Chapter 33. Otolaryngology

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Emergencies

Croup

Croup is a syndrome characterized by inspiratory stridor, most often caused by parainfluenza virus. It is most likely to occur in children under the age of 2 years, in late fall or early winter epidemics (Strome et al, 1985). The trachea is inflamed below the glottis, usually sparing the lungs, distal bronchi, and the supraglottic regions.

Children with *croup* usually develop fever, a barking cough, *inspiratory stridor,* and hoarseness over a 24-hour period. Physical findings include *inspiratory* (and occasionally expiratory) *stridor* and *tachypnea.* The diagnosis is made clinically, but a lateral neck radiograph may help to differentiate *croup* from *supraglottitis* or *foreign body aspiration.* Blood gases should always be obtained if signs of respiratory distress are present.

The most important treatment decision is whether or not to hospitalize the child. Generally, children with a respiratory rate under 40 per minute can be managed on an outpatient basis if the parents are reliable and if the child is able to maintain hydration. Relief may be obtained by using a hot shower to generate humidification. A cool mist ultrasonic humidifier should be also run constantly for several days until symptoms abate.

Patients who are hospitalized should receive intravenous fluid replacement and humidified air. *Racemic epinephrine* (0.25-0.5 mL mixed with 2 mL normal saline by nebulization) may be repeated every 30 to 60 minutes as needed to relieve respiratory distress. The use of *steroids* is controversial because the data are conflicting as to their effectiveness. If steroids are used, the recommended dosage is dexamethasone 0.5 to 1 mg per kg IV or IM as a single treatment. If the above-mentioned treatment fails to result in improvement, intubation or tracheotomy is required.

The decision whether to intubate or to perform a tracheotomy is always difficult. Intubation is less invasive but may be complicated because of the age of the child and the degree of subglottic edema. *Nasotracheal intubation* is preferred, with emergency tracheotomy capability immediately available if the attempt is unsuccessful.

Supraglottitis

Supraglottitis (epiglottitis) is an acute infection of the larynx above the vocal cords, usually caused by bacteria. The majority of cases occur in children under the age of 6 years, and are almost always the result of *Haemophilus influenza* infections. In adults, however, *beta-hemolytic streptococci, pneumococcus,* or *Staphylococcus aureus* may also be causative (Dayal, 1981).

The illness is characterized by the abrupt onset of high fever (38°C) and a severe, often progressive course. Severe throat pain is present, which makes it difficult for the patient to swallow secretions. Respiratory difficulty is an ominous sign and may begin to occur a short time after the onset of symptoms. The rapidity of the progression of symptoms is dependent on the age of the child and the virulence of the organism involved. The diagnosis should be strongly suspected whenever fever, dysphagia, and respiratory difficulty are seen simultaneously.

On physical examination, the patient appears acutely ill, and may have difficulty swallowing secretions. Nuchal rigidity may occasionally be seen, raising the possibility of meningitis. Tachypnea and use of accessory muscles of respiration may be seen. Severe cases may also be accompanied by cyanosis.

The presumptive diagnosis should be made from the history and general appearance of the patient and should be confirmed at the time the airway is secured. Once an airway is in place, laryngeal secretions should be obtained for culture. Blood cultures and serologic antigen studies should also be performed.

Antibiotics should be given to ensure coverage against the relatively common occurrence of ampicillin-resistant *H. influenzae.* Acceptable initial choices include *ampicillin* and *chloramphenicol* or a *third-generation cephalosporin* such as *cephtriaxone.* Therapy should be continued for at least 7 days. In addition to antibiotics, humidified air - usually with low-flow oxygen - should also be administered. The prognosis for recovery is excellent. Virtually all deaths occur because of respiratory embarrassment from inadequate airway management.

Peritonsillar Abscess

Abscesses in the *peritonsillar space* are most commonly a complication of *acute tonsillopharyngitis* and, thus, are almost always due to infection with *group A beta-hemolytic streptococci.* Symptoms of acute pharyngitis progress to severe unilateral throat pain and difficulty swallowing. Severe pain may limit oral intake and may cause drooling from pooled saliva and muffled speech, sometimes referred to as a "hot potato" voice.

Examination usually reveals a patient with fever, mild dehydration, increased oral secretions, and difficulty opening the mouth (trismus). The involved tonsil is swollen and displaced toward the midline and downward. It may be difficult to determine whether swelling is due to an abscess or cellulitis. Palpation of a fluctuant area with a gloved finger is useful to confirm an abscess. Other diagnostic possibilities include infectious mononucleosis, other parapharyngeal space infections, and tronsillar or pharyngeal tumors. Diagnostic tests should include a throat culture or a latex slide test for streptococcus, a culture of any aspirated material, a complete blood count, and a mono spot test.

All children should be admitted to the hospital. Adults who have significant dehydration or who cannot cooperate with outpatient drainage procedures must also be admitted. Others may be managed as outpatients with either needle *aspiration* or outpatient *incision* and *drainage.* After topical anesthesia with benzocaine or lidocaine, an 18-gauge

spinal needle is inserted into the area of fluctuance. The patient should then be placed on oral penicillin, 500 mg QID for 10 to 14 days or until the episode resolves. If symptoms recur, the patient should be hospitalized for incision and drainage and treated with *intravenous antibiotics. Tonsillectomy* is recommended following resolution because of the tendency for recurrence.

Foreign Body Aspiration

Foreign body aspiration is most commonly seen in small children, but may occur at any age. Objects may become lodged in the *pharynx, larynx, trachea,* or *bronchi.* Symptoms depend on the site of the foreign body. *Pharyngeal* locations primarily produce discomfort, whereas *laryngeal* foreign bodies cause total or near total occlusion. *Tracheobronchial* occlusions cause coughing and intermittent or constant stridor, wheezing, and cyanosis. More distal foreign bodies may initially go unnoticed but subsequently result in wheezing, respiratory distress, and pneumonia.

Physical findings are variable and also depend on the location and the severity of respiratory obstruction. Any combination of *tachypnea, cyanosis, stridor,* and *wheezing* may be found. Obstruction of the *right mainstem bronchus* causes decreased breath sounds and reduced chest expansion. A one-way ball valve effect may occur, resulting in hyperexpansion and hyperresonance to percussion.

The chest x-ray film may show signs of local, lobar, or whole lung atelectasis, and if the object is radiopaque, it may also be seen. If sufficient time has elapsed (usually 24 hours or more), pneumonic infiltrates may be present. *Anteroposterior* and *lateral views* of the neck should also be obtained. If the diagnosis is still in doubt, *fluoroscopy* often allows the site of obstruction to be localized.

Management involves maintenance of the airway and removal of the foreign body. Pharyngeal objects can usually be removed with a mirror and forceps. *Laryngoscopy* or *endoscopy,* used under anesthesia, is required for removal at the level of the larynx and beyond. Removal is usually followed by immediate relief of symptoms, unless infection is present.

Head and Neck Trauma and Respiratory Embarrassment

Trauma to the head and neck region may cause respiratory difficulty in a number of ways, including *dislodged dentures, aspiration of blood and mucus, tongue trauma,* or *laceration of the airway.* Patients presenting with facial trauma and respiratory distress must be immediately assessed, suctioned, and given an oral or nasopharyngeal airway. If the patient is still in distress, an *emergency tracheotomy* or *cricothyrotomy* should be performed. Once the airway is stabilized, attention can be directed to other traumatized areas.

Laryngeal trauma will result in local pain and dysphonia. *Laryngoscopy* should be performed to confirm the diagnosis and assess the damage. A *tracheostomy* should be performed if the airway is unstable. *Tracheal trauma* or *separation* may result in similar signs and symptoms, including *subcutaneous emphysema.* In addition to respiratory endoscopy, the

esophagus should also be assessed for concomitant injury. A tracheotomy should be placed as far from the site of the injury as possible, and the tracheal separation repaired surgically.

Epistaxis

Epistaxis, or nosebleed, most commonly originates from the rich capillary network in the anterior septum known as *Kiesselbach's plexus.* In hypertensive and elderly patients, it may occur posteriorly in areas of the nose that are very difficult to visualize. The most common causes of nosebleed are drying of the nasal mucosa and trauma from picking the nose (Kirchner, 1982).

If *manual pressure*, applied to the nasal septum with the head upright for 10 full minutes, does not stop the bleeding, careful examination and identification of the point of bleeding is required. Proper equipment and lighting is an absolute must and should include a *nasal speculum, bayonet forceps,* and *suction capability* (Johnson, 1981). The physician must first determine whether the bleeding is anterior or posterior. If the bleeding site is not readily apparent, nasal vasoconstriction is accomplished with cotton balls impregnated with 5 per cent cocaine solution or a mixture of 4 per cent lidocaine and 1/100.000 epinephrine. After several minutes, the bleeding should be slowed so that identification of the bleeding point is possible. *Cautery* with 25 per cent *trichloroacetic acid* or *silver nitrate* is usually effective in controlling bleeding. Care should be taken to dry the nasal mucosa prior to cauterization. A silver nitrate stick should be applied to the bleeding site for about 20 seconds (Johnson, 1981). Following cauterization, patients should be advised not to blow the nose, to open the mouth when sneezing, to avoid aspirin, to use cool-mist humidifier, and to apply antibiotic ointment or petroleum jelly to the nose several times per day.

If cautery is unsuccessful, half-inch iodoform gauze should be impregnated with petrouleum jelly or antibiotic ointment and inserted into the nose in layers with bayonet forceps. As much packing as possible should be used without deforming the septum, and should be left in place for 2 to 5 days. Patients who have nasal packing in place should generally be treated with antibiotics because of the high likelihood of sinusitis. Pain medication is often necessary. Patients should be cautioned that nasal obstruction often persists for several days after the packing has been removed. An alternative to the use of gauze packing is the use of an *epistaxis balloon,* which is inserted into the nose and filled with air or saline.

If the bleeding originates from a posterior location, packing must be performed differently. A *posterior pack* is fashioned using two or three 4-inch gauze pads with three silk sutures ties around the middle. A soft, rubber catheter is introduced into the nose, grasped by a hemostat, and pulled from the nasopharynx through the mouth. Two of the sutures are tied to the end of the catheter, which is then pulled back through the nose, bringing the posterior pack into the nasopharynx. It is usually necessary to guide the pack into the nasopharynx with a finger. The third suture is left trailing from the mouth. A firm anterior pack is then placed, followed by a rolled gauze pad across the nose, which also secures the posterior pack firmly in place. Epistaxis catheters or Foley catheters may also be used to control bleeding from a posterior site; however, excessive pressure on the nasal ala or columella will cause tissue necrosis.

If bleeding cannot be controlled by any of the above-mentioned methods, or if it is recurrent, a referral for *arterial ligation* must be considered.

The Ear

Signs and Symptoms

Otalgia

Otalgia (ear pain, earache) is a symptom and not a diagnosis. Otalgia can be either primary or secondary. Primary otalgia arises from pathologic conditions of the ear itself. Secondary otalgia originates at periauricular sites or is referred from a distant origin. More than 50 per cent of cases of otalgia originates from a source other than the ear. The intensity of the otalgia is not necessarily proportional in seriousness to the disease causing it; mild otalgia or vague pain may result from laryngeal or esophageal carcinoma, whereas dental caries may cause severe pain.

The cause of pain originating from the external ear and canal will be obvious from inspection in most instances, eg, a furuncle of the external canal or otitis externa. Exceptions include an early neoplasm of the external canal or the neuralgia of herpes zoster oticus. Otalgia of middle ear origin should be readily apparent. Tic-like pain may originate from the geniculate complex of the seventh nerve or the tympanic branch of the ninth nerve. The most common periauricular causes of otalgia include parotitis, lymphadenitis, and temporomandibular joint dysfunction. Periauricular lymphadenitis may arise from lesions in the scalp.

Temporomandibular joint dysfunction is a common cause of otalgia and is often a result of faulty dental occlusion or excessive jaw movement.

Lesions in the area of the palatine, pharyngeal, and lingual tonsils, as well as those adjacent to the tongue, may cause ear pain via the glossopharyngeal nerve. Squamous cell carcinoma of the base of the tongue may be associated with referred otalgia and require palpation and biopsy for diagnosis. Laryngeal, hypopharyngeal, esophageal, and lung lesions may cause secondary otalgia from branches of the vagus nerve.

If the cause of otalgia is not readily apparent from a routine otorhinolaryngologic examination, then office laryngoscopy followed by a chest x-ray study and barium swallow may be required (Paparella, 1980).

Otorrhea

Drainage from the ear is a common otologic complaint. It is important to document the nature of the otorrhea, related symptoms, and possible causative events such as trauma. The most common cause of otorrhea is *cerumen*, which may range from pale yellow to dark brown and from liquid to solid. Profuse bleeding from the ear is rare except in cases of severe trauma or in patients with clotting disorders. Middle ear and mastoid infections, acute perforations, tumors, and external otitis may be associated with mild bleeding. Serous drainage may occur with bleb rupture from bullous myringitis, otitis externa, or external canal

dermatitis. Purulent otorrhea indicates infection. The color may range from yellow to green. A malodorous discharge is associated with tissue necrosis and is usually found in an infected cholesteatoma. A mucoid discharge without odor is an indication of middle ear mucosal disease or eustachian tube dysfunction, or both, which is often of a temporary nature.

Spontaneous cerebrospinal fluid otorrhea is rare. It may accompany a temporal bone fracture or may be secondary to a tumor or surgery. The fluid is clear and the diagnosis may be confirmed by analysis of sugar, protein, sodium, and cells. Cerebrospinal fluid also produces a halo sign when a drop is placed on filter paper. The halo sign is a ring of clear fluid surrounding a circle of blood-stained moisture, produced by the greater diffusion of spinal fluid than blood.

Examination of the Ear

Otoscopy

The most convenient method to illuminate the external canal and tympanic membrane is the diagnostic otoscope. It includes a halogen light source, an air-sealed head, and rubber tubing for pneumatic otoscopy. An open operating head should be available for removal of cerumen. When performing otoscopy, the physician should remember that the bony canal is very tender when manipulated. Prior to any instrumentation of the ear canal, the patient should be warned to prevent a sudden head movement. Infants should be examined while in the parent's arms. A bottle or pacifier may help to distract the infant. Young children will often respond positively to games such as watching the "Tinkerbell" otoscope light, blowing out the light, and looking for "bunnies in the ears". Every effort should be made to avoid a red eardrum produced by the child's crying. To visualize the tympanic membrane, the pinna must be pulled posteriorly and superiorly. The entire annulus should be seen, including the pars flaccida. The largest speculum possible should be used in order to obtain a tight seal for pneumatic otoscopy. A hand-held bulb or tubing in the mouth is used for changing pressure in the external canal. The flexibility of the normal tympanic membrane and middle ear allows the eardrum to move crisply in both directions. A weak tympanic membrane or one affected by negative middle ear pressure moves out with negative pressure, then passively returns without the need of positive pressure. The malleus should be examined for fixation or diminished mobility. With a retracted tympanic membrane, the short process of the malleus is very prominent, while the manubrium becomes more horizontal and foreshortened. The tympanic membrane in serous otitis changes from a shiny gray to amber, sometimes with air bubbles. Pus produces a white color in the tympanic membrane and causes it to bulge and lose landmarks. Blood causes the tympanic membrane to appear blue. Dense white plaques represent tympanosclerosis and indicate healed otitis media. In many normal ears, the incus can be seen shining through the posterosuperior quadrant of the drumhead (Strome, 1985).

Hearing Evaluation

An office hearing evaluation consists of tuning fork tests and clinical speech testing. *Tuning fork tests* help detect abnormal hearing and differentiate conductive loss from sensorineural loss. These tests provide a gross estimate of hearing but are not as accurate as an audiogram. Tuning fork tests are not usually successful in children less than 4 years of age.

Clinical speech testing may be performed in patients over 5 years of age. The whispered voice test is performed by using bisyllabic words of equal stress, such as baseball, airplane, cowboy, railroad, eardrum, ice cream, and hot dog. The opposite ear is masked by using a Báfrány's box or a partially occluded suction tubing. The hand should be held in such a way as to prevent the patient from lip reading. The results of the test should be expressed as normal hearing or as mild to moderate or severe hearing loss. This type of testing is much more accurate than the use of a watch tick. For children, calibrated noise makers are available that give some frequency information. Again, one must be careful to shield the noise maker from the visual field of the child to eliminate visual cues.

Another indirect measure of hearing in a young child is an assessmnet of their speech. If a child by 18 months of age has not said at least one word that is intelligible to an outsider, then hearing impairment should be considered as a possible cause. By age 2 years, a child should be able to put two words together and be understood. By age 3 years, the child should be able to put three-word sentences together.

Audiometric Diagnosis. A hearing evaluation should be an extension of a physical examination and can range in complexity from a simple office evaluation to sophisticated audiometry.

Tuning fork tests using a *512-Hz* tuning fork should always be performed and can define normal from abnormal hearing, conductive loss versus sensorineural loss, and the frequency range of the loss. These tests provide a gross estimate and are not a substitute for an audiogram.

The *Rinne test* is performed by first placing the tuning fork on the mastoid tip, the aligning the prongs next to the meatus and parallel to the ear canal. The patient is asked which position sounds louder. If the air-conducted sound is louder than the bone-conducted sound, then the hearing is either normal or these is a sensorineural loss. If the bone-conducted sound is louder than the air-conducted sound, then there is a conductive loss in that ear.

The *Weber test* evaluates symmetry of hearing by placing the 512-kHz fork on the forehead or central incisors. The patient reports whether the sound is heard loudest in the middle or whether it lateralizes to one ear or the other. If the tone is loudest in the middle, then either the hearing is normal or the hearing loss is symmetrical. Lateralization indicates a conductive hearing loss in that ear or a significant sensorineural loss in the opposite ear.

Basic audiometry involves *pure-tone testing* and *speech reception threshold testing.* In pure-tone testing, a single frequency tone is presented via headphones. The intensity is varied until the tester determines the lowest intensity that is audible. This is repeated in each ear at various frequencies. The test is then repeated using a bone conduction vibrator placed over the mastoid. Air conduction is equal to bone conduction in sensorineural losses and in normal hearing. In conductive hearing loss, bone conduction scores are better than those of air conduction. The results are expressed as decibels of hearing loss with a range of 0 to 100 dB.

