40-60 mg initially and tapered over weeks) should be added to the antibiotic regimen. If used initially, they are continued until mucosal healing is complete. There is no general agreement as to whether steroids are clinically beneficial in decreasing the granulomatous response and subsequent stricture formation. When strictures do form while the patient is on steroids, they are generally more pliable and readily dilated, as contrasted with strictures in untreated patients. To be effective, steroids should be started immediately following identification of burns or within 24-48 hours after ingestion of the caustic. The usefulness of steroids is questionable if there is evidence of complications such as perforation and mediastinitis. An extra measure of caution must be taken when endoscoping a patient on steroids.

   e. Oral intake of liquids initially is permitted when the patient is swallowing saliva and when there is little risk of perforation. Solids should probably be avoided until healing is complete.
2. Subacute and chronic measures

a. **Esophagoscopy** should be performed 2-3 weeks after antibiotic therapy is begun. At this time steroids and antibiotics may be discontinued only if mucosal healing is complete.

b. **Barium swallow** is not a substitute for esophagoscopy. Since most strictures form within 8 weeks, patients should be evaluated at 2- to 3-week intervals with barium studies that may have to be repeated during the next 2 months.

c. **Bouginage.** If the barium study suggests narrowing, esophagoscopy should again be performed to assess the lumen size. If there is evidence of stricture formation, bouginage with dilatation of the esophagus are discontinued. Bougies should be passed carefully to avoid false passages and a perforation. The frequency of dilatation depends on the ability of the esophageal lumen to retain patency after bouginage. Difficult strictures and strictures in children may require a retrograde dilatation following gastrostomy.

3. Surgical intervention

a. **Acute measures.** Acute surgical removal of an extensively burned esophagus may be required. Segments of colon, stomach, or jejunum can be used to effect a conduit from the pharynx to small bowel, as needed. In selected cases, free tissue transfer of intestine with microvascular anastomosis is most efficacious.

b. **Chronic measures.** When chronic dilatation fails to achieve an adequate food passage or when it runs a high risk of perforation, esophageal removal and replacement has to be considered.