40. Otolaryngology

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Otolaryngology is a regional surgical specialty devoted to the study of the head and neck. An understanding of head and neck anatomy, physiology, and pathology is necessary to manage the diverse disorders ranging from hearing and communication abnormalities to facial plastic and reconstructive problems.

Ear

Sound Physics

Sound waves travel as alternating compressions and rarefactions of the elastic medium through which they are transmitted (sound travels in air at 1100 ft/s (~336 m/s)). Sound waves exhibit several physical characteristics, including amplitude and frequency, that are related to the subjective psychoacoustic attributes of loudness and pitch. The intensity range of human hearing is great: The most intense tone that can be perceived as sound is several billion times louder than the faintest detectable one.

The auricle serves to localize sound in space and to amplify sound waves impinging on the tympanic membrane through the resonance capabilities of the external auditory canal. Sound waves strike the tympanic membrane and produce in-and-out vibrations that transmit sound energy to the ossicular chain. The tympanic membrane protects the round window from simultaneous sound exposure by interposing an air-filled middle ear space. With a large perforation of the tympanic membrane, sound simultaneously strikes the round window and the tympanic membrane, decreasing the energy transmitted to the oval window.

Acoustic energy at the tympanic membrane is transformed by the malleus, incus, and stapes into energy within the oval window perilymph. Sound is amplified by the lever mechanism of the ossicular chain as a function of the large ratio of the tympanic membrane area to the area of the small stapes footplate. Therefore, the middle ear and ossicular system act as an efficient device for transferring acoustic energy from one elastic medium (air) to another medium of different impedance (fluid).

Movement of the stapes footplate produces a traveling wave in the scala vestibuli perilymph that is propagated along the basilar membrane of the cochlea from the base to the apex and down the scala tympani to the round window. Vibration of the basement membrane produces a shearing movement between the tectorial membrane and the hairs of the hair cells, generating electrical activity within the cochlea and auditory nerves. The point of maximal displacement of the basilar membrane is dependent upon the frequency (or pitch) of the wave. High-frequency tones cause maximal displacement near the base of the cochlea, and low-frequency tones cause maximal stimulation near the apex.

Cochlear frequency encoding involves depolarization of afferent neurons that synapse in the cochlear nuclei. The attendant perception of loudness and pitch depends upon the total number of neurons activated and their frequency specificities.

Clinical Audiology

Sound carried by air to the ear and perceived in the normal way represents hearing by **air conduction.** Tests of hearing by air conduction provide information about the patency of the external auditory canal, the efficiency of sound transmission by the ossicular chain, and the integrity of the cochlea, acoustic nerve, and central auditory pathway. A defect in the auditory system from the external auditory canal to but not including the cochlea produces a **conductive hearing loss** and raises the intensity threshold for perception of sound. The term conductive hearing loss applies only to air conduction. Serous otitis media is the most common cause of conductive hearing loss in children. Other causes include ceruminous impaction in the external auditory canal, large tympanic membrane perforations, ossicular chain discontinuities from trauma or infection, otosclerosis, and temporal bone neoplasms involving the external auditory canal or middle ear.

If the sound source is placed on the skull or teeth, the vibration will directly stimulate the cochlear perilymph and bypass the external and middle ear. **Bone conduction** hearing tests thus examine the integrity of the cochlea, eighth nerve, and central auditory pathway. Bone conduction hearing losses are secondary to lesions of the sensory (cochlea) and neural (acoustic nerve) compoentns of the auditory system and are designated **sensorineural** hearing losses. The most common cause of sensorineural hearing loss is aging (presbycusis), which is associated with progressive loss of outer hair cells within the organ of Corti and neural degeneration. However, noise exposure, ototoxic drugs including neomycin and other aminoglycosides, temporal bone fractures, labyrinthine infections, and arterial insufficiency may also produce sensorineural hearing disorders. Composite or mixed losses have both conductive and sensorineural elements.

Hearing is clinically evaluated by a careful history and physical examination that includes tuning fork testing, whispered voice assessment, and audiometric analysis. Because of the importance of hearing in speech acquisition and intellectual maturation, it is critical to diagnose disorders early. By noting the verbal respondes to phonetically balanced words (eg, baseball, hot dog, cowboy, railroad), the examiner can grossly establish the level of hearing loss in each ear. The degree of loss may be estimated from Table 41-1 by determining the voice level at which the words are no longer perceived.

It is important to provide masking (noise) by rubbing the tragus or hair in front of the ear not being tested when the loud whispered level is reached. Otherwise, sound may cross over to the nontested ear, yielding a false result.

Further characterization of the type of hearing loss requires the use of tuning forks to differentiate a conductive from a sensorineural defect. In the **Weber test,** placement of a 512-Hz tuning fork on the skull in the midline or on the teeth stimulates both cochlea simultaneously. If the patient has a conductive hearing loss in one ear, the sound will be perceived loudest in the affected ear (ie, it will lateralize). With a unilateral sensorineural hearing loss, the tone is heard in the unaffected ear. The **Rinne test** compares air conduction (AC) with bone conduction (BC) in one ear, and commonly utilizes the 256- and 512-Hz tuning forks. Sound stimulation by air in front of the pinna is normally perceived twice as long as sound placed on the mastoid tip (ie, AC > BC). With conductive hearing loss, the duration of air conduction is less than bone conduction (ie, BC > AC; negative Rinne test).