The *speech reception threshold* is determined by presenting a list of bisyllabic words at a frequency of 1000 Hz. The intensity of the words is varied until a level is reached at

which the patient can repeat half of the test items.

The speech reception threshold for each ear should approximate the average of the pure tones at 500, 1000, and 2000 Hz (± dB) in each ear.

Speech discrimination tests are used to test the clarity of articulated speech. A list of monosyllabic words is given at 40 dB above the speech reception threshold. The results are reported as a percentage of the words of the list that are repeated correctly. Normal discrimination scores are 90 per cent of above.

There are special audiometric tests to determine whether a hearing loss is caused by a cochlear or retrocochlear lesion (eighth nerve to auditory cortex). Other than auditory brainstem response, tone decay and reflex decay tests are the most commonly used basic audiometric tests for a retrocochlear lesion.

Impedance Audiometry. There are three important components of impedance audiometry. These three components include tympanometry, physical volume test, and acoustic reflex threshold.

Tympanometry is an objective measure of the compliance of the tympanic membrane as a function of mechanically varied air pressures in the external auditory canal. Tympanic membrane mobility reflects pathology of the middle ear space. With eustachian tube obstruction, there is absorption of the static air in the middle ear space by blood vessels. This creates negative air pressure in the middle ear space, followed by a transudation of fluid and retraction of the tympanic membrane. This negative middle ear pressure can be identified by tympanometry. There are five different curves produced by tympanometry. The type A curve is found in patients with normal middle ear function. The curve shows normal middle ear pressures at the point of maximal compliance. The type As curve is characterized by normal middle ear pressure and limited compliance relative to the mobility of the normal tympanic membrane. The s denotes stiffness, which is seen in otosclerosis, scarred tympanic membranes, and some cases of tympanosclerosis.

The Ad type curve shows large changes in compliance with relatively small changes in air pressure. The d indicates disarticulation as seen in discontinuity of the ossicular chain or in large monomeric tympanic membranes.

The type B curve demonstrates little or no compliance with changes in air pressure in the middle ear. This type of curve is seen in instances of middle ear fluid, adhesive otitis media, and perforations of the tympanic mebrane or with a patent ventilating tube in the eardrum.

Finally, the type C curve indicates negative middle ear pressure of -200 mm H_2O. This may or may not indicate the presence of middle ear fluid.

The *physical volume test* will help clarify the etiology responsible for B type tympanograms. A type B tympanogram with volumes larger than 2.0 mL in children is usually indicative of a perforation or patent ventilation tube. Type B tympanograms with a normal volume measurement is indicative of a nonmobile intact tympanic membrane.

The *acoustic reflex threshold* is a measurement of the level at which the stapedial muscle contracts. The individual with normal hearing produces an acoustic reflex with pure tone signals between 70 and 100 dB hearing threshold level (HTL). Because the acoustic reflex is mediated by loudness, it is a sensitive indicator of cochlear pathology. The acoustic reflex test can also be used to confirm the presence of a conductive hearing loss. Another application of the acoustic reflex test is for the detection of retrocochlear pathology.

Auditory Brainstem Response Audiometry. *Auditory brainstem response audiometry* is an objective auditory test that does not require a subjective response from the patient. The aim of the auditory brainstem response audiometry is to record the potentials that arise in the auditory system as a result of sound stimulation. This test can be used to assess or approximate the threshold of hearing in the higher frequencies. *Auditory brainstem response audiometry* is also useful in detecting retrocochlear lesions such as an acoustic neuroma. The patient with multiple sclerosis and hearing loss may also demonstrate an abnormal auditory brainstem response.

Sensorineural Hearing Loss in Children

Sensorineural hearing loss in childhood may go undetected for years, particularly if it is restricted to the high frequencies. This can result in speech and language difficulties that can have a profound effect on intellectual development. It is important to detect these losses at an early age so that corrective measures can be undertaken.

Hearing actually begins in utero. It has been shown clinically that a 3-month-old infant responds preferntially to a tape recording of his mother's voice. The first word is spoken at about 1 year, with two-word sentences being spoken by age 2 years. By age 4 years, the basic steps to normal language acquisition have been completed.

It is important to determine the cause of a hearing loss in children. This begins with a detailed interview with the parents covering the gestational, perinatal, postnatal, and family histories. The gestational history should seek to uncover maternal infection, trauma, immunologic disorders, nutritional disturbances, or endocrine imbalance. Maternal infections that can affect the fetus include rubella, cytomegalovirus, toxoplasmosis, influenza, syphilis, and herpes types 1 and 2. Maternal trauma may take the form of a disturbance in the placent or umbilical cord, irradiation, drug ingestion, maternal alcoholism, and associated nutritional disturbances.

Endocrine disturbances of the mother such as thyrotoxicosis, diabetes, and pseudohypoparathyroidisim may also predispose the fetus to aural damage.

Perinatal events that lead to hypoxia and hearing loss include placenta previa, abruptio placentae, prolonged difficult labor, nuchal or prolapsed cord, and prematurity. Neonatal jaundice and an unconjugated bilirubin of greater than 25 mg per 100 mL may lead to hearing loss. The use of aminoglycosides to treat septicemia or meningitis in an infant with an immature renal system may also cause a hearing loss.

The postnatal history must include a careful questioning of the response of the infant to sound and the onset of vocalization. Postnatal viral infections that cause hearing loss

include adenovirus, chickenpox, Epstein-Barr virus, herpes zoster oticus, influenza, measles, mumps, encephalitis, and viral hepatitis. Of these, *mumps* is the leading cause of acquired unilateral sensorineural hearing loss in children. Bacterial meningitis may cause either a unilateral or bilateral sensorineural hearing loss.

The *family history* is very important since congenital deafness is inherited in 50 per cent of cases. Up to 80 per cent are inherited as an autosomal recessive trait, and 20 per cent as an autosomal dominant trait. The majority of these cases represent single-gene mendelian inheritance and not part of a recognizable syndrome associated with malformations of other organs or body systems. Autosomal dominant hearing loss is usually mild, flat, and progressive when compared with autosomal recessive losses. Only 1 to 3 per cent of genetic deafness is caused by x-linked inheritance.

The *physical examination* should include a thorough inspection of the pinna. The tympanic membranes should be examined with the pneumatic otoscope or the microscope. An effort should be made to uncover a recognized syndrome by using an organ system approach. A search for craniofacial, dental, cardiac, and renal abnormalities should be undertaken. In addition, endocrine dysfunction, neurologic disease, dermal abnormalities, and skeletal dysplasia may be found. Finally, a consideration of metabolic storage disease and chromosome abnormalities completes the evaluation.

A hearing evaluation using auditory brainstem response should be obtained in all high-risk newborn infants.

Medication Ototoxicity

Ototoxic drugs used in clinical practice include aminoglycosides, furosemide, ethacrynic acid, salicylates, cis-platinum, and erythromycin.

The aminoglycoside antibiotics destroy the hair cells and stria vascularis of the inner ear, resulting in irreversible hearing loss. The degree of ototoxicity is increased by noise exposure and use with other ototoxic drugs.

Streptomycin, gentamicin, and tobramycin are primarily vestibulotoxic. Netilmicin is a new synthetic aminoglycoside that is apparently less ototoxic than any of the presently available aminoglycosides. The effect of most aminoglycosides is insidious. Hearing loss may not become apparent until weeks or months after therapy has been discontinued. Early effects can be detected by high-frequency audiometry and electronystagmography. Since aminoglycosides are excreted by the kidney, renal impairment renders a patient much more susceptible to these ototoxic drugs. Aminoglycosides can cross the placental barrier, resulting in hearing loss in unborn children. Monitoring peak and trough blood levels when administering these medications is helpful in preventing toxicity.

Quinine can be the cause of temporary or permanent sensorineural hearing loss. Elderly patients taking quinine for leg cramps may develop tinnitus and hearing loss from this medication. The ingestion of therapeutic doses of quinine by the pregnant woman may cause severe bilateral sensorineural hearing loss in the fetus.

Salicylates cause reversible hearing loss and tinnitus. The salicylates block an enzyme system within the inner ear, resulting in the uncoupling of oxidative phosphorylation within the cochlea. Normal hearing returns 24 to 72 hours after discontinuation of the drug. Ingestion of 6 to 8 grams per day is required to produce toxicity. Hearing loss occurs whenever salicylate serum levels reach 20 mg per cent or above.

Cis-platinum has both auditory and vestibular toxicity. The hearing loss is usually bilateral and appears first at high frequencies (6000 and 8000 Hz). The hearing loss may be asymmetric and may not appear until several days after treatment.

Patients at high risk for ototoxicity from erythromycin include individuals with hepatic or renal failure or those with Legionnaires' disease. The daily dose of erythromycin should not exceed 1.5 grams if the serum creatinine concentration is above 1.8 mg per cent. The otoneurologic changes observed with erythromycin administration are reversible following cessation of therapy (Meyerhoff, 1984).

Chronic Otitis Media With Effusion

Chronic otitis media with effusion develops secondary to eustachian tube obstruction, barotrauma, or radiotherapy. The fluid may be either serous or mucoid. Clinical experience and experimental evidence suggest that a continuum of serous to mucoid fluid is seen. The pathogenesis of serous effusions involves negative pressure within the middle ear, which occurs as the result of mucosal absorption of middle ear gas, which causes transudation of fluid from the blood vessels of the mucoperiosteum. *Serous otitis media* is the most common cause of hearing loss in children. More than 30 per cent of all children have had three or more episodes of otitis by their second birthday. If the effusion persists, secondary infection can develop and results in proliferation and activation of secretory cells in the middle ear.

As thefluid thickens, it is then known as a mucoid effusion. The patient with chronic otitis media with effusion presents with hearing loss and a fullness or pressure in the involved ear. Infants and toddlers with the disorder may present with pulling at the ears, *nocturnal awakening*, and general fussiness.

Physical examination reveals retracted eardrums and fluid, with or without bubbles. With serous fluid, the tympanic membrane is amber, whereas mucoid effusion is associated with a dull-appearing drum but with distinct margins. Pneumatic otoscopy reveals little or no movement. Tuning fork tests and audiometry show a conductive hearing loss that rarely exceeds 40 dB. Management involves the removal or elimination of any precipitating factors, including sinusitis, allergic rhinitis, and obstructing of chronically infected adenoid tissue. Eighty per cent of patients with otitis media with effusion are free of effusion within 2 months of an episode of acute otitis media or upper respiratory infection. If the effusion is still present at 2 months, then a 2-week trial of an antimicrobial agent effective against beta-lactamase-producing bacteria might be of benefit prior to consideration for surgery. Decongestants and antihistamines have not proved to be effective management for effusions; however, they may be helpful in patients with documented nasal allergy. The insertion of pressure-equalizing tubes is indicated if an effusion persists for 3 months or longer. Approximately 80 per cent of patients with pressure equalizing tubes respond after one insertion and require no further therapy. The child with a persistent conductive hearing loss

secondary to otitis media with effusion is at risk for cognitive and language delay. The nasopharynx of adults with a unilateral effusion should be carefully examined for a nasopharyngeal carcinoma (Bluestone, 1988).

Evaluation of Dizziness

The office evaluation of the patient who has a chief complaint of dizziness begins with a detailed history. The history can be conveniently divided into five parts. First is the differentiation of true vertigo from lightheadedness or disequilibrium. True vertigo has a rotational component that can be in the form of objects spinning or turning around the individual, or severe spinning or turning feeling from inside. This is to be differentiated from the lightheadedness, giddiness, or "swimming-type" of sensation in the head. True vertigo is most often associated with a disorder of the vestibular system. Rarely is loss of consciousness associated with a vestibular disorder. Inquires regarding the association of the vertigo with nausea or vomiting are important to ascertain the severity of the vertigo.

The second part involves gathering information about associated phenomena, participating factors, the periodicity of the attacks, and their frequency. Allergies, head injuries, position change, hyperventilation, fatigue, neck injury, and history of seizures are also important events that may be associated with dizziness.

The next part is specific for symptoms of ear disease. Questions about hearing, tinnitus, fullness or stuffiness in the ears, otalgia or discharge from the ears should be asked.

The fourth part attempts to rule out associated brainstem phenomena such as double vision, numbness of the arms, face, legs, or weakness of the arms or legs, difficulty with speech, confusion, or loss of consciousness.

The final part involves questions about high blood pressure, diabetes, or previous ear surgery.

After a thorough history, the physical examination should begin with the recording of vital signs, including blood pressure in the supine, sitting, and standing positions. The pulse should be taken carefully to evaluate arrhythmias. A thorough ear, nose, and throat evaluation is next. Tuning fork evaluation, including the Weber and Rinne tests, should also be performed. The eyes should be carefully examined for nystagmus, and the fundi should be examined carefully for papilledema. A neuro-otologic evaluation should include a careful examination of the cranial nerves, followed by cerebellar testing. Rhomberg testing should be performed as well as tandem walk, heel walk, and toe walk. If there is a positional component to the dizziness, then a test involving rapid position changes should be performed to aid in the search for nystagmus and evidence of benign paroxysmal positional vertigo. Further tests should usually include an audiogram. If there is a vertiginous component to the dizzniess, thenan electronystagmogram should also be considered. Additional tests will be directed by the findings of the history and physical examinations but may include magnetic resonance imaging, computerized tomography, and various blood tests such as FTA-Abs and erythrocyte sedimentation rate.

Electronystagmography

Electronystagmography is an objective study of the vestibular system, based on the principle that the eye is a dipole with a positive charge at the cornea and a negative charge at the retina. Electrodes are placed about the eye to detect any movement of the eye such as might occur with nystagmus. Nystagmus may be either spontaneous or induced by a position change or a caloric stimulus. By comparing one ear with the other, a relative hypofunction can be identified. Other tests within the electronystagmography battery include positional testing, optokinetic testing, pendulum tracing, and spontaneous nystagmus.

Electronystagmography will not provide a diagnosis for vertigo but will help localize the lesion if it is in the vestibular system.

Disorders Associated With Dizziness

Ménière's disease

Ménière's disease is a term for the coexistence of recurrent vertigo, tinnitus, and hearing loss. Although the cause is uncertain, symptoms are produced by distention and pressure buildup in the vestibular and cochlear apparatus of the inner ear. The disease most commonly occurs in women around the age of 50, and often progresses from isolated vertigo to include tinnitus and hearing loss. Although the vertigo tends to lessen over time, the hearing loss tends to worsen because of gradual distortion of the vestibular and cochlear receptor sites.

The episodes of vertigo occur suddenly and without warning and are often accompanied by nausea and vomiting. Dizziness may last from a few minutes up to several hours, but patients often report feeling unsteady for several days. Although most patients have disease on one side only, about one third will eventually have a bilateral condition. The results of examination between attacks are normal, except for hearing loss, if present. During attacks, nystagmus that is directed away from the involved ear may be seen.

No effective cure for Ménière's disease exists. Management of an acute attack of vertigo includes bed rest and antivertiginous medication. Severe attacks may require hospitalization for parenteral fluids and medication. Long-term management strategies to reduce frequency of attacks include cessation of smoking, decreased caffeine consumption, and a low salt diet. Several surgical approaches have been used to treat intractable cases. About 10 per cent of patients eventually require surgery, primarily because of frequent uncontrolled attacks of vertigo. A decompressive shunt procedure performed on the endolymphatic sac resolves the vertigo in two thirds of patients, while allowing the patient to retain reasonable hearing ability. A vestibular nerve section resolves the vertigo in 95 per cent of patients but does not improve the hearing loss. No current operation consistently improves the hearing loss. Confirmation of the effectiveness of these procedures is still in progress.

Vestibular Neuronitis

Vestibular neuronitis primarily affects adults, causing the abrupt onset of severe vertigo, nausea, and vomiting without hearing loss. Although proof is lacking, it is thought to be a viral infection of the labyrinth and is self-limited, usually lasting a few days. Like most patients with acute vertigo, a change of head position markedly exacerbates symptoms. Patients may experience residual vertigo for several weeks.

Examination usually reveals only horizontal nystagmus. Caloric testing shows depression of the vestibular response. Treatment should include antihistamines, bed rest, and dehydration. Progressive positional exercises should be performed once the acute vertigo is gone. The Cawthorne-Cooksey exercises are designed to retrain the eye and body musculature to use vision and proprioceptive signals to compensate for the lost vestibular signals (Table 33-1).

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo is the most common cause of vertigo in the elderly and is thought to be the result of stimulation of the labyrinthine mechanism from displacement of some of the otoconia, which rest atop hair cells in that organ (Slater, 1988). Initially, vertigo may be severe but gradually improves over several weeks to months. Examination reveals nystagmus in the affected head position characterized by a 2 to 20 second latent period and fatigue after 20 to 30 seconds. Since the nystagmus may be subtle and brief, it may not be detectable with the naked eye. The use of Frenzel glasses can help the physician make the diagnosis of this form of nystagmus. Many believe that the condition will resolve more quickly if the patient continually stimulates the vertigo by putting the head into the precipitating position. Patients should be instructed not to avoid the offending head position. Positional exercises are also helpful, presumably by helping the patient adapt to the asymmetrical input from the two labyrinths.

Labyrinthitis

Labyrinthitis is the *most frequent complication of otitis media,* owing to extension of infection within the temporal bone. There are three types of labyrinthitis that include, in order of descending severity, perilabyrinthitis, serous labyrinthitis, and suppurative labyrinthitis. Perilabyrinthitis, or labyrinthine fistula, which may be produced surgically, occurs secondary to bone erosion by a cholesteatoma or develops after a Valsalva maneuver or an explosion. The patient complains of dizziness or hearing loss, or both, particularly if he or she presses against the tragus, manipulates the auricle, or quickly turns the head. A Valsalva-type maneuver may reproduce the dizziness, and a loud noise may also cause vertigo momentarily. A positive fistula test is present in two thirds of the patients with a labyrinthine fistula. The positive fistula test consists of nystagmus and vertigo and is produced when positive and negative pressure is applied to the soft tissue covering the fistula. A strong positive fistula test is always an indication for surgical examination of the labyrinth. When erosion is associated with chronic otitis media, then a radical or modified-radical mastoidectomy is performed.

Table 33-1. Cawthorne-Cooksey Exercises for Patient With Vertigo

May Be Done in Bed, During Acute Phase

Eye movements, first slow, then fast
- Up and down
- Side to side
- Focus on a finger from 10 to 30 cm from face

Head movements, first slow, then fast, then with eyes closed
- Forward and backward
- Side to side

Should Be Done While Sitting

Head movements as above
Shoulder shrugging and circling
Bending forward to pick up objects from the ground

Should Be Done While Standing

Eye and head movements, shoulder shrugging and circling as above
Go from sitting to standing position with eyes open, then closed
Throw small balls from hand to hand above eye level
Throw ball from hand to hand under knee
Change from sitting to standing position, turning around in between

Moving About

Circling around center person who throws large ball to and fro
Walking across room with eyes open, then closed
Walk up and down slope with eyes open, then closed
Walk up and down steps with eyes open, then closed
Perform a game involving stooping and stretching and aiming, such as skittles, bowling, or basketball.

Adapted from Balow, RW: The dizzy patient. Symptomatic treatment of vertigo. Postgrad Med, 73(5):317-324, 1983.

Serous labyrinthitis is due to diffuse intralabyrinthine inflammation without pus formation and is not followed by permanent loss of auditory and vestibular function. Serous labyrinthitis may be secondary to acute or chronic otitis media, with or without cholesteatoma formation. Serous labyrinthitis in acute otitis media is treated with intravenous injection of antibiotics and a myringotomy for drainage. Serous labyrinthitis secondary to chronic otitis media usually requires surgical intervention for drainage of the suppurative process or removal of the cholesteatoma matrix.

Suppurative labyrinthitis is a diffuse intralabyrinthine infection with pus formation and is associated with permanent loss of auditory and vestibular function. Suppurative labyrinthitis

may be secondary to direct extension of the purulent process in the middle ear or from the spread of meningeal inflammation into the labyrinth through the internal auditory canal or, less frequently, the cochlear aqueduct. Clinical symptoms include nausea and vomiting, intense, vertigo, tinnitus, hearing loss, and nystagmus. Treatment consists of intense antibiotic treatment and surgical drainage of the labyrinth.

Herpes Zoster Oticus

Herpes zoster oticus *(Ramsay Hunt syndrome)* is characterized by cutaneous eruptions about the auricle and external ear canal, facial nerve paralysis or palsy, vertigo, and hearing loss. There may be a prodromal period of general malaise and neuralgia prior to the onset of cutaneous eruption. The facial paralysis, hearing loss, and vertigo may occur alone or in various combinations after the onset of pain. The management consists of topical or systemic antibiotic therapy for control of any secondary bacterial infection. Systemic analgesics may also be necessary for pain control. The role of corticosteroids in the management of herpes zoster oticus has not been well established. Acyclovir may be helpful in decreasing the healing time and lessening the pain associated with these lesions. The facial paralysis of herpes zoster oticus is generally more severe than that seen in Bell's palsy and may be persistent. Sensorineural hearing los may also persist following the resolution of the infection.

Otitis Externa

Otitis externa (swimmer's ear) commonly occurs during the summer months. Moisture from frequent swimming in combination with high temperatures creates optimum conditions for growth of bacteria in the external canal. Another common cause of recurrent otitis externa is due to excessive cleaning of the protective cerumen in the canal (Bell, 1985). The bacteria most commonly responsible for otitis are *Pseudomonas, Proteus,* and, occasionally, *Staphylococcus* and *Streptococcus.* The patient first experiences itching that usually progresses to ear pain, occasionally becoming quite severe. In addition, the patient often complains of a plugged sensation that also may be quite bothersome.

The most common physical finding is pain on traction of the external ear. The canal is erythematous and edematous, and may contain whitish exudate and desquamated debris.

Treatment should begin with gentle cleansing and suction of the ear canal. In cooperative patients, this can often be accomplished with the use of a small tuft of cotton on a wire applicator. Instillation of antibiotic-steroid drops (eg, polymyxin B-neomycin-hydrocortisone), four to five times a day, usually results in healing within a few days. Marcy (1985) notes that 2 to 5 per cent acetic acid ear drops are also effective in eliminating the infectious agent. If severe swelling is present, a cotton wick should be inserted so that the drops can penetrate the canal. The wick may then be removed by the patient in 1 or 2 days. If cellulitis is present in the periauricular area, an oral antibiotic such as cephalexin should be prescribed.

An unusual form of external otitis is referred to as necrotizing (or malignant) external otitis. This entity is most often seen in diabetic patients and is due to *Pseudomonas* or *Proteus* infection. It usually involves not only the canal but the surrounding subcutaneous tissues and, often, the bone. Pain is usually more severe and examination may reveal granulation tissue

on the floor of the ear canal at the bony cartilaginous junction. A diabetic or immunosuppressed individual with swimmer's ear deserves careful consideration to exclude this entity. Management requires long-term parenteral antibiotics and judicious debridement in an inpatient setting.

Otomycosis

Individuals especially susceptible to otomycotic external infections include hearing aid users, immunocompromised persons, or those who have undergone open cavity mastoidectomies. The organisms are usually saprophytes rather than pathogens and occur superimposed on an underlying bacterial infection of the external or middle ear. Fungal infections occur more frequently in tropical or subtropical climates and are associated with intense heat and humidity. Among the more commonly seen fungi are the *Aspergillus, Mucor,* yeast-like fungi, dermatophytes, and actinomyces. Itching is the initial and most prominent symptom, followed by fullness, hearing loss, and pain. The ear canals of such patients are mildly erythematous and may have a moist accumulation of debris. The primary management of saprophytic fungal infections is complete cleansing and debridement of the ear canal. This is usually performed under microscopic control with suction or irrigation and instruments, or both. The canal is then wiped with m-cresyl-acetate or 1 per cent thymol and 70% alcohol. After cleansing, the insufflation of 5 per cent iodo-chlorhydroxyquin in boric acid powder is effective in preventing a recurrence. This treatment may have to be repeated at weekly intervals for 1 or 2 weeks.

Furuncles of the External Auditory Canal

Furuncles are staphylococcal infections of the pilosebaceous units of the outer third of the external auditory canal. They present as localized swellings that may become fluctuant and extremely tender. Early lesions are treated with local head and antistaphylococcal antibiotics. Fluctuant lesions should be drained. Narcotics may be necessary for the first 24 to 48 hours for pain control.

External Auditory Canal Foreign Body

Impacted cerumen is one of the most common ear complaints in family practice. Prevention should always be stressed by discouraging the use of cotton tipped applicators. Even for patients who produce large amounts of cerumen, avoiding insertion of objects smaller than the little finger will usually result in flaking and natural extrusion. Once impaction has occurred, cerumen may be removed by an ear curette or by irrigation. Irrigation should not be used if there is a history of a drainaing ear or a perforation of the tympanic membrane. Use of body temperature water (37°C) will help prevent the occurrence of vertigo (Black, 1986). A large amount of dry cerumen may need to be softened with triethanolamine polypeptide oleate-condensate (Cerumenex) or a similar preparation. Patients with very hard, dry wax may require the use of drops for several days prior to irrigation. A combination of curetting and irrigation may also be employed in difficult cases. If minor trauma results, antibiotic-steroid ear drops should be used for 24 to 48 hours.

Bullous Myringitis

Bullous myringitis is most commonly associated with a viral or mycoplasmal upper respiratory infection. Symptoms are usually limited to mild to moderate ear pain or a sensation of ear fullness. Examination reveals blebs on the surface of the tympanic membrane, which have thin walls and contain fluid. The pain usually subsides in 1 or 2 days. If it is difficult to determine whether an associated otitis media is present, antibiotics should be prescribed followed by re-examination in 48 to 82 hours.

Acute Otitis Media

Acute otitis media is second only to viral upper respiratory infections in prevalence during childhood. Over two thirds of all children experience at least one episode of otitis media during the first 3 years of life. At least 10 per cent of these children have persistent effusions lasting 3 months or more. Most cases occur during the winter and early spring months and are associated with respiratory syncytial virus, influenza virus, and adenovirus infections (Henderson, 1982). The most common causative organism, accounting for about one third of cases, is pneumococcus, followed by *H. influenzae* and *Branhamella catarrhalis* (about 20 per cent each). Streptococci, staphylococci, and viruses are responsible for the remainder. Bodor (1982) reported that when otitis media was accompanied by purulent conjunctivitis, 73 per cent of patients had *H. influenzae* infections.

Symptoms of acute otitis media consist of ear pain, mild to moderate fever, and unilateral hearing loss. Diagnosis depends on careful examination of the tympanic mebrane. Erythema, bulging of the tympanic mebrane with distortion of landmarks, and discoloration from middle ear fluid are diagnostic of acute otitis. Patients who are acutely ill, newborns, and those who have not responded satisfactorily to antibiotic treatment should be considered for tympanocentesis.

Since the organism is not usually known, the choice of antibiotic is empiric. The initial drug of choice is amoxicillin (20 to 50 mg per kg per day). If the patient does not respond to this antibiotic, alternatives include erythromycin (30 to 50 per kg per day) combined with sulfamethoxazole (50 to 100 mg per kg per day) in four divided doses; trimethoprim-sulfisoxazole (8 and 40 mg per kg per day) in two divided doses; cefaclor (40 mg per kg per day); or amoxicillin-K clavulanate (40 mg per kg per day) in three divided doses. Follow-up examination should be performed 2 to 3 weeks later. In patients who have early recurrences on otitis, Carlin and colleagues (1987) noted that patients were more likely to be harboring a different organism than the one that caused the initial infection. Persistence of middle ear fluid is common, but gradual progress toward resolution should be expected. In patients with middle ear effusion, decongestants or another course of antibiotic therapy may be tried. Cantekin and associates (1983) showed in a double-blind, randomized trial that use of decongestants resulted in no better results than giving a placebo. Most clinicians prefer a conservative, noninterventional approach unless the patient continues to have significant symptoms. The decision to place ventilation tubes or to perform tonsillectomy or adenoidectomy, or both, is very subjective and difficult to make. If significant hearing loss or speech delay is present following recurrent otitis media, or if resolution of effusion is delayed longer than 12 weeks, consideration should be given to performing ventilating procedure (Ghory, 1982).

Chronic Otitis Media

Chronic otitis media describes a process whereby irreversible changes have occurred in the tympanic membrane, middle ear, or mastoid. This is a disease that requires surgery, which may vary from the correction of a small central perforation to the removal of an extensive cholesteatoma and drainage of a posterior fossa abscess. (Chronic otitis media can be active, with continuous suppuration, or inactive, representing the sequelae of previous infections.)

The cause of *chronic otitis media* is usually eustachian tube dysfunction or trauma and involves a defect in the structure of the tympanic membrane. The eustachian tube dysfunction may be a result of a cleft palate, obstructing adenoids, tumor, chronic sinusitis, allergic rhinitis, hypothyroidism, smoking, or collagen diseases.

Chronic otitis media can be further divided into tubotympanic disease and attic-antrum disease. Tubotympanic disease may be either a permanent perforation or a persistent tubotympanic mucosal infection. In a persistent perforation, there is a hole in the pars tensa, in which the margin is completely covered with healed epithelium. The ear is usually dry, although it may produce discharge intermittently secondary to water passing through the external meatus or from the spread of mucus through the eustachian tube from nose-blowing or sneezing. When the middle ear is infected, the mucosa is red and edematous and the discharge is mucopurulent and odorless. Hearing loss depends on the size and location of the perforation. Hearing may be normal in a small anterior perforation. Large posterior perforations cause a greater degree of hearing loss. A hearing loss of greater than 30 dB usually indicates ossicular involvement.

The patient should be instructed to avoid getting water in the ear by plugging it with a molded wax plug or with a tightly fitted petrolatum cotton plug. Any pathology of the nose, paranasal sinuses, or nasopharynx should be treated. The ear that produces discharge should be cultured and cleaned, preferably with a microscope and suction or alternatively with the operating head of an otoscope and a cotton-tipped applicator. Appropriate antibiotic drops or powder, or both, may then be used in the ear.

Small, predominantly dry, central perforations that do not interfere with hearing may never need to be closed surgically. However, if the patient wishes to be active in water sports, then repair is preferable. The small-to-medium-sized perforation sometimes closes with a paperpatch procedure (the application of a thin piece of sterile paper to cover the perforation while closure occurs from epithelial migration) performed in the office. The ear should be free of infection for a few months prior to any procedure. Closure of a perforation prevents recurrent infections that cause mucosal changes of the windows and ossicles that may "stiffen" them or lead to disruption of the ossicular chain.

The chronically draining ear without a cholesteatoma exhibits an odorless mucopurulent discharge through a near-total defect in the tympanic membrane. The exposed ossicles are buried in a thick, exuberant, red mucosa. Polyps may be present and should not be removed except under microscopic control. Any pathology of the nose, paranasal sinuses, or nasopharynx must be corrected. Patients with this condition typically do not have otalgia, fever, or vertigo.

Cultures of the discharge should be obtained and appropriate antimicrobial drops and daily middle ear suctioning begun. Alternatively, the patient can be hospitalized and administered a parenteral beta-lactam antipseudomonal drug. The patient who does not respond to this regimen or develops an intratemporal suppurative complication requires surgery of the middle ear and mastoid.

Complications of Otitis Media

Suppurative intracranial complications of otitis media have decreased with the advent of antimicrobial agents. The complications that do occur are more often associated with chronic suppurative otitis media and mastoiditis, with or without a cholesteatoma. The middle ear and mastoid air cell system is adjacent to many important structures, including the sigmoid sinus, the posterior fossa dura, and the middle fossa dura. Suppuration in the middle ear and mastoid may spread to these structures, resulting in the following intracranial complications: meningitis, extradural abscess, subdural empyema, focal encephalitis, brain abscess, lateral sinus thrombosis, and otitic hydrocephalus. The patient who has acute or chronic otitis media and develops one or more of the following signs or symptoms - especially while receiving medical therapy - should be suspected of having a suppurative intracranial complication: *persistent headache, lethargy, malaise, irritability, severe otalgia, onset of fever, nausea,* and *vomiting.* The following would be signs and symptoms demanding an intensive search for an intracranial complication: stiff neck, focal seizures, ataxia, blurred vision, papilledema, diplopia, hemiplegia, aphasia, dysdiadochokinesia, intention tremor, dysmetria, and hemianopsia. Fever is common with acute otitis media, but persistent or recurrent fever, particularly after the administration of appropriate antimicrobial therapy, may be a sign of the spread of the infection.

Meningitis is the *most common* intracranial suppurative complication of acute and chronic otitis media. The most common cause of meningitis is an upper respiratory infection with a simultaneous middle ear infection. When meningitis is suspected, tympanocentesis and myringotomy should be perfomred for identification of the causative organism and establishment of drainage.

Extradural abscess develops from either a cholesteatoma or infection, causing destruction of bone adjacent to the dura. This results in granulation tissue and purulent material collecting between the lateral aspect of the dura and the adjacent temporal bone. Symptoms can include severe earache, low-grade fever, and headache in the temporal region, with deep, local, throbbing pain. Otorrhea may accompany the extradural abscess and is characteristically profuse, creamy, and pulsatile. Computerized tomography may reveal a sizeable extradural abscess. Treatment consists of appropriate antimicrobial therapy and surgical drainage, including a mastoidectomy.

Subdural empyema is a collection of purulent material between the dura externally and the subarachnoid membrane internally. This can occur by direct extension or, more rarely, by thrombophlebitis through venous channels. Patients with empyema are very toxic, are febrile, and have severe headache in the temporoparietal region. Central nervous system findings may include seizures, hemiplegia, dysmetria, belligerent behavior, somnolence, stupor, deviation of the eye, dysphagia, sensory deficits, stiff neck, and a positive Kernig's sign. Hemiplegia and recurrent seizures in a patient with suppurative middle ear and mastoid disease are

indicative of a subdural empyema. A subdural empyema may be confirmed with computerized tomography. Treatment includes intensive intravenous antimicrobial therapy and neurosurgical drainage.

Otogenic abscess of the brain may follow from acute or chronic middle ear and mastoid infections, or follow the development of an adjacent infection such as lateral sinus thrombophlebitis, petrositis, or meningitis. Temporal lobe abscesses are more common than cerebellar abscesses. Signs of invasion of the central nervous system occur about a month after an episode of acute otitis media or an exacerbation of chronic otitis media. Systemic signs include fever and chills. Signs of generalized central nervous system infection may occur and include severe headache, vomiting, drowsiness, seizures, irritability, personality changes, altered levels of consciousness, anorexia, weight loss, and meningismus. In addition, there may be specific signs of temporal or cerebellar involvement, such as vertigo, focal seizures, visual field defects, and nystagmus. Temporal lobe abscesses may be completely silent. Terminal signs include coma, papilledema, or cardiovascular changes. Treatment consists of antimicrobial agents and drainage or resection of the brain abscess, or both, as well as surgical debridement of the primary focus, either the mastoid or adjacent infected tissues.

Lateral sinus thrombophlebitis results from inflammation of the adjacent mastoid. The mastoid infection in contact with the sinus walls produces inflammmation of the adventitia, followed by penetration of the vein. The mural thrombus may become infected and may propagate to occlude the lumen. Clinical signs include high, spiking fevers and chills and signs of increased intracranial pressure, including altered states of consciousness, headache, papilledema, and seizures. Bacteremia is frequent and may result in spread of infected thrombi causing pneumonia and empyema, bone and joint infection, and, less commonly, thyroiditis, endocarditis, and abscess of the kidney. Computerized tomography is an invaluable aid in making the diagnosis and should precede a lumbar puncture. Management includes appropriate use of antimicrobial agents. The sinus should be uncovered and any perisinus abscess drained. The lateral sinus should be opened and the thrombus removed. On rare occasions, the internal jugular vein may have to be ligated.