In the presence of a sensorineural hearing loss, the duration of both air conduction and bone conduction are reduced; however, the 2:1 ratio remains the same (ie, AC > BC; positive Rinne test). The results of these 2 tuning fork tests are synthesized to determine the type of hearing loss.

Table 41-1. Estimation of hearing loss by voice test. The degree of hearing loss may be estimated by determining the voice level at which the patient understands the examiner's voice. If the patient hears the examiner's whispered voice distinctly, hearing is probably normal.

Examiner's Voice Level	**Decibel Equivalent**	**Degree of Loss**
Soft whispered voice (inaudible to examiner)	20 dB	Mild
Moderate whispered voice (just audible to exam)	35 dB	Mild to moderate
Loud whispered voice	40-50 dB	Moderate
Soft spoken voice	60 dB	Moderate to severe
Moderately loud spoken voice	70-80 dB	Severe
Loud spoken voice	90-120 dB	Very severe to totally deaf.

Further quantification of the type and degree of hearing loss and the ability to hear and understand speech requires pure tone audiometry, speech reception threshold testing, and speech discrimination analysis. An audiometer is an electronic device capable of delivering pure sound frequencies by both air and bone conduction from 125 to 8000 Hz at intensities ranging from 0 to 110 decibels (dB) in 5-dB steps. The decibel is a logarithmic ratio of 2 sound pressure levels (SPL) or intensities that are related to a reference intensity, measured in dynes per square centimeter. The threshold intensity of sound perception in a normal individual is 0 dB. Pure tone audiometry in such a person will demonstrate at all frequencies normal thresholds for both air and bone conduction. On the other hand, a conductive hearing loss raises the threshold for sound perception in air. With air conduction hearing loss of 60 dB, for example, the threshold is 1 million-fold greater than normal. Sensorineural hearing losses result in equal increases in the threshold for air conduction and bone conduction and produce a characteristic audiogram.

Speech perception can be evaluated by presenting the patient with a list of phonetically balance bisyllabic spondee words (eg, baseball, railroad) at an intensity corresponding to 50% comprehension. The resulting speech reception threshold (SRT) should approximate the average hearing levels for the speech frequencies of 500, 1000, and 2000 Hz. These frequencies are extremely important for understanding conversation in English and fall within the 300- to 3000-Hz transmission range of the telephone. The ability to discriminate speech is ascertained by presenting a list of 50 phonetically balanced monosyllabic words 25-40 dB above the speech reception threshold. The percentage of words the patient repeats correctly is called the discrimination score and should normally be between 90% and 100%. In conductive hearing loss, the discrimination score decreases as the peripheral analysis of sound is impaired.

Because the previous tests require a voluntary response, they are of little use if age or illness prevents the patient from performing the required task. For example, infants with a high risk of hearing loss (eg, congenital rubella) are unable to give voluntary responses; therefore, an objective method of determining auditory thresholds is necessary to fully evaluate a suspected hearing loss. Evoked response or brain stem audiometry measures electrical responses from the acoustic nerve, cochlear nuclei, and inferior colliculi from the surface of the scalp. These techniques are particularly useful in evaluating patients with suspected sensorineural hearing losses secondary to acoustic neuroma, cerebellopontine angle tumor, or a brain stem lesion. Brain stem evoked response audiometry is especially important in newborn nurseries for identifying infants at risk of hearing impairment. These include low-birth-weight infants (less than 1500 g), infants with low Apgar scores, and infants with hyperbilirubinemia (greater than 20 mg/dL) or neonatal meningitis.

Conductive Hearing Loss

1. Otitis Media With Effusion

The term nonsuppurative otitis media denotes a broad range of middle ear effusions characterized by an inflammatory exudate and various amounts of mucus. Several distinct types are recognized: (1) serous otitis media - a sterile, pale, low viscosity transudate; (2) secretory otitis media - a chronic "glue" ear with infiltration of lymphocytes, histiocytes, plasma cells, leukocytes, and markedly increased glandular production of mucus; and (3) aerotitis media secondary to barotrauma or direct temporalbone injury. Although many factors may be implicated, the common denominator appears to be auditory tube (eustachian tube) dysfunction. The physiologic role of the auditory tube is to protect the middle ear from nasopharyngeal secretions, clear middle ear secretions into the nasopharynx, and, more importantly, ventilate the middle ear space. Auditory tube dysfunction develops from barotrauma when the nasopharyngeal pressure exceeds middle ear pressure during rapid descent or from adenoidal hypertrophy that produces lymphatic obstruction of the auditory tube. In addition, over half of children with cleft palate deformities manifest auditory tube dysfunction secondary to malfunction of the tensor veli palatini muscle.

If auditory tube function is compromised - as it frequently is in young children - early diagnosis and treatment is critical to prevent impairment of speech development. On physical examination, the tympanic membrane is retracted, the short process of the malleus is extremely prominent, and the light reflex is frequently lost. Pneumatic otoscopy discloses marked reduction of tympanic membrane mobility, and a characteristic yellow or amber color is often seen in the middle ear space accompanied by air bubbles.

Unfortunately, otoscopy is not a reliable method of assessing middle ear effusions. The development of impedance audiometry, however, has markedly improved the early diagnosis and treatment of otitis media with effusion. The overall compliance of the tympanic membrane and middle ear system, which varies inversely with its impedance, is measured by delivering to the tympanic membrane a continuous 220-Hz tone signal via a sealed probe tip and recording the amount of energy reflected from the surface. The pressure in the external auditory canal is varied - from +400 mm water to -400 mm water - and the reflected energy is recorded. The resultant tympanogram correlates well with the presence or absence of effusion. A normal type A tympanogram is characterized by a peak compliance at 0 mm

water pressure; the absence of a peak of maximal compliance is commonly encountered in middle ear effusions (type B tympanogram). By contrast, type C tympanograms exhibit a peak of maximal compliance less than -100 mm of water and are chiefly associated with a retracted tympanic membrane with or without effusion.

Intial management of otitis media with effusion consists of identifying the cause. In adults, a malignant neoplasm of the nasopharynx such as carcinoma or lymphoma should be carefully excluded. In children, allergic rhinitis or adenoidal hypertrophy should be considered. Conservative treatment with sympathomimetic amines and anithistamines is attempted first. Failure of medical treatment necessitates surgical intervention with myringotomy and insertion of ventilating tubes. Current indications for sustained middle ear ventilation include (1) significant conductive hearing loss secondary to persistent middle ear effusion; (2) persistent tympanic membrane atelectasis and negative middle ear pressure less than 150 mm water; or (3) prevention of recurrent acute otitis media refractory to prophylactic antibiotic therapy. Finally, adenoidectomy per se, although recommended by many physicians, does not statistically reduce the incidence of otitis media with effusion.

2. Chronic Otitis Media

The long-term sequela of chronic auditory tube dysfunction is chronic otitis media, which involves chronic perforation of the tympanic membrane that may or may not be associated with acute suppuration or destruction of the ossicular chain, or both. The perforation of the tympanic membrane may take 2 forms: (1) a central (or safe) perforation, in which a remnant of the tympanic membrane is interposed between the rim of the perforation and the annulus of the tympanic membrane; and (2) a marginal (or dangerous) perforation, in which the annulus of the tympanic membrane has been destroyed, primarily in the posterosuperior quadrant. In the former case, the middle ear is usually dry; in the latter, suppuration commonly occurs. The pars flaccida is frequently involved in marginal perforations, and central perforations are restricted to the pars tensa. However, suppuration may develop in either situation as a result of the introduction of staphylococci or gram-negative rods (commonly *Pseudomonas*) via the auditory tube or external auditory canal. Foul-smelling otorrhea should be treated by vacuum drainage of the external auditory canal and instillation of 2% aqueous acetic acid drops in the external auditory canal. In recalcitrant cases, parenteral azlocillin, ticarcillin, or ceftazidime may prove beneficial if no cholesteatoma is present.

Complications of chronic otitis media such as seventh nerve paralysis, labyrinthitis, or intracranial suppuration are less frequently associated with central perforations than with marginal ones. If squamous epithelium migrates into the middle ear or mastoid, a keratinizing aural cholesteatoma develops. The desquamating epithelium produces bone-destroying collagenase and tends to remain infected. The associated middle ear inflammation interferes with the tenuous blood supply to the stapes and the long process of the incus, resulting in ossicular desctruction with a conductive hearing loss of 50-60 dB. White amorphous debris is often observed in the pars flaccida. Radiography of the temporal bone may demonstrate an unsuspected large radiolucent defect secondary to bone destruction. If untreated, a cholesteatoma may progressively destroy the ossicular chain and erode into the inner ear, producing profound hearing loss.

Bacterial invasion of the cranium from an infected cholesteatoma may occur as a result of osteitis or thrombophlebitis or along a preformed pathway. In the preantibiotic era, the onset of severe temporoparietal headache and nuchal rigidity in a patient with chronic otitis media was an ominous sign. The most common intracranial complication of chronic otitis media is meningitis, secondary to either pneumococcus or another streptococcal organism. Additional potential problems include epidural abscess, temporal lobe abscess, cerebellar abscess, lateral sinus thrombosis, subdural empyema, and otitic hydrocephalus. These complications can be prevented by early surgical treatment of chronic otitis media and cholesteatoma.

Simple central tympanic membrane perforations are repaired by grafting the tympanic membrane with fascia temporalis or any mesenchymal tissue. Inthe absence of cholesteatoma, associated ossicular discontinuity is repaired with an autogenous homograft or alloplastic materials to reestablish the sound-transforming capability of the middle ear. With advanced middle ear and mastoid disease and associated cholesteatoma, more radical surgery is required. In a radical mastoidectomy, the remnants of the tympanic membrane, ossicles, and cholesteatoma are removed and the mastoid air cells, antrum, and middle ear are converted into an open cavity that is periodically inspected and cleaned. If the cholesteatoma lies above and superficial to the tympanic membrane and middle ear ossicles, either an intact canal wall mastoidectomy or modified radical mastoidectomy is performed. The primary goal of each is to eradicate infection and provide a temporal bone free of cholesteatoma.