Tympanic Membrane Perforations

Tympanic membrane perforations may be secondary to infection or trauma or may be iatrogenically produced by a ventilating tube. A perforation due to acute otitis media is pinpoint in size and occurs in the pars tensa region. These perforations heal within 24 hours and are of no clinical significance. They are often so small that tympanometry is required to detect their presence. Rarely, acute otitis media may produce a larger perforation. Traumatic perforations produce a perforation of the pars tensa. These perforations are often large and are accompanied by pain, bleeding, a hollow feeling in the ear, and hearing loss. Patients with these perforations require careful examination and audiometric evaluation to rule out an ossicular chain discontinuity or sensorineural hearing loss. Associated vertigo must be investigated to rule out a problem at the oval or round windows. Uncomplicated pars tensa perforations are treated expectantly. If the perforation does not heal spontaneously in 3 months, then it may require surgical closure. Antibiotic drops are indicated only if there has been contamination by water or debris. Systemic antibiotics are not necessary, but pain medication may be required for the first few days. The patient should be cautioned to avoid

water contamination of the middle ear by using cotton impregnated with petroleum jelly for plugging of the ear. An audiogram should be obtained at the end of treatment to document the return of hearing.

Perforations may also occur secondary to placement of ventilating tubes for middle ear effusions or infections, or both. These perforations occur when large-diameter or "long-term" tubes are used. They occur more commonly in patients who have had a tube in place for 2 years or longer. These perforations can be repaired when the underlying eustachian tube dysfunction has resolved.

Acute Mastoiditis

Acute mastoiditis consists of three stages. The *first stage* involves signs and symptoms consistent with acute otitis media. There are pain, fever, and hearing loss. X-ray studies of the mastoid air cell system shows clouding. If resolution does not occur at this stage, then it may progress to the *second stage, acute mastoiditis* with *periostitis.* At this stage, the infection has spread to the periosteum covering the mastoid process. Patients with acute mastoiditis with periostitis have fever, otalgia and postauricular erythema, tenderness, and slight swelling. The pinna may be displaced inferiorly and anteriorly, with loss of the postauricular crease. X-ray studies again show clouding of the mastoid air cell system. Patients with their condition should be hospitalized and have a tympanocentesis, followed by a myringotomy for drainage and perhaps insertion of a tympanostomy tube. They are placed on appropriate antimicrobial therapy and observed for the first 24 to 48 hours. Most patients improve, but if they do not, a complete simple mastoidectomy is performed.

The most advanced stage of acute mastoiditis is *acute mastoid osteitis.* Patients with this condition present with swelling, redness, and tenderness to touch over the mastoid bone. The pinna is displaced outward and downward, and swelling or sagging of the posterosuperior canal wall is present. Purulent discharge may issue through a tympanic membrane perforation. An occasional patient with acute mastoid osteitis may present with a normal-appearing middle ear and tympanic membrane. In these patients, the middle ear involvement drains through the eustachian tube. Computed tomography shows the haziness and distortion of the mastoid air cell system. There is loss of the sharpness of the shadows of the cellular walls owing to demineralization, atrophy, and ischemia of the bony septa. When this occurs, the process is known as coalescent mastoiditis. Management of acute coalescent mastoiditis involves a complete simple cortical mastoidectomy with an accompanying myringotomy and drainage of the middle ear air cell system. Intravenous antibiotic therapy is also administered.

Congenital and Acquired Cholesteatoma

Keratinizing stratified squamous epithelium within the middle ear or other pneumatized portions of the temporal bone is called a *keratoma* or a *cholesteatoma.* Cholesteatomas may be either congenital or acquired. A *congenital cholesteatoma* represents a congenital cyst of epithelial tissue and appears as a white, cyst-like structure within the middle ear or temporal bone. The most common cholesteatoma is the acquired type, which is secondary to middle ear disease. Seventy-five per cent of the acquired cholesteatomas are located in the attic or posterior superior quadrant. The pathogenesis involves a functional obstruction of the eustachian tube due to constriction rather than dilatation of the tube during swallowing. This

results in impaired ventilation of the middle ear-mastoid air cell system, which in turn causes fluctuating or sustained high-negative middle ear pressure. The tympanic membrane becomes flaccid and eventually collapses onto the ossicles and medial wall of the middle ear. The most flaccid parts are the posterosuperior and pars flaccida areas. A retraction pocket develops that may become adherent to the ossicles or surrounding structures, or both. Cholesteatoma formation can then occur as demonstrated.

The signs and symptoms of cholesteatoma may be completely absent for many years. Recurrent or continuous foul-smelling discharge and progressive hearing loss are the usual presenting signs. Children do not usually complain of hearing loss, tinnitus, or fullness in the ear, particularly if the lesion is unilateral. Otalgia and fever may signify the development of a suppurative intratemporal or intracranial complication. Similarly, facial paralysis, severe vertigo, headache, and vomiting signify a suppurative complication.

Cholesteatomas appear as white, shiny, greasy flakes of debris in the attic or posterosuperior quadrant of the tympanic membrane. They may also be accompanied by polyps and a foul-smelling discharge.

The audiogram usually reveals a conductive hearing loss, although hearing may be normal. A mixed conductive and sensorineural hearing loss may also be present. The sensorineural component may be due to a serous labyrinthitis or a fistula.

Cholesteatomas are managed *surgically*. It is beyond the scope of this writing to describe the various surgical approaches used in the management of cholesteatoma. The goals of surgery are listed from most important to least important as follows: (1) to give a dry, safe ear, (2) preservation of hearing, and (3) improvement of hearing. One or more operations may be required to achieve these results (Bluestone, 1988).

Otosclerosis

Otosclerosis is an inherited autosomal dominant trait with poor penetrance that is much more common in Caucasians, less common in blacks, and rare in orientals. A positive family history is elicited in 50 to 70 per cent of the cases. About 10 per cent of Caucasians develop otosclerosis and 1 per cent become symptomatic. Women present clinically with otosclerosis twice as often as men, and most of the patients present between the ages of 11 and 30 years. Eighty per cent of cases eventually become bilateral. Pregnancy seems to accelerate the process. Patients with otosclerosis present with hearing loss. The otoscopic examination is usually unremarkable, but tuning fork tests usually confirm the diagnosis. A conductive or mixed hearing loss is detected. The conductive component will vary between 10 and 50 dB, depending on the degree of fixation. If there is cochlear involvement, the hearing loss will either be flat or sloping down across all frequencies, or have a "cookie-bite" configuration. There are generally four management options to consider in otosclerosis: (1) Observation, particularly if the hearing loss is mild. (2) The option of the use of a hearing aid to improve the hearing should always be discussed with the patient. (3) Sodium fluoride has been recommended as a medication that might halt or retard the progression of otosclerosis. (4) Surgery is the final option. The aim of surgical management is to restore a mobile mechanism for transmitting sound vibrations to the inner ear. Stapedectomy or stapedotomy is highly successful in the properly prepared and selected patient.

Benign Tumors of the Ear

Benign tumors of the ear include *osteoma*, *exostosis*, *Winkler's disease*, *keratosis obturans*, and *glomus jugulare* tumors.

Osteomas consist of cancellous bone that arises as a pedunculated tumor from either the tympanosquamous suture or the tympanomastoid suture. Symptoms include hearing loss and discomfort. Surgical treatment is only necessary when the lesion is symptoms.

Exostoses are dense compact bone and are the most common tumors of the external auditory canal. They become symptomatic when they cause an accumulation of debris in the canal, resulting in infection or obstruction. The one causative factor is believed to be prolonged swimming in cold salt water.

Winkler's disease consists of a benign nodular painful growth on the helical rim, most often occurring in men. The nodule is tender, preventing some patients from sleeping on the affected side. Treatment consists of cortisone injections. Surgical excision may be required if cortisone does not relieve the pain.

Keratosis obturans is a rare collection or accumulation of large plugs of desquamating squamous epithelium deep within the external auditory canal. This process may cause an erosion of the bony portion of the external auditory canal. It is associated with chronic pulmonary disease, sinusitis, and bronchiectasis. Pain is the presenting complaint. The cause is unknown but is believed to result from faulty migration of squamous epithelium. Treatment consists of periodic debridement of the desquamated squamous epithelium.

Glomus jugulare tumors, also called nonchromaffin perigangliomas, arise from glomus bodies located in the adventitia of the dome of the jugular bulb or along branches of the tympanic plexus. These tumors grow slowly but are destructive by invasion of surrounding structures. They are sometimes multicentric in origin; up to 10 per cent have a definite association with carotid body tumors. The tumor typically is associated with a pulsating tinnitus, followed by hearing loss, and finally, invasion of the tympanic membrane. An isolated facial paralysis may develop, followed by multiple cranial nerve involvement, including nerves IX, X, XII, and XII. Examination in the early stages may reveal a reddish swelling behind the tympanic membrane, which pulsates. Radiologic techniques employed for diagnosis include high-resolution computerized tomography scanning, arteriography, and jugular venography. Unless there are contraindications, glomus tumors should be surgically removed. If there are contraindications to surgery, then radiotherapy may arrest tumor growth.

Acoustic Neuroma

Acoustic neuroma (schwannoma) accounts for approximately *8 per cent* of all brain tumors and *80 per cent* of all posterior fossa tumors. Patients with acoustic neuromas typically present with a gradual, progressive, unilateral sensorineural hearing loss with poor speech discrimination. At first, patients may only notice the accompanying unilateral tinnitus and not the hearing loss. Therefore, any unexplained unilateral, progtressive hearing loss, and tinnitus should raise suspicion of an acoustic neuroma. Approximately 10 per cent of patients with

an acoustic neuroma present with a sudden unilateral sensorineural hearing loss. Any patient who present with an unexplained unilateral sensorineural hearing loss with a duration of 1 month or longer should undergo contrast-enhanced computerized tomography or a magnetic resonance imaging study. Approximately *10 per cent* of patients with acoustic neuroma present with episodic vertigo. More commonly, the vestibular presentation is one of unsteadiness rather than true vertigo. The majority of acoustic neuromas arise from the vestibular division of the eighth cranial nerve. The tumors damage vestibular function slowly enough for compensation of the resulting asymmetry to occur. Acoustic neuromas are usually removed surgically, although poor surgical candidates with small tumors may undergo *gamma knife* radiation therapy. Schwannomas may also arise from other sites, as indicated in the magnetic resonance imaging scan.

Malignant Neoplasms of the Ear

Eighty-five per cent of ear malignancies involve the auricle, 10 per cent involve the external auditory canal, and only 5 per cent involve the middle ear and mastoid. *Otorrhea* and pain are the earliest symptoms. The pain is often intense and out of proportion to the pathologic and clinical findings. Bleeding, fullness in the ear, and conductive hearing loss may also be present. Late findings include perceptive deafness (15 per cent), vertigo (13 per cent), and facial nerve paralysis (13 to 35 per cent). Squamous cell carcinoma constitutes two thirds of the malignancies of the external ear, whereas basal cell carcinoma constitutes the remaining one third. In general, surgical resection provides a better prognosis than radiotherapy. The most common tumor of the middle ear is squamous cell carcinoma. Computerized tomography is useful in delineating the extent of bone destruction in carcinoma of the ear. Early attempts at treatment of cancer of the temporal bone consisted of radical mastoidectomy, followed by radiotherapy, and resulted in a 5-year cure rate of less than 25 per cent. Recent development of temporal bone resection techniques has increased the 5-year cure rate to 44 per cent.

Auricle Trauma

Auricle trauma can be categorized as lacerations, hematoma, burns, and frostbite. Lacerations can range from simple lacerations to complete avulsions and are often associated with multiple trauma. The ear should be carefully cleaned of all foreign debris. Cartilage should not be sutured except to reform the contour of the ear. Perichondrium should be closed using fine absorbable suture. The skin should be approximated using interrupted 6-0 monofilament nylon, and a sterile mastoid dressing should be applied. An avulsed auricle should be repaired in the operating room. The auricle can be preserve in sterile iced saline.

Hematomas are usually secondary to blunt trauma that produces a collection of blood between the perichondrium and the cartilage and presents as a smooth blue mass. Prompt drainage is required to prevent aseptic necrosis of the underlying cartilage. If hematomas or seromas are seen early before clot formation, they may be aspirated with an 18-gauge needle and a pressure dressing applied. If they recur or cannot be aspirated, they should be opened, drained, and compressed. The dressing rolls should be left undisturbed for 1 week and the patient placed on an antistaphylococcal antibiotic.

Emergency management of the burned auricle involves gentle local cleansing, topical antibiotic application, and the avoidance of any pressure on the ear. Late complications include perichondritis and chondritis, which require intravenous antibiotics and drainage if fluctuance develops.

Frostbite of the auricle occurs particularly when the temperature falls below 10°C, which blocks the sensory nerve input, depriving the patient of the warning of impending danger. The ear becomes white and shiny with bulla formation. Rapid rewarming is necessary with compresses at a temperature of 38°C to 42°C. The ear is then treated like a burn by applying antibiotic cream to any breaks in the skin and avoiding any pressure. No debridement should be performed until lack of viability is determined with certainty.

Barotrauma

Barotrauma results from a change in atmospheric pressure while the eustachian tube is occluded. Barotrauma is increasing in frequency because of increased air travel and scuba diving.

The eustachian tube functions as a one-way valve. Air can leave the middle ear passively, but an active process is required for air to enter the middle ear. Airplane ascent produces a decrease in pressure, leading to an increased volume of air. Descent leads to an increase in ambient pressure, which collapses the cartilaginous portion of the eustachian tube.

Factors that favor the development of barotitis include swelling of the nasopharyngeal end of the eustachian tube secondary to an upper respiratory infection or allergy, ignorance of the need to equalize pressure, rapid rate of descent, and sleeping during descent.

With moderate barotrauma, there is vascular engorgement and mild hemorrhage in the tympanic membrane. With more severe barotrauma, hemotympanum or perforation of the tympanic membrane may result. Symptoms vary with the severity of the barotrauma, and may include severe pain, decreased hearing, fullness, low-pitched tinnitus and, occasionally, vertigo.

The condition may be treated by performing a Valsalva maneuver when a sensation of fullness is first noted. Topical and systemic decongestants may be helpful. For hemotympanum, myringotomy is performed only if the individual is a pilot and must immediately return to flying. Perforations are repaired only if they have not healed within 3 months.

Recurrent barotitis should be treated by eliminating any underlying pathology in the nose or sinuses, such as severe septal deviation, hypertrophic lymphoid tissue, allergic rhinitis, and chronic sinusitis. Insertion of ventilation tubes may be necessary for barotitis secondary to flying.

Hearing Loss From Acoustic Energy

Hearing loss from acoustic energy is the most commonly acquired and preventable cause of sensorineural hearing loss. The hearing loss may be secondary to extremely high

levels of acoustic energy, eg, an explosion or chronic noise exposure in excess of 80 dB. At least 10 million people in industry suffer from noise-induced hearing loss. Noise may be recreational, military, environmental, or social in origin and may produce either temporary or permanent hearing loss. Temporary threshold shifts last less than 16 hours and have their greatest effect at 4000 Hz. With continued exposure, the audiogram at 4000 Hz becomes deeper and wider until high-frequency perception is completely lost, then low frequencies are increasingly affected. A 16-hour interval is required between the last noise exposure and the measurement of hearing. For compensation purposes, at least one month should lapse between last exposure and final assessment. There is no age or sex difference in susceptibility to noise-induced hearing loss. When noise is combined with ototoxic drugs, there is more organic damage than either would produce alone. There is no evidence that a person with pre-existing sensorineural hearing loss is more susceptible to noise-induced hearing loss. There is a large amount of individual variation in susceptibility to noise-induced hearing loss.

Other factors that must be considered before attributing a sensorineural hearing loss to noise include presbycusis, ototoxic chemicals and drugs, familial hearing loss, trauma, and chronic otitis media.

There is no treatment that reverses a *permanent* threshold shift. Hearing aid amplification and lip-reading are of benefit. Prevention is the key to reducing the incidence of noise-induced hearing loss. Reduction of the source of the noise would be best but is also the most difficult to achieve. The best ear protection is a combination of earplugs and fluid-sealed muffs. Cotton is not effective as an earplug. A hearing conservation program should be instituted if difficulty in hearing occurs while in a noisy environment, if tinnitus develops after working in noise, or if there is a temporary loss of hearing perceived by the worker.

Temporal Bone Fractures and Labyrinthine Concussion

Temporal bone fractures are described as being either *longitudinal, transverse,* or *mixed.* Longitudinal fractures constitute 80 per cent of temporal bone fractures and result from direct lateral blunt trauma to the skull in the parietal region of the head. The fracture extends from the squamous portion of the temporal bone, along the roof of the external auditory canal. These fractures often disrupt the tympanic membrane, resulting in bleeding from the ear. There may be conductive hearing loss secondary to disruption of the ossicles of the middle ear. Spinal fluid leaks are rare in longitudinal fractures. Vestibular and cochlear function are usually preserved, although mild high-frequency sensorineural hearing loss is sometimes seen secondary to the concussive effect. Facial paralysis is rare and, if present, is often delayed in onset, since it is secondary to trauma and edema instead of interruption of the nerve.

Transverse fractures account for approximately 20 per cent of temporal bone fractures and are usually caused by a severe blow to the occipital portion of the skull. They occur in severely injured patients and result in profound sensorineural hearing loss and total loss of vestibular function. Facial paralysis may be present in up to 50 per cent of the cases and is usually caused by interruption of the facial nerve. Transverse fractures result in a hemotympanum rather than exterior bleeding. Cerebral spinal fluid leaks are frequently seen in transverse fractures and are detected when clear fluid drains from the eustachian tube into the nasopharynx. Labyrinthine concussion is secondary to head injury. The patient complains of mild unsteadiness or lightheadedness, particularly with change of head position.

Audiometric testing reveals a high-frequency hearing loss. The electronystagmogram may show spontaneous or positional nystagmus. Occasionally, the caloric response is hypoactive.

Sudden Sensorineural Hearing Loss

One definition of sudden sensorineural hearing loss is a loss that is greater than 30 dB, in three contiguous frequencies that occurs in less than 3 days. In the majority of patients, the hearing loss occurs suddenly and the cause is not clinically apparent. Approximately half of the patients have some associated imbalance or vertigo. The reported incidence of 1:10.000 persons per year is probably lower than the true incidence, since many people recover before seeking medical attention. The prognosis for recovery is poor in those patients with vertigo or profound hearing loss, or in those who are older than 40 years of age.