3. Otosclerosis

In otosclerosis, a localized disease ofthe otic capsule, new spongy bone replaces normal bone, producing ankylosis or fixation of the stapes footplate. The resulting conductive hearing loss starts insidiously in the third and fourth decades of life and progressively involves both ears in 80% of individuals. Otosclerosis is an inherited disease, more common in whites and in women; in adults with normal-appearing tympanic membranes, it is the most common cause of progressive conductive hearing loss. The hearing loss may be treated in selected cases by microsurgical removal of the stapes and reconstruction with a wire prosthesis (4.5-5 mm in length) crimped over the long process of the incus. The medial end of the prosthesis is placed over a vein, fascia, or fat graft inserted in the oval window. Stapedectomy corrects the conductive hearing loss in most patients and may cause a minor sensorineural hearing loss in less than 1%.

Sensorineural Hearing Loss

Disorders affecting the cochlea and auditory neurons distort the perception of sound, producing a sensorineural hearing loss. The deficit is generally greater in the higher frequencies and is associated with decreased speech discrimination scores (ie, ability to understand complex speech is impaired).

The most common cause of sensorineural hearing loss is **presbycusis,** a gradual deterioration that starts after 20 years of age in the highest frequencies and involves all speech frequencies by the sixth and seventh decades. The impaired hearing stems from degenerative changes in the hair cells, auditory neurons, and cochlear nuclei. Tinnitus (ringing in the ear) is a common complaint. Sound amplification with an electrical hearing aid may benefit

patients with relatively good speech discrimination (a score greater than 60%). Profound bilateral sensorineural hearing loss may be treated with an implanted device, the cochlear implant.

Injury to the inner ear or acoustic trauma from a sudden very loud noise (ie, painful; greater than 140 dB) may produce a permanent sensorineural hearing loss. More important, prolonged exposure to intense nonpainful sound such as industrial noise above 90 dB will result in destruction of the outer hair cells of the organ of Corti and a sensorineural hearing loss that characteristically affects perception at 4000 Hz. These patients also experience tinnitus. Further noise exposure should be prevented with ear protectors (earmuffs or earplugs).

Additional causes of sensorineural hearing loss include diabetes mellitus, cochlear artery insufficiency, aminoglycosides, ototoxic diuretics (eg, ethacrynic acid), and tumors of the vestibular nerve (vestibular schwannoma) or cerebellopontine angle. Rubella in the first trimester of pregnancy, Rh incompatibility, birth trauma, hyperbilirubinemia, prematurity, congenital syphilis, meningitis, or congenital anomalies are often associated with defective hearing, and infants at risk should be tested early by evoked response audiometry. Unfortunately, over 90% of infants with congenital sensorineural hearingloss have no known risk factors. Hearing loss in these children is caused by a non-X-linked recessive mutation and is associated with no obvious physical abnormalities. If profound hearing loss antedayes the acquisition of speech, oral communication is severely impaired.

Otalgia

Ear pain may be caused by a primary disorder of the ear or may be referred from structures with a common sensory innervation. Inflammation of tissues innervated by the fifth cranial nerve - including the nose, paranasal sinuses, nasopharynx, mandible, and salivary glands - may produce otalgia. Inflammatory lesions of the oropharynx, the larynx, and the base of the tongue are commonly associated with otalgia. Unfortunately, however, neoplastic lesions do not produce otalgia until they are significantly advanced.

Inflammation of the external auditory canal **(otitis externa)** is commonly caused by bacteria (*Proteus mirabilis, Pseudomonas* sp, staphylococci) and occasionally by otomycoses (*Aspergillus niger, Candida albicans*). Predisposing causes are water immersion, high humidity, instrumentation in the external auditory canal, and ceruminous impaction. Patients complain of otalgia, pruritus, otorrhea, and decreased hearing and intermittent blockage of the canal. Pain on traction of the pinna or tragus differentiates otitis externa from acute otitis media. Hyperemia, edema, and otorrhea are seen on inspection of the external auditory canal. Treatment consists of precise debridement, topical broad-spectrum antibiotics (eg, polymyxin B and neomycin), and hydrocortisone to reduce canal wall inflammation. In neomycin-sensitive individuals, topical 2% aqueous acetic acid is effective. Contact with water should be avoided, and dry heat hastens resolution. Diabetics are particularly at risk of developing *Pseudomonas* mastoiditis. They are treated with immediate debridement and an intravenous semisynthetic penicillin such as mezlocillin, piperacillin, or azlocillin.

Inflammation that progresses to involve the auricular appendage may result in **perichondritis** and then **chondritis** with cartilaginous necrosis. Perichondritis or chondritis

of the pinna may also follow auricular trauma and hematoma, frostbite, or surgical drainage of a furuncle of the external auditory canal. It is manifested by edema, erythema, and tenderness of the pinna. Treatment consists of systemic penicillin and incision and drainage of any hematoma or abscess. Cotton soaked in an antiseptic solution (eg, povidone-iodine) is placed within the recess of the auricle, and a mastoid dressing is applied. Failure to adequately drain an auricular hematoma or abscess will result in a "cauliflower ear" deformity secondary to cartilaginous destruction and fibrosis.

Acute Otitis Media

Acute suppurative otitis media is a very common problem in pediatric and family practice. Twenty percent of children under 8 years of age experience at least one episode. Recurrence is common, especially if the initial episode occurs during the first 12 months of life. Viruses may produce otitis media, but suppuration is predominantly caused by bacteria, specifically *Streptococcus pneumoniae, Haemophilus influenzae,* or *Staphylococcus aureus. H influenzae* infection occurs more commonly in the age group under 5. In the newborn, gram-negative infections with *Escherichia coli, Klebsiella pneumoniae,* and *P mirabilis* predominate.

The presenting clinical signs and symptoms are variable: In adults, otalgia and a conductive hearing loss are most common; infants may exhibit only fever, lethargy, or irritability. Spontaneous perforation of the tympanic membrane with purulent otorrhea and hemorrhage are often present in infants when first seen by a physician. Prompt antibiotic therapy hastens resolution of the disease and prevents the development of temporal bone and intracranial complications. In nonallergic patients over 6 weeks of age, ampicillin or amoxicillin is recommended, and erythromycin and sulfisoxazole may be used in penicillin-allergic individuals. Patients who fail to respond probably have an infection caused by ampicillin-resistant *H influenzae* type B and should be given trimethoprim-sulfamethoxazole, Augmentin (amoxicillin and calvulanic acid), or a cephalosporin, such as cefaclor. In infants under 3 months of age, therapy must also be directed against enteric organisms. Ampicillin parenterally, combined with cefotaxime or chloramphenicol, is preferred. Either combination may be given parenterally to patients with impending otologic or intracranial complications, such as meningitis, labyrinthitis, or lateral sinus thrombosis. Cefuroxime and ceftriaxone may prove to be effective alternatives. Topical vasoconstrictors, such as phenylephrine, should be instilled into the nasal cavities, and systemic vasoconstrictors, including ephedrine, pseudoephedrine, or phenylpropanolamine (Propandrine), may be prescribed to improve auditory tube function. Myringotomy is indicated to relieve severe pain unresponsive to narcotics, or to identify antibiotic-resistant organisms. The development of sudden facial paralysis is an indication for emergency myringotomy. The incision should be made in the posteroinferior quadrant, midway between the umbo and the annulus.

Acute Mastoiditis

Acute mastoiditis is a complication of acute otitis media and develops as a result of pus retention in the mastoid. The destruction of bony septa results in a coalescence of mastoid air cells and subsequent erosion of the cortices of the mastoid process of the temporal bone. Otalgia, aural discharge, and fever are characteristically seen, and examination reveals severe mastoid tenderness, lateral displacement of the pinna, or postauricular mastoid swelling

secondary to subperiosteal abscess. Antibiotics must be given, and if a subperiosteal abscess develops, the mastoid and its air cells must be surgically drained. A complete mastoidectomy through a postauricular incision includes exenteration of the infected bone and pus and inspection of the dura of the posterior and middle cranial fossa to exclude epidural abscess.

Foreign Bodies in the Ear

Foreign bodies of the external auditory canal frequently traumatizes the epithelium and may also perforate the tympanic membrane or disrupt the ossicular chain, producing a conductive hearing loss. All kinds of objects are inserted into the external canal, especially by children. In addition, insects such as cockroaches may enter the canal, attaching their pincers to the tympanic membrane. Most foreign bodies can be removed with a right-angled hook or forceps if they are not lodged significantly medial to the isthmus of the external auditory canal. Gentle expulsion of nonvegetable matter with a soft rubber syringe - similar to the removal of cerumen - is effective. In younger children or adults with firmly imbedded objects, a general anesthetic is necessary to avoid injuring the tympanic membrane or ossicular chain. Insects may be suffocated by instilling mineral oil into the external auditory canal.

Tinnitus

Tinnitus is the subjective sensation of noise in the ear or head not of psychogenic origin. Tinnitus is reported by patients as a constant or intermittent buzzing, ringing, or humming sound. Tinnitus is experienced by patients with presbycusis, noise-induced hearing loss, ceruminous impaction, and otitis media (acute and chronic). Exposure to ototoxic drugs, endolymphatic hydrops (Ménière's disease), and vestibular schwannoma (acoustic neuroma) are other causes. Highly vascular lesions of the temporal bone may elicit pulsatile tinnitus; examples are glomus jugulare or tympanicum tumors (non-chromaffin-producing neoplasms of paraganglionic cell origin). Aneurysms or arteriovenous malformations rarely cause true tinnitus. Stress, nicotine, and caffeine aggravate tinnitus. Treatment is determined by the primary disease, but since most cases are caused by presbycusis, effective therapy is often unavailable. Masking of tinnitus with frequency-specific hearing aids or extraneous noise such as a radio or stereo - especially before sleep - may be helpful in motivated patients. Biofeedback therapy has been used successfully in anxious patients. Recent success has been reported with an electrical radiofrequency transmitter transmitting audiofrequency across the skin.