Table 33-2. Assessment of Patients with Sudden Hearing Loss

Initial Assessment (Within 2 Weeks of Hearing Loss)

History and otoneurologic examination
Laboratory data
- Complete blood count (CBC)
- Complete erythrocyte sedimentation rate (CESR)
- Glucose
- Glucose tolerance test (TT) or Hb A_{1c} (optional)
- Fluorescent treponemal antibody-absorption test
- Cholesterol level
- Triglyceride level
- Acute and convalescent sera for viral antibody titers (optional)

Audiological evaluation
- Air and bone conduction
- Speech audiometry
- Alternate binaural loudness balance
- Auditory brainstem response
- Upright and recumbent audiograms (for suspected fistulas only)

Hallpike's caloric test with electronystagmography; positional tests, fistula test, or ENG with impedance testing (for suspected fistulas only)

Further Evaluation if Hearing Loss Does Not Return in 1 Month, or if Hearing Loss Progresses

Radiographic views of internal auditory meatus
Computerized tomography scan, with contrast.

The most common cause of idiopathic sensorineural hearing loss is viral cochleitis. Multiple viruses including mumps, influenza B, rubeola, and cytomegalovirus have been found to be responsible for idiopathic sensorineural hearing loss. Partial or complete occlusion of the cochlear vasculature that may occur in Waldenström's macroglobulinemia, polycythemia vera, or sickle cell anemia, or following cardiopulmonaty bypass surgery can result in sudden hearing loss. Cochlear membrane breaks are also potential causes of sudden

hearing loss. Round or oval window fistulas may occur following abrupt compression or decompression of the ears, head injuries, heavy lifting, and straining. Physical findings include fluctuating hearing and/or tinnitus, which may improve overnight and worsen during the day.

The initial work-up should include a history, otologic and neurologic examinations, audiologic testing, and laboratory studies. See Table 33-2 for a list of suggested tests. If hearing loss does not return in 1 month or is progressive, then computerized tomography with contrast or magnetic resonance imaging is obtained.

Patients with moderate hearing loss of presumed viral etiology who have no vestibular symptoms may respond to steroid therapy. Carbogen (5 per cent CO_2 and 95 per cent O_2) has been found to improve perilymphatic oxygen levels. There is evidence to suggest that this treatment may help improve hearing in the speech frequencies.

Finally, patients who have a definitive history of antecedent barotrauma should have an immediate exploration of the middle ear to repair a fistula. Patients with an uncertain diagnosis of a fistula should be placed on bed rest with the head elevated.

Presbycusis

Presbycusis is defined as the effect of aging on the auditory system, characteristically resulting in a bilateral symmetrical neurosensory hearing loss in the frequencies above 2000 Hz, although other patterns occur. At first, conversation is not impaired because the frequencies involved are typically above those of speech, which is 500 to 2000 Hz. As the upper frequencies become involved, the patient typically complains of the inability to understand speech. This lack of understanding is the result of a decreased ability to discriminate consonants, particularly those spoken by women and children. When speech discrimination begins to fail, then conversation becomes more and more difficult, particularly in a group setting. The ability to ignore competing speech also becomes impaired and maintaining communication becomes increasingly more and more difficult, which often results in isolation of the individual. Approximately *one third* of the population over age 65 has a significant hearing impairment. It is difficult to ascertain what portion of hearing impairment is caused by aging of the auditory system and what portion is caused by other traumatic or metabolic factors. Efforts have been made to identify histologic and audiologic correlates. Schuknecht has divided presbycusis into four types: sensory presbycusis, neural presbycusis, strial presbycusis, and cochlear presbycusis. Whatever type of presbycusis is present, the individual can be helped with a properly fitted hearing aid. There are also many less expensive *assistive listening devices* available on the market that may be helpful in certain listening situations. There is current research involving an implantable hearing device which would drive the ossicular chain by electromagnetic means. This may become available in the near future to improve sensorineural hearing loss from many causes.

Facial Nerve Paralysis

Though trauma or surgery can result in facial nerve paralysis, the cause is usually never determined and the condition is referred to as Bell's palsy. Current theories favor a viral etiology (Olsen, 1984). It is relatively common, affecting about one in 60 or 70 persons.

Paralysis occurs abruptly and is complete within 48 hours of onset. About 80 per cent of patients recover complete function within a few weeks or months, but recurrences are seen in about 10 per cent.

Examination reveals partial or complete paralysis in all branches of the seventh nerve. A complete neurologic examination should be performed. Electroneurography performed within the first 2 weeks may help determine the prognosis. Evidence of denervation on electroneurography indicates that a much longer recovery period is expected. Treatment should include protecting and splinting of the eye, massaging facial muscles, and administering corticosteroids.

Corticosteroids (eg, prednisone, 60 to 80 mg daily), given during the first 5 days and tapered over the next 5 days, may help to shorten and lessen the paralysis. If the patient does not seek treatment before 48 hours, the use of prednisone is probably not worthwhile. If complete facial paralysis lasts longer than 2 weeks, electroneurography provides some degree of prognostic information. Surgical decompression of the facial nerve has been performed in patients with severe denervation, but results are inconsistent and controversial.

The Nose and Sinuses

Choanal Atresia

Choanal atresia is a unilateral or bilateral obstruction of the posterior choanae (opening from the posterior of the nose to nasopharynx). The condition occurs in 1 per 5000 to 8000 births, and the obstructing tissue may be membranous only (10 per cent) but is usually osseous (90 per cent). It usually occurs in females and is usually unilateral. During prenatal development, the nasal cavities form as the nasal placodes invaginate posteriorly until they encounter the nasobuccal membrane, which usually attenuates and ruptures around the 6th week of gestation. Theories as to the cause of choanal atresia include persistence of the buccopharyngeal membrane, persistence of the nasobuccal membrane, and misdirection of mesodermal elements in the choanal region.

Neonates are obligate nasal breathers, and acute respiratory distress, which may be life threatening, usually occurs with bilateral atresia. Adaptation to oral breathing may take several weeks. Unilateral atresia can be overlooked until it is diagnosed later in life on the basis of persistent unilateral nasal discharge. There are often associated anomalies, including the CHARGE syndrome (coloboma, cardiac (heart), choanal atresia, retarded growth, genital, and ear).

Diagnosis requires a high index of suspicion. A wisp of cotton or a cold mirror will demonstrate lack of air flow through the nose. A No 6 French catheter does not pass 3 to 4 cm from the nostril, and radiopaque dye reveals the nature of the obstruction. Treatment usually requires emergency airway management, and a small oral airway is effective for a short time. As soon as the infant can safely tolerate general anesthesia, a transnasal, transseptal, or transpalatl approach is employed by the surgeon to open the choanal region.

Allergic Rhinitis

Allergic rhinitis is commonly encountered in family practice and usually begins in patients younger than 20 years of age (Busse, 1983). It may occur seasonally from pollen allergies, or perennially from allergy to house dust, animal dander, or food. Patients usually present with itching of the nose and eyes, sneezing, nasal obstruction, watery nasal discharge, and increased lacrimation.

Examination reveals narrowing of the nasal airway with a pale, purplish hue to the mucosa and a clear, watery discharge. Nasal polyps may also be noted. Diagnosis is usually evident from a careful history of the relationship of symptoms to allergen exposure. A nasal smear for eosinophils may help in differentiating this condition from nonallergic nasal problems. Other helpful diagnostic procedures may include skin tests and radioallergosorbent tests.

Treatment requires a multifactorial approach, including elimination of exposure, desensitization therapy, antihistamines, and nasal instillation of corticosteroids. Recently a nasal preparation of cromolyn has become available that may help to prevent symptoms when used prior to exposure. In severe cases and for episodes that are limited to short periods during the year, a short course of systemic corticosteroids may be very helpful. Topical steroid sprays such as beclomethasone and flunisolide are also helpful for patients whose symptoms have not responded to antihistamines. Concomitant bacterial infection, especially sinusitis, is common.

Nasal Polyposis

Nasal obstruction, partial or complete, may be caused by *nasal polyps.* These polyps are soft, smooth, translucent, lobulated tissue masses that arise from nasal or sinus mucosa. They consist of edematous mucosa and submucosa and may be unilateral or bilateral, single or multiple. They are the most comonly seen intranasal masses, occurring with equal frequency in both sexes and at any age.

Nasal polyps usually occur in association with other specific clinical entities such as allergic disorders, cystic fibrosis, aspiring-induced asthma, chronic sinus infection, and the recently described syndrome of recurrent respiratory disease, azoospermia, and nasal polyposis. There is a particularly high correlation between polyps and allergic rhinitis, with about half of these patients developing significant nasal polyposis. Also, over half of the patients with aspirin intolerance and asthma are noted to have nasal polyposis.

Diagnosis is made on the basis of the history and physical examination. Polyps differ from most other nasal masses because of their pale, wet appearance and because they are mobile, insensitive to pain, and do not bleed. Sinus roentgenograms are indicated to assess the degree of associated sinus disease so that an effective management plan can be undertaken. A sweat test should be performed in any child with polyposis in order to rule out cystic fibrosis. The differential diagnosis include encephalocele, inverting papilloma, carcinoma, olfactory neuroblastoma, and angiofibroma.

The first approach in therapy should be medical, using antibiotics, antihistamines, steroids, and allergic hyposensitization, as appropriate. Surgical management is used secondarily and may include polypectomy, ethmoidectomy, and possibly other surgical techniques for the sinuses.

Vasomotor Rhinitis

Vasomotor rhinitis is a misnomer since it is not a type of inflammation. It is due to dilation of the nasal vessels and consequent nasal discharge. The condition appears to be more common in patients who are suffering from chronic anxiety states. Many of these patients may simply be intolerant of the normal production of nasal mucus (500 to 700 mL per day). The condition occurs more commonly in adolescents and young adults and is more common in women. Patients primarily complain of nasal obstruction and clear nasal discharge. Examination often reveals mild swelling of the nasal mucosa. Systemic decongestants and antihistamines are the primary treatment for this condition. Patients must be educated about the deleterious effects of long-term use of topical decongestants. Patients who present with chronic, intractable symptoms may be considered candidates for submucous resection or cryosurgery of the nasal turbinates.

Rhinitis Medicamentosa

Rhinitis medicamentosa is not an inflammatory condition but a chronic, reactive vasodilation due to excessive use of topical nasal vasoconstrictors. After several days' use of topical vasoconstrictors, a rebound phenomenon occurs that consists of rhinorrhea, edema, and loss of ciliary function. A vicious circle develops that leads to increasingly frequent use of the offending medication. Patients soon come to feel addicted to the use of the nasal drops or sprays and typically have a great deal of trouble discontinuing their use. The patient's only recourse is to suffer nasal obstruction for about 2 to 3 weeks, after which time the normal tone returns to the nasal vasculature. Systemic decongestants, nasal instillation of normal saline, and aerosolized nasal corticosteroids may help to relieve symptoms during this period of withdrawal from the medication.

Viral Rhinitis

Colds are the most common type of infection in humans, occurring at least once or twice a year in adults and five to eight times per year in children. Although they are little more than a nuisance in the adult, they are often accompanied by sinusitis and middle ear infections in children. The main causative factor is one of may strains of rhinovirus. Other agents may cause a similar condition and include *Mycoplasma, Neisseria, Haemophilus influenzae,* and *Staphylococcus aureus.* Peak occurrences of viral rhinitis occur in September, January, and April.

A low-grade fever is rapidly followed by irritability, sneezing, and nasal discharge. Nasal secretions become progressively thicker, often purulent. Myalgias, headache, and a nonproductive cough are also common.

Treatment recommendations include acetaminophen, fluids, rest, and decongestants. Aspirin should not be given to children because of the increased risk of Reye's syndrome in

cases of influenza or varicella infection. Instillation of nasal decongestants is useful for infants who have trouble breathing and eating from nasal obstruction. Saline or 0.125 to 0.25 per cent phenylephrine may be used. Symptoms rarely last for more than a week.

Acute Sinusitis

Acute sinusitis may involve any or all of the paranasal sinuses, which include the frontal, maxillary, sphenoid, and ethmoids. Acute sinusitis, in contrast to subacute and chronic infections, is defined as lasting from 1 to 3 weeks. It most commonly follows nasal obstruction from viral rhinitis. Mucosal swelling causes blockage of the ostia, resulting in obstruction and subsequent progression of the infection. Other predisposing causes include allergic rhinitis, deviated nasal septum, foreign body, and frequent swimming. Systemic predisposing factors include diabetes, malnutrition, and blood dyscrasias (Kern, 1988). The most common organisms found are streptococci, pneumococci, *H. influenzae*, and staphylococci. Sinusitis may occasionally be caused by gram-negative and anaerobic organisms, fungi, and mycobacteria.

The early symptoms of sinusitis are those of viral rhinitis, followed by a feeling of fullness over one side of the face or a dull, localized headache. Frontal involvement is often associated with a generalized headache and may progress rapidly. Maxillary sinusitis often causes radiation of pain to the teeth. Ethmoid sinusitis results in pain over the bridge of the nose and behind the eye. Sphenoid involvement also causes retro-orbital pain and may cause an occipital headache. On examination, patients may also have evidence of periostitis, leading to swelling and erythema over the involved sinus. The nasal mucosa is often erythematous and edematous, and purulent nasal drainage is often present. Tenderness to palpation may be present over the frontal or maxillary sinus.

Table 33-3. Causative Organisms in Acute Sinusitis

Agent	Incidence (Per Cent of Cases)
Bacteria	30
Streptococcus pneumoniae	30
Haemophilus influenza	20
Anaerobic bacteria	10
Staphylococcus aureus	4
Streptococcus pyogenes	2
Branhamella catarrhalis	2
Aerobic gram-negatiove bacteria	9
Viruses	
Rhinovirus	15
Influenza	5
Parainfluenza	3
Adenovirus	< 1.

Sinus x-ray studies are helpful in confirming questionable cases and may show mucosal thickening or air-fluid levels. If symptoms are limited to the maxillary sinus, a simple Waters view will usually suffice. Cultures may be taken from the posterior nasopharynx, but their usefulness is debated.

Treatment is directed at the infection itself and toward relieving congestion of the nasal mucosa to allow drainage of the involved sinus. Either ampicillin or erythromycin is effective against the majority of organisms causing sinusitis (Table 33-3). Treatment should be continued for a minimum of 10 days and, for recalcitrant cases, may need to be continued for as long as 21 days. A topical nasal decongestant spray should be used three times a day for a maximum of 5 days. Systemic decongestants may also be used.

Ethmoid Sinusitis

The ethmoid sinus complex is the primary key to the health of the nose and the other pairs of sinuses. Ethmoid sinus development begins with the appearance of small slits along the lateral wall of the nose during the 5th month of fetal development. At birth, the maxillary and ethmoid sinuses are the only sinus cavities, and during childhood these two groups are responsible for most of the complications of sinusitis occurring in children. Each ethmoid sinus complex consists of 4 to 17 cells, with the anterior group draining into the recess just beneath the middle turbinate and the posterior group beneath the superior turbinate.

Clinical manifestations of *ethmoid sinusitis* include upper facial pain, discharge, visual dysfunction, headaches, fever, and chronic cough. Because only a thin bony wall separates the ethmoid complex from the eye and the brain, complications of ethmoiditis may threaten both of these regions.

In addition to the history and physical examination, radiographic studies are essential for adequate assessment of the ethmoid sinuses. The computerized tomography scan has become the gold standard for precise evaluation.

Medical management includes antibiotics, antihistamines, decongestants, topical steroids, and allergic hyposensitization as appropriate for each individual patient. The goal is to relieve sinus obstruction and re-establish drainage and aeration of these cells. Usually, thic can be accomplished with medical management, but when this fails, surgical intervention is indicated to avoid complications.

Chronic Nasal and Sinus Infection

Chronic or recurrent infection involving the nose and paranasal sinuses is a common and often frustrating challenge for primary physicians and otolaryngologists. Most instances of *chronic rhinosinusitis* are the result of pathologic conditions in the nasal airway, and many patients with these conditions cannot be controlled by medical management alone. With time, chronic infection results in disruption of normal airflow patterns (obstructed nasal breathing) and nasal ciliary clearance of mucus (anterior and posterior nasal discharge). When this occurs, more serious and even life-threatening complications become likely.

In some patients, the predisposing causes of the chronic condition are correctable. These include such factors as environmental smoke, dust, fumes, and pollen - all of which must be assessed and limited or controlled. Trauma to the nose or abnormal growth/development may result in internal deformity of the nasal septum, a problem that is reviewed later.

Endoscopic nasal and sinus diagnosis and surgery provide an important advance in this field. The ability to precisely identify the source and nature of the pathology is now linked with new techniques that permit removal of abnormal tissue and preservation of areas that can regain their normal function. Procedures are now performed on an outpatient basis that are safer and more effective than prior operations that required several days of hospitalization.

Acute and Chronic Frontal Sinusitis

The frontal sinus begins to develop between the 1st and 2nd years after birth and reaches its full size by about age 20. This sinus drains into the middle meatus under the middle turbinate.

Acute frontal sinusitis may develop secondary to various conditions that interfere with adequate sinus aeration and drainage. Typical predisposing problems are allergic rhinitis, polyps, septal deviation, tumors, or nasal infection. Common symptoms are headaches that are worse in the morning, mucopurulent discharge, and fever. Examination often reveals edema and tenderness over the sinus. Radiographic studies may show an air-fluid level or complete opacification of the sinuses. Complications include osteomyelitis, Pott's puffy tumor of the forehead, orbital cellulitis, and intracranial infection.

Treatment of acute frontal sinusitis is directed at re-establishing sinus drainage using topical and systemic decongestants along with antibiotic therapy. Heat, humidified air, and rest are often helpful adjunctive measures. When medical management fails, the frontal sinus may require a drainage procedure (trephination).

Chronic frontal sinusitis may result from inadequately treated acute frontal sinusitis and is a surgical problem. The common symptoms are nasal discharge and frontal headaches. Roentgenograms show thickened mucosa or bony sclerosis, or both. The surgical procedure used is osteoplastic fat obliteration.