Temporal Bone Fractures

Fractures of the skull base from blunt trauma commonly involve the temporal bone. Hemorrhagic otorrhea, ecchymosis of the postauricular area (Battle's sign), and disturbances in cochlear or vestibular function may be encountered. Eighty percent of fractures of the temporal bone are longitudinal to the petrous ridge; the remainder are transverse or perpendicular. Longitudinal fractures are chiefly secondary to parietal blows with the fracture line extending across the floor of the middle cranial fossa and through the roof of the external

auditory canal, rupturing the tympanic membrane. The incudostapedial joint is frequently disrupted and requires tympanoplastic reconstruction. The labyrinth is often spared, and only 35% of patients develop a sensorineural hearing loss. Twenty percent of patients develop delayed seventh nerve paralysis caused by ischemia and compression rather than neural disruption.

Transverse fractures, on the other hand, are caused by a blow to the occiput and are associated with a higher mortality rate. The fracture may involve the foramen magnum, pass through or near the jugular foramen, and cross the internal auditory canal to reach the foramen lacerum or spinosum. Often the fracture line will splinter to reach the medial wall of the inner ear. The tympanic membrane remains intact, with a blue-black hemotympanum, and cerebrospinal fluid rhinorrhea is not uncommon. In 38-50% of cases, the seventh nerve is lacerated, resulting in immediate facial paralysis. In addition, disruption of the membranous labyrinth leads to complete loss of cochlear-vestibular function and subsequent sensorineural hearing loss and vertigo.

Because of the potential risk of meningitis, all temporal bone fractures should be immediately treated with prophylactic penicillin and, if there is a cerebrospinal fluid leak, head elevation, fluid restriction, and diuretics. The development of progressive facial paralysis requires immediate neuronal decompression of the fallopian canal. If the nerve has been anatomically disrupted, debridement and end-to-end anastomosis or nerve grafting will be necessary.

Neoplasms of the External & Middle Ear

Squamous cell and basal cell carcinomas are the most common tumors of the pinna, occurring in sun-exposed individuals. Small lesions may be removed by V-wedge excision or radiation therapy. Tumors invading the cartilage require surgical excision. Squamous cell or basal cell carcinomas arising in the external auditory canal require wide excision for the best chance of cure. En bloc resection of the external auditory canal is possible for lesions not involving the middle ear or mastoid, whereas more invasive tumors require temporal bone resection. Radiation therapy is reserved for neoplasms that cannot be resected. Squamous cell carcinomas arising in the middle ear should be treated by resection of the temporal bone if feasible or radiotherapy if they cannot be excised.

Glomus jugulare and glomus tympanicum tumors are vascular neoplasms that usually arise from the jugular bulb and tympanic plexus, respectively. Both tumors spread cephalically and posteriorly into the middle ear and mastoid. Both are non-chromaffin-producing paragangliomas and are histologically the same as carotid body tumors, the most common chemodectomas of the neck. The natural history of these tumors is one of slow, progressive growth and gradual invasion of the jugular foramen and its nerves (cranial nerves IX, X, XI) as well as cranial nerves VII, XII, and VIII. The overall incidence of central nervous system invasion is less than 20%.

The principal manifestation of a tumor of the middle ear is conductive hearing loss, and pulsatile tinnitus is often present if the tumor is highly vascular. Brown's sign is frequently present (pulsation of the tympanic membrane that is inhibited by pressure applied to the tympanic membrane by a pneumatic otoscope). Digital subtraction angiography, CT

scan with contrast medium, and retrograde jugular venography are used to delineate the superior and inferior borders of the tumor. The treatment of choice is surgical removal if the lesion does not involve the carotid siphon and has not spread by distant metastases or direct extension into the cranium. Only 20% of the lesions can be cured by radiotherapy.

Congenital Deformities of the Ear

Lop ear, the most common congenital deformity of the auricle, is the result of failure of development of the antihelical fold or excessive protrusion of the conchal cartilage. Treatment consists of otoplasty, which is the surgical creation of an antihelical fold or reduction of conchal cartilage projection, or both. Treatment before 5 or 6 years of age, when the auricle is three-fourths adult size, avoids ridicule of the child by peers.

Preauricular cysts and sinus tracts are common unilateral or bilateral congenital defects found anterior to the upper helix or tragus. They develop following incomplete fusion of the auricular hillocks. Many subsequently become infected, requiring complete excision, which may be hazardous because of ramification of the sinus tracts near facial nerve branches. More severe congenital deformities of the auricle are less common. Microtia, with stenosis of the external auditory canal, and complete atresia of the auricle and external auditory canal are unusual. Deformities frequently associated with developmental anomalies of the middle ear produce profound conductive hearing loss. Initial treatment requires first establishing the presence of adequate hearing in the opposite ear. If hearing in the opposite ear is normal, surgical repair is not recommended, because the potential risk to the seventh nerve, which takes an anomalous course through the temporal bone, is too great. If there is profound conductive hearing loss on both sides, however, surgical reconstruction of the middle ear is indicated if feasible; if not, bilateral bone conduction hearing aids should be used. Auricular reconstruction for severe microtia or complete atresia is a challenging cosmetic surgical problem, often requiring soft tissue flaps and autogenous cartilaginous grafts, and should be done prior to middle ear reconstruction.