Nasal Tumors

Tumors of the nasal passage and sinuses, other than polyps, are uncommon but of considerable importance because of their location and their tendency to impair nasal function. They usually become symptomatic by causing obstruction, pressure, or bleeding. Radiographic studies are useful in determining tumor extension to adjacent sites, and a biopsy is required to define the precise nature of the growth.

The first category of nasal masses is *tumor-like lesions* of the nose and paranasal sinuses. *Gian cell reparative granuloma* is felt to be an aberrant form of the local reparative reaction in response to an inflammatory process. This lesion may occur in any of the sinuses, but it is most commonly found in the maxillary or ethmoid region and is seen more frequently

in young patients.

Ossifying fibroma is a cellular fibroma that produces calcified intercellular material. Seen most often in children as a painless cheek mass, it can expand and obliterate the maxillary sinus. Excision usually results in cure. A similar disorder, *fibrous dysplasia*, differs in that it arises from the proliferation of fibro-osseous tissue inside the affected facial bones and is not a true neoplasm. The more common monostotic form usually involves the frontal or sphenoid bones, causing a single, unilateral facial swelling. The polyostotic form affects females more frequently and may be associated with skin lesions and sexual precocity, in which case it is termed Albright's syndrome.

The second category is *benign neoplasm*, with nasal *papilloma* being the most common example. Exophytic papillomata are very firm and usually are cured by simple excision. *Inverting papilloma* is a softer, verrucous lesion that usually arises from the lateral nasal wall. These neoplasms grow slowly and invade the underlying bone. They tend to recur following excision, and 4 to 15 per cent eventually are found to be malignant.

An *osteoma* is a benign neoplasm that grows slowly and is often asymptomatic. This tumor arises more frequently in pubertal males, and it usually involves the frontal sinus, with the ethmoid region being the next most common site. Surgical excision is necessary if the osteoma grows sufficiently large to obstruct the sinus ostium.

Malignant neoplasms are the third category, and these are relatively rare, accounting for about 3 per cent of all upper aerodigestive tract cancers. Known etiologic factors are nickel, wood dust, and Thorotrast. Most patients are over the age of 50, most of these tumors arise in the maxillary sinus, and over half of the tumors are advanced (T_3 or T_4) when the diagnosis is made. *Squamous cell carcinoma* is the most common histologic type, and combined surgery and radiation therapy are employed in managing patients with these lesions. Cure rates of about 30 per cent are reported.

Adenocarcinoma is found predominantly in the ethmoid sinuses. The patients are slightly younger, the tumors are somewhat more slow growing, and the prognosis is slightly better than for squamous cell carcinoma.

Lymphoma may arise in extranodal form in the nose or paranasal sinuses. Subclasses of nasal lymphoma include reticulosarcoma, lymphosarcoma, and plasmacytoma. Most of these tumors arise in the maxillary antrum, and they occur in patients at a younger age than most carcinomas. Unlike lymphoma elsewhere in the body, dissemination of sinus tumors is not common and the prognosis is better than for other lymphomas and carcinomas.

Melanoma is arare and is found in older patients, with a mean age of about 75. These tumors tend to arise high in the nasal cavity and tend to invade the ethmoid sinuses. In contrast to skin melanoma, these malignancies do not appear to arise in pre-existing lesions and many of them may remain localized for a prolonged period. However, they carry a very poor prognosis, with a 5-year survival rate of about 5 per cent.

Esthesioneuroblastoma is a rare tumor arising in the roof of the nose. It usually occurs in young adults, and the common symptoms are nasal obstruction and loss of sense of smell. These tumors tend to spread submucosally, making it difficult to assess accurately the extent of the disease. Metastasis occurs in about 20 per cent of patients, and aggressive surgical excision is combined with radiation therapy in most instances. Cure rates are approximately 50 per cent with aggressive treatment.

Nasal Trauma

The nose is the most frequent injured structure in the head and neck region. The bony skeleton of the external nose is formed by the paired nasal bones that join in the midline and are supported by the nasal process of the frontal bone and the frontal process of each maxilla. The middle third of the nasal skeleton is composed of the upper lateral cartilages, while the lower lateral cartilages support the lower third. Internal nasal structures of importance are the quadrangular cartilage and the bony vomer upon which it rests.

In adults, the nasal bones remain attached to each other when fractures, whereas they frequently separate in children. Therefore, unilateral depressed *nasal bone fractures* are much less common in adults than in children. Inside the nose, displacement of the quadrangular cartilage from the vomer or angulation of the quadrangular cartilage is a common occurrence in response to a strong anterior blow. Hematoma formation is common at these various fracture sites, making these areas susceptible to infection after a few days. In most cases, fibroblasts are activated but are quickly replaced by osteoblasts, which begin to lay down callus, leading to overall thickening of the bone and rapid healing that is quite advanced within 10 days.

Diagnosis of fracture is made on the basis of a visually apparent deformity or the palpation of loose or displaced fragments. Prior studies have shown that less than half of these patients have had an intranasal examination, an essential component of a thorough work-up and an important step in planning the proper treatment. Radiographic studies usually are not helpful and may be confusing.

In the absence of a significant septal fracture, reduction of displaced nasal bones can be accomplished using regional anesthesia (Bailey, 1982). Reduction is best performed between 3 and 7 days after injury. Intranasal packing is required for 7 to 10 days and an external splint is useful to maintain the reduction.

If there is a concomitant *fracture of the nasal septum* with the external pyramid, alignment of the septum must be accomplished and maintained to prevent a later shift of the external nose by the internal structures. In the case of an isolated fracture-dislocation of the quadrangular cartilage, reduction is necessary to prevent the late complication of nasal airway obstruction. Open reduction, sometimes requiring general anesthesia, may be needed to avoid the 40 per cent failure rate reported with closed reduction techniques.

In the instance of delayed treatment for septal deviation, the goals of surgery are two-fold: to correct any dorsal deviation that gives the impression of a crooked nose and to restore a patent nasal airway on each side. Osteotomy or refracturing of misaligned nasal bones may be necessary, often combined with straightening and repositioning the nasal cartilages that

shape the contours of the lower two thirds of the external nose.

Nasal and septal surgery in the child raises concern over the possible disruption of nasal and facial growth centers. Experience has shown that severe nasal trauma may displace and disrupt nasal structures and redirect the lines of growth, resulting in deformity. Therefore, most surgeons have concluded that there is less potential for harm in operating carefully upon selected patients than in delaying correction of traumatic deformities until these young patients become adults.

Frontoethmoid Fractures

Severe traumatic injury may result from high energy impact at the junction of the nose and forehead. Anatomically, this nasal complex region is not a single unit but a combination of adjacent structures that are relatively fragile. When fractured, the upper portion of the nasal skeleton can be driven inward and superiorly toward the anterior cranial fossa. This may result in heavy bleeding from tearing of the anterior ethmoid arteries, disruption of the medial palpebral ligaments of the eyelids, injury to the lacrimal system, and cerebrospinal fluid leak. The trochlea can be avulsed, causing diplopia (double vision).

On clinical examination, the cardinal sign of this injury is a broad, flat, depressed, and unstable nasal dorsum. There is usually a laceration over the nasal bridge and considerable edema of the adjacent soft tissue. There may be epistaxis and nasal obstruction as well.

Ophthalmologic assessment is a mandatory step for any patient with an injury in this area, to detect any associated eye injuries. Radiographic studies are necessary to define the exact status of the facial bones and skull base.

Definitive repair of *ethmoid complex fractures* is accomplished quickly, before fragments become fixed and healed in a poor position. Open reduction techniques with wiring of fractures and repair of the palpebral ligaments and reconstruction of the other soft tissue elements is required to prevent serious late complications.

Traumatic CSF Leak

Cerebrospinal fluid otorrhea occurs in about 6 per cent of cases involving basilar skull surgery. Fortunately, 90 per cent of these leaks close spontaneously, but persistent cerebrospinal fluid otorrhea is not uncommon following longitudinal fractures of the temporal bone. The diagnosis is made on the basis of chemical testing of the fluid, which reveals a glucose content that is two thirds of the blood glucose level. The most accurate test involves identifying two electrophoretic bands of transferrin.

Cerebrospinal fluid rhinorrhea may follow closed head injury or midfacial fractures. About 80 per cent of these cerebrospinal fluid leaks will be evident within 48 hours. A high index of suspicion is important because 20 per cent of the patients found to have a leak within 1 week of the injury will develop meningitis.

Treatment of cerebrospinal fluid leak is usually carried out by otolaryngologists and neurosurgeons. Antibiotic coverage is usually employed initially, and surgical repair is

indicated for those patients whose leaks do not cease spontaneously in a timely manner.

Turbinate Dysfunction

The nasal turbinates are three shelf-like projections from the lateral wall of the nose. Their mucous membrane covering is lined with pseudostratified, columnar ciliated epithelium. The turbinates warm and moisten the inspired air, and they participate in the movement of the blanket of mucus that acts as a filter by catching and holding 95 per cent of the particles in the inspired air. By this function, they play a vital role in the body's overall defense against infection.

The turbinates become involved in many disease processes such as *acute rhinitis* (common cold), *allergic rhinitis, vasomotor rhinitis* (autonomic imbalance), and *rhinitis medicamentosa* (abuse of nose drops).

Chronic hypertrophic rhinitis is the end stage of the above types of rhinitis, and it is characterized by enlarged, meaty, obstructive turbinates. The diagnosis is confirmed by spraying the nose with a topical sympathomimetic solution (ephedrine) and observing the lack of a decongesting effect. Surgical management is the only effective treatment for this problem.

Oral Cavity and Pharynx

Acute Pharyngitis and Tonsillitis

Acute pharyngitis is one of the most common reasons patients visit the family physician. The two most common causes are viral and streptococcal. Recently the role of other agents such as *Chlamydia* and *Mycoplasma* has been debated. McMillan and colleagues (1986) reported that 40 per cent of 320 patients with sore throat and positive strep cultures compared with 11.9 per cent of controls. Sixteen per cent had positive viral cultures compared with 2.9 per cent of controls. While 15.8 per cent were positive for *Mycoplasma,* 17.6 pwer cent of controls also had positive cultures. This study supports the common belief that beta-hemolytic streptococci represent the major cause of significant bacterial pharyngitis (Mandel, 1985).

Patients with streptococcal pharyngitis present with sore throat, fever, and odynophagia. Myalgias, arthralgias, abdominal pain, headache, and vomiting may also occur. When cough and rhinorrhea are present, a viral etiology is much more likely. Physical examination reveals pharyngeal erythema, often with a patchy, purulent tonsillar exudate. Petechiae in the soft palate and tender anterior cervical lymph nodes are often present.

Diagnosis is made by either a throat culture or a latex fixation test for streptococcal antigen (rapid strep test). Recent evidence suggests that this technique is as sensitive and specific as the throat culture and, thus, is likely to replace that procedure in the near future (Fischer, 1986). Rapid identification allows early treatment of streptococcal pharyngitis, which can reduce the duration of symptoms to less than 24 hours (Bass, 1986). DeNeef (1986) notes that use of the rapid test minimizes costs and time away from work. When the rapid test is negative, a culture should generally be performed to clarify the diagnosis, since false-negative

rates have been reported to range between 5 and 10 per cent.

Treatment consists of increased fluids, acetaminophen, warm saline gargles, and antibiotics. Penicillin is the drug of choice. If oral medication is preferred, penicillin V 250 QID for 10 days should be given to adults and about 50.000 units (15 to 50 mg) per kg per day should be given in four divided doses to children. Alternatively, intramuscular benzathine penicillin may be given according to the following guidelines: 600.000 units for children under 6 years of age; 900.000 for children between 6 and 9 years of age; and 1.2 million units for anyone over 9 years of age. Erythromycin is the drug of choice for patients who are allergic to penicillin.

Stomatitis and Oral Manifestations of Systemic Disease

Many oral problems are localized and are not associated with systemic diseases (eg, gingivitis, glossitis). In other instances, disease processes involving the rest of the body affect the mouth. It may be difficult to differentiate between these two categories and to decide which consultant to involve.

Infection of the oral cavity may be of bacterial etiology. *Streptococcal gingivostomatitis* is an example and is caused by *Streptococcus viridans* or beta-hemolytic streptococcus. It differs from other forms of gingivitis in that it does not result in loss of gingival tissue. *Tuberculosis* may be associated with the oral cavity on rare occasions, with a predilection for the dorsum of the tongue. In other instances, the palate or the gingiva will be primary sites.

Viral stomatitis may take several forms, including *herpes zoster, herpes labialis* (herpes simplex virus), *herpangina* (Coxsackie virus), or a *viral wart.*

Acute necrotizing gingivitis (trench mouth) is a fusospirochetal infection that causes a grayish-yellow pseudomembrane that bleeds easily, fetid breath, fever, and cervical lymphadenopathy. It causes necrosis of the interdental papillae and recession of the gingival margin. *Oral syphilis* is rare, but may be associated with tertiary syphilis as a palatal perforation or a tongue mass.

Mycotic stomatitis is a category that includes acute and chronic conditions. *Acute pseudomembranous candidiasis* (thrush) is caused by *Candida albicans* and is seen most often in infants and debilitated, diabetic, or immunocompromised patients. It may occur as a side effect of the administration of antibiotics, corticosteroids, or cytotoxic drugs. *Chronic hyperplastic candidiasis* is signaled by an isolated white patch resembling oral leukoplakia. Both of these infections respond to antifungal agents such as nystatin or myconazole.

Actinomycosis (lumpy jaw) is a chronic infectious-granulomatous disease usually caused by *Actinomyces istraelli* and characterized by tissue invasion and spread, with the formation of multiple sinus tracts. It presents as a bluish swelling of the tongue or gum or as a palpable neck mass. It is quite responsive to treatment with penicillin or ampicillin.

Other systemic diseases causing oral cavity abnormalities are summarized in the Table.

Table 33-4. Diseases That Cause Abnormalities of the Oral Cavity

Hematopoietic System	
Iron deficiency anemia	Loss of lingual papillae Pale or fiery red tongue
Sideropenic dysphagia (Plummer-Vinson syndrome)	Angular cheilosis Thin vermilia Atrophy of lingual papillae Esophageal webs
Pernicious anemia	Oral cavity paresthesias Taste disturbances Xerostomia (dry mouth) Lobulated tongue, loss of papillae
Thrombocytopenia	Bleeding, ecchymoses, purpura, and petechiae
Malignant neutropenia (may be drug induced)	Infective ulcerations Fever, sore throat, sweating, headache, and prostration
Chronic idiopathic neutropenia	Subacute gingivitis Loosening of teeth Recurrent aphthous ulcers
Nervous System	
Melkersson-Rosenthal syndrome	Unilateral facial paralysis Facial swelling Fissured tongue
Musculoskeletal System	
Dermatomyositis	Gingival edema
Scleroderma	Fibrotic, rigid lips Pale oral mucosa Immobility of tongue
Sjögren's syndrome (sicca syndrome)	Xerostomia Enlarged salivary glands Keratoconjuctivitis
Mikulicz's disease	Enlarged salivary glands
Skin	
Pemphigus vulgaris	Large bullae in oral cavity
Erythema multiforme (Stevens-Johnson syndrome)	Stomatitis Skin bullae
Discoid lupus erythematosus	Erythematous lesions, followed by scaling, then atrophic lesions
Psoriasis vulgaris	Geographic tongue
Reticular lichen planus	Delicate white-gray buccal lesions.

Several categories of systemic diseases may cause lesions in the oral cavity. These categories include endocrine, nutritional, and metabolic system diseases as well as hematopoietic system disorders and nervous system disorders. *Diabetes mellitus* may cause tongue dryness in addition to gingival bleeding hypertrophy and purple discoloration.

Addison's disease often results in changes of oral mucosa pigmentation (white or very dark-appearing areas). *Acromegaly* is associated with mandibular hyperplasia and marked enlargement of the tongue (macroglossia). *Ascorbic acid deficiency* (scurvy) causes the gingival tissue to become quite swollen and to bleed easily. *Severe protein depletion* (kwashiorkor) results in an acute necrotizing gingivitis, candidiasis, atrophy of the tongue papillae, and cracking of the skin at the angles of the mouth. *Waldenström macroglobulinemia* may be associated with the mucosal purpura and bleeding gums. *Amyloidosis* is associated with tongue enlargement.

Deep Neck Infections

The deep neck cervical fascia completely envelops the neck, extending from the nuchal line of the skull and the cervical spine and wrapping around to attach to the hyoid bone and the clavicle. In the preantibiotic era, nearly all of the deep neck infections originated in the pharynx and tonsils, but now dental, otologic, nasal, and salivary gland origins are quite common. Most deep neck infections are caused by Streptococcus spp, but *Staphylococcus aureus* and anaerobes are also significant pathogens. There are five major distinct types of deep neck infections: pharyngomaxillary, retropharyngeal, submandibular, parotid, and masticator space infections.

Pharyngomaxillary space infections arise in the space bounded by the hyoid bone, the temporal bone, the lateral pharyngeal wall, and the mandible. The most common sources are infections of the pharynx and tonsils. Initial manifestations include fever, sore throat, and pain on swallowing (odynophagia). Trismus and medial displacement of the tonsil soon follow. Treatment for this abscess is intravenous antibiotics and drainage through an incision made below the angle of the mandible.

Retropharyngeal space infection occurs in the region deep to the posterior pharyngeal wall. Sites of origin include the nose, sinuses, and adenoids and nasopharynx. These abscesses are usually seen in children younger than 4 years of age, and when seen in adults, the physician should consider tuberculosis. Early signs and symptoms include refusal of food, followed by fever and respiratory obstruction. Later signs are neck extension and tilting of the head toward the side of less involvement. The infection may spread into the mediastinum. Intravenous antibiotics, incision and drainage, and, occasionally, a tracheotomy are the main therapeutic steps.

Submandibular space infections involve the anatomic space bounded by the floor of the mouth and the deep cervical fascia between the mandible and the hyoid bone. Most follow dental infections or a tooth extraction. There may be skin redness and fluctuance or mouth and tongue swelling. The tongue may be sufficiently displaced posteriorly to require a tracheotomy for airway obstruction. Intravenous antibiotics and incision and drainage are mainstays of therapy.