Vestibular System

The vestibular system serves to maintain balance, posture, and spatial orientation in concert with vision and peripheral proprioception. Loss of 2 of these 3 sensory modalities is severely incapacitating. The vestibular end organs are dynamic structures that respond to linear acceleration (saccule and utricle) and to angular acceleration (semicircular canals). Angular acceleration of the head displaces endolymph and deflects hair cell cupulae in the cristae. Hair cell deflection results in either an increase or decrease in neuronal impulses to the vestibular nuclei. Because the 6 semicircular canals are arranged in 3 pairs with one member of a pair (right) lying in a plane parallel to the plane of the other member (left), differences in acceleration between the right and left side of the body are monitored in the vestibular nuclei. Normally, when resting or moving at a constant rate, sensory input from the paired horizontal semicircular canals and 2 pairs of posterior and superior semicircular canals is balanced. Angular acceleration with subsequent hair cell deflection results in unilateral increased vestibular output and increased muscle tone in the extraocular and skeletal muscles, maintaining balance, posture, and spatial orientation. Failure of the vestibular apparatus to sustain the organism's balance produces vertigo, nystagmus, falling, and past pointing. Sudden unilateral diminution of function in the vestibular system, in Ménière's disease, acute

labyrinthitis, or temporal bone fracture, causes an imbalance of neuronal information arriving in the temporal lobe cortex. The cortical interpretation is constant motion or vertigo. Similarly, neuronal imbalance arring at the extraocular motor nuclei and reticular formation produces rapid nystagmus and nausea, vomiting, and parasympathetic discharge. In response to overwhelming vestibular disequilibrium, the cerebellum inhibits vestibular nuclei, but only incompletely. Ultimately, restoration of balance will require (1) functional repair of diseased end organ, requiring hours or days; (2) central nervous system suppression of the normally functioning side; or (3) generation of new neuronal output in the hypofunctioning labyrinth.

Clinical Assessment of the Dizzy Patient

Nowhere in medicine and surgery is a carefully taken history more important than in evaluating patients with dizziness. Nonvertiginous or extravestibular dizzines is far more common than otogenic or true vertiginous imbalance. The sensation of lightheadedness or syncope points to a different cause than the perception of the room spinning around. The clinical evaluation of the vestibular system includes examination of cerebellar function (gait, Romberg, 2-step Fukuda, finger-to-nose, dysdiadochokinesias, and dysmetria testing) and the cranial nerves and observation of spontaneous or positional nystagmus. Because visual fixation with the eyes open may suppress nystagmus, it is important to record eye movements with electronystagmography. This is a technique for recording changes in the corneoretinal potential with skin electrodes and an electronic apparatus. Spontaneous, positional, and positioning nystagmus may be recorded to determine whether the vertigo is peripheral or central in origin.

Additional useful information is obtained by performing the Hallpike caloric stimulation test. Caloric stimulation of the labyrinth via the tympanic membrane induces convection currents within the horizontal semicircular canal, producing cupular deflection in one directin with 30°C water and in the opposite direction with 44°C water. With the patient supine and the head elevated 30 degrees, irrigation of the external auditory canal with cold water (30°C) produces the rapid component of nystagmus to the opposite side; warm water (44°C) produces nystagmus to the same side. The mnemonic device is COWS (*c*old to the *o*pposite and *w*arm to the *s*ame). Decreased vestibular response to caloric stimulation (canal paresis) indicates a vestibular end organ, vestibular nerve, or brain stem lesion.

Ménière's Disease

Ménière's disease, or endolymphatic hydrops, is characterized by a triad of symptoms of unknown cause: episodic vertigo, fluctuating sensorineural hearing loss, and tinnitus. The tinnitus is usually low-pitched and roaring. The hearing loss is more severe in the lower frequencies, frequently progresses over many years, and remains confined to one ear in most patients. Pathologically, there is generalized dilatation of the membranous labyrinth that includes the scala media and endolymphatic sac and is associated with occasional membrane breaks and intermingling of endolymph and perilymph.

Patients with severe hydrops should be treated with diuretics and salt restriction to prevent recurrent attacks. Antihistamines such as meclizine, dimenhydrinate, and cyclizine are used, as well as benzodiazepines such as diazepam to reduce the severity of vertigo. Associated vertigo, nausea, and emesis are controlled by combining the synergistic effects of a cholinergic antagonist (scopolamine) and an adrenergic agonist (dextroamphetamine).

Surgical treatment is currently reserved for patients with severe incapacitating vertigo or tinnitus or to prevent further deterioration of hearing. In patients with useful hearing, decompression of the endoplymphatic sac and insertion of a shunt between the membranous labyrinth and subarachnoid space improves symptoms in over half of cases. Vestibular neurectomy through a middle cranial fossa approach transects the superior and inferior vestibular nerves and is similarly successful. With severe loss of hearing and speech discrimination, a total transmastoid labyrinthectomy is used, which relieves vertigo in over 80% of patients.