Parotid space infections usually follow acute parotitis, often in postoperative, dehydrated, or debilitated patients. There is pain, swelling, and warmth over the parotid gland. *Staphylococcus aureus* is a common pathogen, and successful treatment may require intravenous antibiotics, hydration, sialogogues, low dose radiotherapy, and incision and

drainage.

Masticator space infections involve the region just anterior to the pharyngomaxillary space and often result from infection around an impacted third molar. There is trismus and swelling over the angle of the mandible. Intravenous antibiotics and incision and drainage are the therapeutic choices.

Juvenile Nasopharyngeal Angiofibroma

Juvenile nasopharyngeal angiofibroma is a highly vascular, locally aggressive tumor that is found almost exclusively in adolescent males. Grossly, the tumor is a reddish purple, lobulated, sessile mass arising in the roof of the nasopharynx. As it enlarges, it causes progressive nasal obstruction and spontaneous epistaxis. Hearing loss, sinusitis, cranial nerve deficits, and even facial swelling may be noted as the tumor expansion continues. Roentgenograms reveal anterior bowing of the anterior wall of the maxillary sinus, posterior bowing of the anterior wall of the pterygopalatine fissue, and a characteristic tumor blush on angiography. Biopsy is quite hazardous because of the potential for very heavy bleeding. The differential diagnosis includes a nasopharyngeal polyp, lymphoepithelioma, craniopharyngioma, chordoma, and dermoid cyst. Treatment is surgical excision, sometimes preceded by hormonal therapy or embolization as steps used to reduce the operative blood loss. Radiotherapy is reserved for those tumors that are unresectable because of intracranial expansion.

Oral Cavity Malignancy

Squamous cell carcinoma of the tongue and floor of the mouth are the most common malignancies of the oral cavity. These tumors comprise 7 per cent of all cancers in the USA and 45 per cent of all cancers in Bombay, India. Alcohol and tobacco are the most important etiologic factors and act synergistically to induce the development of oral squamous cell carcinoma. These growths are usually preceded by an area of superficial leukoplakia (white patch) that is easily detected during a thorough examination. Bimanual palpation of the tongue and floor of the mouth should be routine during the examination of all patients older than 40 years of age. Diagnosis is confirmed by biopsy, and treatment planning must include assessment of the adjacent mandible and the cervical lymph nodes. Small, superficial cancers in this area can be managed by wide local excision. Larger tumors usually require combined surgery and radiation therapy.

Carcinoma of the palate is usually of the squamous cell type and usually arises near the posterior, free edge of the soft palate. The lesions are ulcerative and cause pain, odynophagia, and a sensation of a mass in the palate. Trismus is a late sign and indicates a poor prognosis. Surgery is utilized for early lesions and is combined with radiotherapy for advanced tumors. After resection, the anatomic and functional palatal defect is rehabilitated by a prosthesis. Prognosis depends upon the stage of the malignancy and ranges from 65-95 per cent for T_1 lesions to 30 per cent for T_3 lesions.

Minor salivary gland cancer commonly occurs on the palate and is exhibited as an asymptomatic, mucosa-covered mass. These tumors grow slowly and are not considered to be curable by radiotherapy. Surgical resection is the therapeutic mainstay.

Buccal (cheek) carcinoma usually occurs in older patients (60 or 70 years of age) with a history of smoking or chewing tobacco. These tumors may be either the superficial verrucous form or deeply invasive. About a third of the patients present with disease limited to the cheek, but unfortunately, patients frequently ignore early symptoms until the lesion passes beyond the point of curability. Small tumors may be cured by using either surgery or radiotherapy.

Malignant melanoma of the oral cavity arises most often on the palate. Grossly, the lesions appear brownish gray with a smooth, lacy pattern, giving a benign appearance. Some authorities recommend biopsy of all pigmented oral cavity lesions arising in Caucasian patients. Treatment is surgical, and the 5-year survival rates are very low at 10 to 15 per cent.

Swallowing Disorders (Dysphagia)

Dysphagia is a very common complaint, especially among the older patient population. Advances in the field of endoscopy and radiology have increased our understanding of the act of swallowing and our ability to assess individual patients. The most common disorders that cause disturbances of swallowing include achalasia, diffuse spasm, esophagitis, diverticulae, and tumors.

Achalasia is a disorder of esophageal motility involving the body of the esophagus and the lower esophageal sphincter. It is characterized by a decreased number of ganglion cells in Auerbach's myenteric plexus. This defect causes diminished or absent peristalsis and failure of the lower esophageal sphincter to relax. As the disease progresses, the esophagus becomes increasingly dilated until an entire meal may be lodged in its lumen. The onset of achalasia is usually insidious and the most common is a sensation of food "sticking" in the lower esophagus. Pain is infrequent, and regurgitatin is common. Treatment consists of dilation in milder cases and an esophagomyotomy in more intractable cases.

Diffuse spasm of the esophagus is characterized by failure of the muscular contractions to follow the usual progressive peristaltic pattern in the distal half. The normal pattern is replaced by a series of repetitive contractions. The cause is unclear but is believed to be a disturbance of vagal tone. Histologically, there is an extreme degree of muscular hypertrophy. Diagnosis is based largely upon radiologic studies that document the functional disturbance. Patients complain of dysphagia and substernal pain that may radiate to the jaw or arms, in some cases mimicking angina. Treatment is similar to that for achalasia.

Esophagitis may be caused by chemical agents (alcohol, spices, tobacco), physical trauma (thermal injury, foreign body), infection (bacterial, viral, *Candida*, parasites), or radiation. Systemic disease may be a factor in some patients (blood dyscrasias, scleroderma, pemphigus, or immunosuppression). Therapy depends on the predisposing factors.

Diverticulae are pouches that form as the result of increased luminal pressure (pulsion diverticulum) or external pulling forces on the esophageal wall (traction diverticulum). *Zenker's diverticulum* is a pharyngoesophageal pouch that is usually seen in elderly males. These pouches cause regurgitation of food, fould odor, dysphagia, and aspiration. The treatment is surgical excision.

Benign esophageal tumors are uncommon and usually present after the 4th decade. They usually give no symptoms when they are small, so consequently they are usually quite large when diagnosed. *Leiomyoma* is the most common of the benign tumors, with cysts, papillomas, polyps, and hemangiomas being rare. Endoscopic excision is a possible treatment for these benign tumors.

Malignant esophageal tumors usually occur in male patients in the 50- to 70-year-old age group. Squamous cell carcinoma is the most common malignancy, and symptoms include dysphagia (with solids initially, then liquids), weight loss, hoarseness (recurrent laryngeal nerve involvement), cough, and pneumonia. Radiographic studies and esophagoscopy are employed for diagnosis. Radiation therapy or surgery may be utilized for treatment, but cure rates are only 10 to 15 per cent.

The Larynx

Examination of the Larynx

Fiberoptic and Mirror Laryngoscopy

Mirror laryngoscopy can be accomplished in most patients unless they have a very sensitive gag reflex. Better visibility is gained by using the largest mirror that can be tolerated by the patient (Johnson, 1984). Spraying the throat with pontocaine or lidocaine prior to examination may help, but if the examination is still difficult, 2 to 5 mg of intravenous *diazepam* will facilitate the process. The patient should be seated upright, leaning forward slightly. The mirror should be warmed slightly to prevent fogging. The tongue should be grasped with a gauze pad and the mirror introduced until it just touches the soft palate. The patient may be asked to "pant like a dog" to suppress the gag reflex, and to say "eee" to approximate the cords and facilitate visualization of the anterior larynx. If only the base of the tongue is visible, it is probably because the patient is not leaning far enought forward.

Both rigid and fiberoptic laryngoscopes are also available for office use. The flexible instrument is inserted through the nose, whereas the rigid scope is inserted through the mouth. The flexible scope is a small version of the flexible fiberoptic sigmoidoscope and is being used increasingly by primary care physicians as a diagnostic instrument. An additional benefit of the fiberoptic scope is the ability to photograph lesions for documentation or future consultation purposes (Dewitt, 1988).

Disorders of the Larynx

Acute Laryngitis

Acute laryngitis in children usually becomes manifest as croup and was discussed earlier. Acute laryngitis in the adult is usually viral in etiology and is a mild, self-limited illness. Patients develop hoarseness, cough, and often become gradually unable to talk above a whisper. Fluids, humidification, and allowing the voice to rest should be advised until symptoms have resolved.

Chronic Laryngitis

Chronic laryngitis is primarily a problem of adults who use their voice for speaking or singing. An acute inflammation is often aggravated by an inadequate period of rest. Chronic bronchitis, excessive alcohol ingestion, and cigarette smoking are also frequent contributing factors. Examination usually reveals edema and, occasionally, thickening or nodularity of the cords. Therapy must focus on removal of all irritating factors and resting the voice, to which the patient is often resistant. A beclomethasone inhaler may help to decrease the inflammation.

Airway Obstruction in the Neonate

Airway obstruction in neonates is characterized by stridor, a rasping, rattling, or musical sound that coincides with the respiratory effort. *Inspiratory stridor* suggests a high obstruction (tongue, pharynx, supraglottis), whereas *expiratory stridor* indicates obstruction of the intrathoracic trachea or bronchi. The most likely causes for neonatal stridor are laryngomalacia, subglottic stenosis, vocal cord paralysis, and vascular ring anomalies.

The nature of the stridor differs with each of the above-mentioned disorders, and careful analysis of the stridor may be pathognomonic.

Laryngomalacia is characterized by a soft, floppy laryngeal skeleton and immaturity of neuromuscular function. Airway intervention is not usually necessary, and the problem resolves by 12 to 18 months of age. *Laryngomalacia* causes a coarse inspiratory stridor, but the vocalizing sounds of the infant are normal. Because of the redundant nature of the arytenoid and aryepiglottic mucosa, the neonate's cry may have a harsh component that is relieved in a prone position.

A neonate with *subglottic stenosis* will have normal-sounding but weak vocalizations obscured by stridor. The cry may be weakened by poor air exchange. The subglottic stenosis usually appears to be concentric, and the point of greatest obstruction is about 2 to 3 mm below the true cords.

Vocal cord paralysis represents about 10 per cent of all congenital laryngeal abnormalities, with unilateral paralysis being more comon than bilateral paralysis. Tracheotomy is often required in the case of bilateral paralysis. *Unilateral vocal cord paralysis* usually results in a breathy cry only, but if stridor is present it is usually louder when awake and may be positional. The infant may sleep quietly when lying on the side of the paralysis and becomes stridulous when placed on the other side.

Laryngeal webs may cause changes ranging from mild hoarseness to aphonia and from mild stridor and cough to gross obstruction with severe distress. The severity of the stridor and the obstruction are proportional to the degree of webbing anteriorly. About three fourths of the webs are glottic, with the remainder divided equally between supraglottic and subglottic webs. Gradual onset and progression of stridor suggests the possibility of an enlarging mass, such as a *subglottic hemangioma.* Feeding difficulties that cause aspiration or cyanosis suggest that the sphincteric mechanism of the laryngeal muscles is deficient (neurologic sensory or

motor deficit). *Subglottic hemangiomas* usually become symptomatic within the first 6 months of life. They occur more commonly in females and more often on the left side. About half of neonates with this condition will have skin hemangiomas. Laser excision is currently recommended as the treatment of choice.

Unsuspected foreign bodies may be another possibility for these problems in neonates and infants. Radiographic studies and endoscopy are often essential steps in pinpointing the exact cause for stridor in this age group.

Among slightly older infants and children, acquired processes become more important as causes for airway obstruction. Prominent among these airway disorders are *viral laryngotracheobronchitis, bacterial tracheitis* (usually *Staphylococcus aureus*), *spasmodic croup,* and *epiglottitis.*

Laryngeal Trauma

Laryngeal trauma can be categorized according to the mechanism of tissue injury as *blunt trauma, penetrating injuries, thermal burns,* and *radiation* injuries. Each of these groups poses special problems in diagnosis and management.

The cartilaginous skeleton of the larynx is suspended from the hyoid bone and generally serves to support and protect the airway. The thyroid cartilage is composed of two halves that join in the midline in a keel-like configuration to form the prominence, or Adam's apple. The cricoid cartilage is shaped like a signet ring and completely encircles the subglottic lumen, the smallest area of the upper airway.

Blunt external trauma is the most common form of laryngeal trauma, and it may result in cartilage fractures. Often, in a motor vehicle accident, the neck is extended and the laryngeal region impacts the dashboard or steering wheel, driving it posteriorly and compressing it against the cervical spine. Patients who sustain this type of accident may fracture the thyroid or cricoid cartilages and lacerate the soft tissue inside the cartilages. Common symptoms are voice changes (hoarseness, aphonia), dysphagia, stridor, pain, local tenderness, subcutaneous emphysema (air in the neck tissues), and hemoptysis. Radiographic studies are essential to rule out cervical spine fracture, and a computerized tomography scan is best for assessing the degree of laryngeal injury.

After the airway is secured, laryngoscopy may be required to clarify the need for surgical repair. In general, surgical exploration is necessary if there is evidence of airway obstruction, subcutaneous emphysema, vocal cord paralysis, mucosal laceration, arytenoid displacement, or cartilage fracture.

Penetrating trauma usually results from a stab or gunshot wound. The signs and symptoms are similar to those of blunt external trauma, the difference being the presence of an obvious neck wound on physical examination. All penetrating laryngeal injuries require operative exploration and repair, along with antibiotic coverage and tetanus prophylaxis.

Thermal laryngeal injuries are usually caused by the aspiration of hot or caustic liquids or by inhalation. These injuries produce an intense inflammatory response with the larynx, often compromising the airway. Intubation is generally required for airway maintenance for the initial postinjury period. Thermal injuries may produce severe pulmonary damage as well. Baseline arterial blood gases, antibiotics, fluid replacement, aminophylline, steroids, and ventilatory support are components of the complex therapeutic regimen that is required in these circumstances.

Radiation injury is an uncommon complication of radiation therapy, but when it is encountered, it requires immediate recognition and treatment. Chondritis and cartilage necrosis have the potential to cripple the larynx or even require its removal. Antibiotics, steroids, and tracheotomy are used to manage this problem.

Laryngeal Papillomatosis

Juvenile laryngeal papillomatosis is the most common benign neoplasm of the larynx in children. It is caused by the human papillomaviruses, a group of related DNA viruses that also cause cutaneous and genital warts. The lesions are red, sessile lesions of the glottis primarily, but often involve the palate, tonsil, pharynx, and nose. The two clinical forms are juvenile onset and adult onset. The juvenile-onset type almost always presents by age 4 and usually is associated with a history of maternal genital warts. The lesions are almost always multiple, aggressive, and recurrent.

The adult-onset type presents in two patterns: as multiple lesions in young adults or as single warts in older adults.

Laryngeal papillomas almost always cause major voice changes and hoarseness. They may become obstructive and at times will also cause problems with aspiration. Laryngoscopy and laser excision is the customary treatment. Interferon has been shown to produce remission and regression of the process, but the effect is temporary and progression resumes when the interferon is discontinued.

Laryngeal Malignancy

Laryngeal carcinoma (almost exclusively squamous cell carcinoma) affects over 10.000 persons in the USA each year. Cigarette smoking and alcohol consumption are the major causative factors. Most laryngeal tumors originate on the true vocal cords (glottic), with nearly all of the remainder involving the false cords and epiglottis (supraglottic) or the pyriform sinuses and vallecula (marginal). The symptoms, signs, treatment, and prognosis differ for each of these sites.

Glottic carcinoma causes hoarseness as an early sign due to the interference with vocal fold movement and glottic closure. Indirect laryngoscopy reveals an irregular, red or white growth (Plate III). Direct laryngoscopy provides an opportunity for biopsy confirmation of the diagnosis and for planning the most appropriate therapy. Early lesions of the true cords can be managed by endoscopic excision, intermediate lesions by partial laryngectomy, and advanced lesions by total laryngectomy or radiotherapy, or both. Cure rates range from over 90 per cent for T_1 glottic carcinoma to about 50 per cent for T_4 lesions.

Supraglottic and marginal zone laryngeal cancer usually is associated with a sensation of a lump in the throat, dysphagia, or an asymptomatic neck node mass. The supraglottic lymphatics are much more plentiful, and early spread to cervical lymph nodes is much more common than with glottic carcinoma. Early tumors can be treated by supraglottic partial laryngectomy, whereas more advanced lesions require total laryngectomy or radiotherapy, or both. Cure rates for supraglottic cancer range from 85 per cent for T_1 lesions to about 40 per cent for T_4 tumors.

The bottom line for primary physicians is the maintenance of a high index of suspicion for laryngeal cancer in patients past 40 years of age. The history of smoking should represent a red flag in the case of older patients with any voice change, dysphagia, sore throat, or neck mass. Persistence of symptoms beyond 2 weeks shifts the burden of proof to the primary physician to rule out the diagnosis of malignancy.

Subglottic (Laryngotracheal) Stenosis

The cross-sectional area of the airway is proportional to the fourth power of the radius of the lumen, so that small changes produced by edema or scarring (particularly in the infant larynx) are greatly magnified. For example, 1 mm of edema in the normal neonatal subglottic larynx reduces the airway by 32 per cent. The subglottic region is particularly prone to damage from an endotracheal tube because the cricoid cartilage is the only rigid structure completely encircling the airway and the submucosa is particularly susceptible to an edematous inflammatory reaction.

Acquired laryngotracheal stenosis has increased in incidence over the past two decades following the widespread use of prolonged intubation of neonates, especially those with prematurity and even smaller larynges. Use of a ventilator adds a piston-like action to the injury caused by an endotracheal tube. Birth weight, tube size, duration of intubation, multiple intubations, and infection are key factors in producing subglottic stenosis. Congenital stenosis has also been a major factor in some reported series.

The usual clinical picture involves an infant who cannot be extubated after prolonged intubation. Other causative factors predominate in older age groups, with external trauma, burns, and granulomatous disease leading the list of causes. Stridor is a common sign, often being biphasic and somewhat subtle at rest, but obvious with crying or exertion. Radiographic studies are helpful in localizing and evaluating the extent of the stenosis. The final step in assessment is endoscopic inspection of the subglottic region to establish the exact nature and configuration of the stenosis.

Treatment methods include dilation and the injection of steroids to decrease obstructive scar formation. Serial laser excision of cicatrix is useful in some instances. Severe stenosis requires open surgical resection of scar and reconstruction of the subglottic region. For neonates, the anterior cricoid splitting procedure has proved to be useful as an early measure for the infant who fails extubation. By releasing the constraint of the cricoid ring, the subglottic dimensions can be increased to accommodate the endotracheal tube without producing scar tissue. Fortunately, the prognosis for successful management of this problem is quite high, with less than 10 per cent of patients being left with a permanent tracheotomy.

Chronic Aspiration

Chronic aspiration results from processes that disrupt the normal act of swallowing and permit saliva or ingested food to enter the laryngotracheobronchial airway. The first phase of swallowing is voluntary and consists of using the tongue to push a food bolus into the oropharynx while contracting the palate to seal off the nasopharynx. All subsequent phases are involuntary, beginning with the movement of the food bolus down the pharynx by the contraction of the pharyngeal constrictors. Then the cricopharyngeus muscle relaxes, allowing the bolus to flow into the proximal esophagus. The larynx is simultaneously elevated to lie under the posteriorly displaced tongue base. The true and false cords move to the midline to close and protect the airway. The pathologic processes that may interfere with normal swallowing include *neurologic, neoplastic,* and *traumatic* disorders.

Evaluation of the patient with chronic aspiration begins with a careful history designed to narrow the possibilities to one of the three major groups mentioned earlier. Then a complete head and neck examination is performed with particular attention to evaluating cranial nerve and laryngeal function. Radiographic studies (especially a cine fluoroscopic examination) and endoscopic examination may be necessary to define precisely the nature of the problem.

Many patients with this condition are elderly and weak, and the major risk to their survival is *aspiration pneumonia.* In this group, the use of small feeding tubes, raising the head of the bed at a 45-degree angle, and changing their habits to reduce bolus size can be effective therapy.

Surgical therapy must be considered if the patient cannot be managed successfully by medical means. The need for this consideration could be shown by signs of pneumonia or could develop as a requirement for improved alimentation. The type of procedure chosen must be appropriate for the cause of the aspiration, the therapeutic objective, and the prognosis for recovery.

Surgical options include the following:

1. Tracheotomy and an inflated balloon cuff around the tracheostomy tube are used to prevent aspiration. This is a short-term solution for patients who can be expected to regain normal swallowing ability.

2. Hypopharyngostomy or esophagostomy is a surgical opening made for temporary or permanent feeding purposes.

3. Teflon injection into a paralyzed vocal cord expands the cord medially to close any opening that permits aspiration.

4. Cricopharyngeal myotomy is a cutting of the muscle fibers in those patients who are aspirating because of a holdup of the bolus caused by cricopharyngeus spasm or paralysis.

5. Laryngeal closure procedures plus tracheotomy seal off the larynx surgically to prevent aspiration.

The key to successful management of patients with this condition lies in the precise assessment of the cause and thoughtful consideration of the patient's prognosis. A plan must be chosen that is safe and effective but potentially reversible in the event of recovery of function.

Vocal Cord Paralysis

The larynx is a complex neuromuscular organ with several key functions, such as phonation, respiration, and protection of the lungs, to name a few. Vocal cord movement is controlled by the coordinating action of the vagus nerve through the superior laryngeal nerve, which supplies the cricothyroid muscle (tenses the cords), and the recurrent laryngeal nerve, which innervates the remaining intrinsic laryngeal muscles. Only one pair of muscles act to pull the vocal cords apart for breathing, whereas several muscle pairs bring them to the midline for phonation and airway protection.

Superior laryngeal nerve paralysis may be subtle, since there are remaining muscles to compensate for the motor function loss. It produces a paralysis of one or both cricothyroid muscles, which results in bowing of the true vocal cord on phonation. Inability to tense the cord causes a loss of ability to sing or speak in the higher pitch range. There may be mild problems with aspiration, but these are usually transient.

Unilateral recurrent laryngeal nerve paralysis usually produces an immobile vocal cord situated in a position just off the midline. Patients may have a hoarse and breathy voice and may have a weakened cough and may aspirate. In many cases, there is a gradual compensation process, with the opposite cord taking up much of the slack. If compensation is incomplete, teflon injection of the paralyzed cord is indicated.

Bilateral recurrent laryngeal nerve paralysis usually is associated with airway obstruction with stridor. The voice is usually strong because the cords are paralyzed near the midline of the airway. Tracheotomy is necessary if the airway obstruction is severe.

In general, vocal cord paralysis is classified as being either central or peripheral in origin. *Central paralysis* is caused by diabetic neuropathy, aortic aneurysm, inflammatory disease (from a virus, influenza, tuberculosis), bronchogenic carcinoma, esophageal cancer, thyroid tumors, mediastinal or neck metastatic cancer, surgical trauma (thyroidectomy), basal skull fracture, chest surgery, or penetrating neck trauma. Therefore, the work-up must include a comprehensive history and physical examination with particular attention to neurologic problems. Chest, skull base, esophageal and neck roentgenograms may be necessary. Lab studies include complete blood count, venereal disease testing, fasting blood sugar, viral antibody titers, rheumatoid factor, and a heavy metal screen.

Management options include watchful waiting, tracheotomy, neck exploration for trauma, teflon injection, and arytenoidectomy. Neuromuscular pedicle reinnervation techniques show promise for restoring laryngeal function in many patients, but these procedures are still in the process of clinical refinement and are not universally accepted.

The Neck

Congenital Neck Cysts and Sinuses

The branchial apparatus is a system of segmental arches separated by external grooves and internal pouches that develop during the 4th week of intrauterine life. Grossly, this apparatus resembles a system of gill slits, and in some children, disorders of development result in the appearance of cysts, draining sinuses, or lymphatic vascular tumors. The most common of these lesions are *branchial cleft cysts*, *thyroglossal duct cysts*, and *lymphangiomas.*

Branchial cleft cyst usually appears as a smooth, round, nontender mass along the anterior border of the sternocleidomastoid muscle and deep to that muscle. These cysts usually do not become apparent until the second decade of life, when the slow accumulation of fluid or the onset of infection involving the cyst calls attention to its presence. These cysts are generally lined by stratified squamous epithelium with hair follicles and sweat and sebaceous glands. Cysts are more common than fistulas or sinus tracts. The exact location of the cyst and its associated tract varies depending on which branchial cleft is the origin. Diagnosis is based on the history of a progressively enlarging neck mass that is not consistent with cervical lymphadenopathy and is located in an appropriate site. Treatment is by surgical excision of the cyst and its tract, and postoperative recurrence is uncommon.

Thyroglossal duct cyst is a remnant of the descent of the mesodermal tissues in the rebgion of the ventral (thyroid) diverticulum to its final developmental status as the thyroid gland. Along the way, this tissue passes through the hyoid bone region, and occasionally, duct tissue remnants are left along the route and form midline cysts. These cysts are usually located immediately inferior to the hyoid bone, but they may be located anywhere from the submental region to the suprasternal notch. The cysts are lined by squamous, ciliated, or transitional epithelium and are surrounded by a fibrous tissue capsule. The cyst fluid is mucinous and often contains cholesterol crystals. About half of these cysts become apparent prior to the age of 10 years as a painless, midline neck mass. Protrusion of the tongue usually causes the mass to move superiorly. Treatment consists of complete excision of the cyst and its tract in patients with infection or cosmetically unacceptable appearance. The procedure includes removal of the central portion of the hyoid bone and deep cone of midline tongue tissue. These steps have greatly reduced the incidence of cyst recurrence (from nearly 50 per cent in earlier years to less than 5 per cent).

The third most common congenital neck mass is *cystic hygroma* (often used synonymously with *lymphangioma*). This tumor is composed of lymphatic elements that are arranged like a cluster of grapes to form a soft, usually compressible mass that can be located at any level between the maxilla and the axilla. Typically, the mass is situated in the lower half of the neck and is noted during the neonatal period. Another pattern of presentation is that of a smaller mass located in the upper neck or lower portion of the face that becomes apparent after the age of 3 to 4 years.

Diagnosis is made on the basis of the painless, progressive enlargement in size and the typical physical findings. Treatment is through surgical excision, with great care taken to avoid injury to important head and neck structures.

Salivary Gland Disease in Children

With the exception of mumps, salivary gland disorders are more comon in adults than in children. Most pediatric salivary gland problems are characterized by a painful swelling or a gradually enlarging mass. The parotid gland and the submandibular gland are both surrounded by capsules that tend to constrain any swelling or infection that arises within the gland. Each gland is composed of a set of lobulated glandular units that drain through a branching series of ducts into one main excretory duct. This arrangement predisposes the gland to recurrent infection if there is blockage of a major duct by inflamation or a stone. The sole function of the salivary glands is the production of saliva for hydration, lubrication, and digestion. Glandular secretion results from both sympathetic and parasympathetic stimulation.

The most common cause of parotid gland swelling in children is *acute viral parotitis,* or mumps, which is a febrile illness that causes painful parotid enlargement. The physical examination reveals a red punctum (opening of a duct inside the cheek) with clear saliva. The disease is usually caused by mumps virus, but it may be caused by echovirus or Coxsackie virus A. There is an 18- to 21-day incubation period after exposure. Often, all four salivary glands are involved, and important complications include encephalitis, orchitis, pancreatitis, and deafness (usually unilateral).

Lymphoma, sarcoidosis, and *granulomatous diseases* (tuberculosis, infection with atypical *Mycobacteria,* actinomycosis, or cat-scratch disease) are related pathologic changes involving adjacent lymph nodes, and any of these conditions may mimic salivary gland disease. Also, several endocrine and metabolic diseases (such as *Sjögren's syndrome, cystic fibrosis,* and *allergic disorders*) may be difficult to differentiate from salivary gland disease.

Several noninfectious, primary, non-neoplastic diseases may cause swelling of the salivary gland in children. *Sialectasia* is a condition in which congenital, saccular degeneration of the smallest set of ducts causes stasis of the saliva and recurrent parotitis. The exacerbations are usually unilateral, last about a week, recur in 3 to 4 months, and are generally considered to be self-limiting. Diagnosis is by sialography, a radiographic dye study. *Sialolithiasis,* an inflamatory disease caused by an obstructing stone in the main duct, is more comon in the submandibular salivary glands than in the parotid glands. Search for and removal of the offending stone is the management strategy.

Hemangioma and *lymphangioma* are the most comon neoplasms of the salivary glands in children. *Benign mixed tumor* (pleomorphic adenoma) is essentially the only benign, solid, neoplastic mass in children.

Salivary Gland Disease in Adults

Sjögren's syndrome is characterized by a triad of xerostomia, keratoconjunctivitis, and a connective tissue disorder (usually rheumatoid arthritis). The presumptive diagnosis is made when two of these three features are present. On the physical examination there is a dry mouth (xerostomia) secondary to a decrease in salivary flow. The parotid glands are enlarged bilaterally, with a diffuse, firm, irregular contour. Fever and glandular tenderness are common. Patients complain of dryness of the eyes with a burning sensation and photosensitivity. On

inspection there is often a superficial ocular keratitis. Dryness and scaling of the skin is common. Diagnosis is confirmed by increased gamma globulins, rheumatoid factor, and antinuclear antibodies. Biopsy of the lip shows histopathologic changes similar to those of the salivary glands (acinar atrophy, lymphocytic sialoadenitis, and ductal hyperplasia). Early treatment with corticosteroids is recommended.

Salivary gland neoplasms are characterized by the appearance of a slowly enlarging, painless mass in most instances. A *mixed tumor (pleomorphic adenoma)* is the most common. Salivary gland tumors represent about 65 per cent of all parotid tumors and 50 per cent of all submandibular gland neoplasms. Mixed tumors are more common in female patients and usually are present during the fifth decade of life. They are almost always limited to the superficial lobe in the parotid and superficial parotid lobectomy is nearly always curative.

Warthin's tumor represents about 5 per cent of all parotid tumors and is the second most common parotid neoplasm. It most frequently arises in older males and is felt as a rubbery, smooth mass in the tail of the gland (posteriorly and inferiorly). Surgical excision is curative in almost all instances.

Other important benign tumors are *oncocytoma, monomorphic adenoma,* and *sebaceous lymphadenoma.*

The most common malignancy of the parotid gland is *mucoepidermoid carcinoma,* a tumor that most often is exhibited in middle-aged women. Surgical excision carries a 90 per cent 5-year survival for low-grade tumors and a 40 to 50 per cent 5-year survival for high-grade malignancy.

Adenoid cystic carcinoma is the most common malignant tumor of the submandibular and minor salivary glands. Sexual distribution is about equal, and the patients are usually in their 40s. Treatment is through surgical excision, but postoperative radiation therapy is frequently employed because of the high percentage of late tumor recurrence. About 40 per cent of patients with this condition develop distant metastases (usually to the lungs); the 5-year survival is 65 per cent, but the 20-year survival is only about 15 per cent.

Other significant malignant neoplasms include *acinous cell carcinoma, malignant mixed tumor* (or carcinoma ex pleomorphic adenoma), *squamous cell carcinoma, adenocarcinoma, undifferentiated carcinoma,* and *lymphoma.*

Salivary Gland Trauma

Salivary gland injuries are serious and frequently associated with long-term morbidity. Unfortunately, they are often overlooked or underestimated in patients who have suffered multiple traumas. These injuries are classified as *acute trauma* (blunt, lacerating, penetrating, avulsion, or blast) or as *chronic trauma* (irritation from dentures, foreign bodies, stones, or irradiation). The origin of the injury may be primarily external, intraoral, or both. A careful history and a thorough physical examination are necessary to clarify the exact injury to adjacent soft tissue, muscle, nerve, vascular tissues, and facial skeleton. A laceration of the salivary gland or main duct usually results in the presence of saliva in the wound. Ductal

injurices should be repaired, if at all possible, prior to any efforts to repair concomitant facial lacerations.

Facial nerve injuries are also of great importance, and careful assessment of facial muscle function is a high priority - with attention to the forehead, eyes, nose, and mouth. The patient should be asked to smile, show the teeth, pucker the lips, close the eyes tightly, and wrinkle the forehead. All details should be recorded as soon as possible to avoid confusion concerning neurologic deficits that might occur at a later date after the injury (and, therefore, carry a different significance).

Penetrating wounds of the lower face carry a high potential for injury to the parotid or submandibular glands. Knife or shotgun wounds and human or animal bites place the site of the wound at risk for serious infection. In addition to the management of the initial injury, the job is not complete until tetanus prophylaxis (and rabies investigation in appropriate circumstances) has been considered.

The major point to be emphasized in assessing and treating lacerations around the parotid is that early recognition and repair of major salivary ducts or primary branches of the facial nerve is the key to a successful outcome. These surgical methods require the use of microsurgical techniques, and referral to an otolaryngologist and head and neck surgeon is appropriate.

Penetrating Neck Injuries

Stab and gunshot wounds are the main causes of *penetrating neck injuries.* Emergency management of patients with these injuries focuses upon protecting or restoring the airway, control of bleeding, and prevention or treatment of hypovolemic shock during the initial minutes of stabilization and evaluation.

Attention is then focused on assessment of the exact nature of the deeper injuries, with particular attention directed to systems that may be disrupted. Airway injury is indicated by hemoptysis, hoarseness, crepitus, or sucking wounds. Pharyngeal and esophageal injury is suggested by dysphagia, crepitus, tachycardia, and fever. The findings with vascular injury are an expanding hematoma, central nervous system deficit, pulse deficit, thrills, bruits, shock, or persistent bleeding. Particular importance should be attached to hoarseness, cranial nerve deficit, or hemiplegia.

In addition to the careful history and physical examination, direct laryngoscopy may be necessary to rule out lacerations of the larynx or pharynx and injury of the recurrent laryngeal nerves.

Radiographic assessment of the pharynx or esophagus is accomplished by having the patient swallow contrast material (thin barium still used most commonly). Angiography is valuable for diagnosis and may be useful in treatment when embolizing techniques are needed. Even though nearly all (92 per cent) of the carotid arteriograms will be negative in these patients, this study is indicated in all patients with central neurologic findings (if their clinical status permits it) because of the importance of a positive finding.

In earlier years (prior to World War II), almost all patients were managed by careful observation and surgery was performed only if the patients' conditions worsened. During World War II, a policy of mandatory neck exploration decreased the mortality from these wounds but carried a 60 per cent rate of explorations with negative results. Currently, the policy of selective exploration is employed based on the presence of any of the following:

1. Any patient with unstable vital signs.

2. Any patient with evidence of airway injury (hemoptysis, hoarseness, crepitus, or subcutaneous emphysema).

3. Evidence of pharyngeal-esophageal penetration (dysphagia, crepitus, positive contrast swallowing study).

4. Signs of vascular disruption (expanding hematoma, bruit, thrill, positive angiogram).

5. Patients with neurologic deficit (cranial nerve deficit, hemiplegia, and so on).

Basically, exploration is performed as a means of determining precisely the nature of the injury and of repairing nerves, vessels, and soft tissue of the airway and pharyngoesophageal passages. Maintaining a high degree of suspicion and promptly pursuing the diagnostic steps are the key elements of managing this patient appropriately.

Thyroid Nodules

Thyroid nodules are most often due to single or multiple colloidal goiters. Other causes include thyroglossal duct cysts, toxic nodular goiters, adenomas, and thyroid carcinoma. Benign tumors must be differentiated from thyroid malignancies. Palpation of a thyroid nodule should be followed by performance of a radioiodine scan to determine whether the lesion is "hot" or "cold". Cold nodules absolutely require either fine needle aspiration or open biopsy to determine the histology of the lesion.

Thyroid Carcinoma

Thyroid carcinoma arises from either epithelial or medullary tissue. Epithelial carcinomas are of three types: (1) papillary, (2) follicular, and (3) anaplastic. The great majority of lesions are papillary; both follicular and anaplastic lesions are unusual. Anaplastic lesions are most commonly seen in elderly patients and have very poor prognosis. Most other thyroid malignancies are treated surgically and have a relatively favorable prognosis.