Acoustic Neuroma

Acoustic neuromas account for about 8% of intracranial tumors and arise twice as often from the vestibular division of the eighth nerve as from the auditory division. Although acoustic neuromas account for about 80% of all cerebellopontine angle neoplasms, other lesions in the cerebellopontine angle may produce a nearly identical clinical picture. These include meningiomas, primary cholesteatomas, metastatic tumors, and aneurysms. Acoustic neuromas, which are derived from Schwann cells, initially produce tinnitus and a high-frequency sensorineural hearing loss. True vertigo is unusual. However, unsteadiness or balance disorders may develop as the tumor enlarges. Hallpike caloric testing commonly reveals canal paresis on the affected side. The acoustic reflex is frequently absent, and CT scan of the brain will reveal a contrast-enhanced lesion at the porus acusticus. Ultimately, magnetic resonance imaging (MRI) may prove more useful than CT in diagnosing cerebellopontine angle neoplasms.

Small intracanalicular tumors (within the internal auditory canal) may be surgically removed through the transmastoid labyrinthine route if no useful hearing remains; a middle cranial fossa approach is utilized to preserve serviceable hearing. Both routes maintain the integrity of the facial nerve. Larger tumors (> 3 cm) are removed via a suboccipital craniotomy; huge ones can only be removed via a combined suboccipital and translabyrinthine approach if the facial nerve is to be preserved.

Labyrinthitis

Acute suppurative labyrinthitis may develop as a complication of acute otitis media or meningitis. The microorganisms responsible for acute otitis media gain access to the inner ear via the oval and round windows, and microorganisms in the meninges enter through the cochlear aqueduct. The clinical manifestations are severe vertigo and a sudden profound sensorineural hearing loss, frequently followed by facial paralysis. Immediate surgical management with labyrinthectomy and radical mastoidectomy is necessary to prevent meningitis. In addition, certain viruses, including mumps, measles, influenza, and adenoviruses, that invade the inner ear may cause endolymphatic labyrinthitis and sudden

profound sensorineural hearing loss. In the prenatal period, rubella virus may attack the developing otic capsule and produce a severe congenital sensorineural hearing loss.

Facial Nerve

Facial Paralysis

Paralysis of the seventh nerve immobilizes the muscles of facial expression: The eye fails to close, the forehead does not wrinkle (as opposed to central or supranuclear facial paralysis with forehead sparing), and the angle of the mouth droops, so the patient drools. Peripheral seventh nerve paralysis suggests serious disease, such as tumor of the cerebellopontine angle, acoustic neuroma, facial nerve neuroma, neoplasm of the middle ear, and parotid gland malignancy. Acute otitis media, temporal bone fracture, and chronic otitis media with or without cholesteatoma may produce facial paralysis. Other causes include surgical trauma, Guillain-Barré syndrome, and herpes zoster oticus (Ramsay Hunt syndrome). When the cause is unknown, the condition is known as Bell's palsy. Although Bell's palsy is the commonest cause of peripheral seventh nerve paralysis, the pathogenesis is mysterious. Current theories implicate vascular ischemia and compressive edema within the facial canal as the cause of neuropraxia and cessation of axoplasmic flow.

All patients with facial paralysis should have a thorough history and physical examination. The following special diagnostic tests should be performed: pure tone audiometry, speech reception thresholds, and speech discrimination; mastoid films to detect erosion of the internal auditory canal or mastoid antrum; glucose tolerance test to rule out diabetes mellitus; and site of lesion testing. Site of lesion testing includes the Schirmer test of lacrimation (absent lacrimation indicates a lesion proximal to the geniculate ganglion); stapedius muscle reflex testing utilizing impedance audiometry (an intact stapedius reflex indicates a lesion distal to the horizontal portion of the facial nerve); and, occasionally, submandibular salivary flow studies (an 80% reduction in salivary flow compared with the normal side is an indication for surgical decompression of the nerve). Finally, nerve excitability testing over the peripheral branches and main trunk of the seventh nerve is often used to predict the success of surgical intervention. If neuronal function is completely lost, nerve excitability testing fails to elicit a motor twitch in the corresponding facial muscle at the same threshold as the uninvolved side. Ordinarily, this phenomenon is observed 72 hours after the initial injury. Progressive deterioration in nerve response carries a poor prognosis and necessitates surgical decompression of the nerve to the level of the lesion (ie, above or below the geniculate ganglion, the latter requiring a transmastoid approach and the former a middle cranial fossa approach).

The initial medical management of Bell's palsy is controversial. Approximately 70% of patients recover completely, but the prognosis for complete recovery falls to 10% in the presence of a dry eye. Early treatment with corticosteroids (eg, 60-80 mg of prednisone daily with gradual tapering over 7-10 days) is felt by some experts to hasten resolution of edema and improve the outcome (ie, prevent permanent paralysis or synkinesis).

Rehabilitation of the paralyzed face exhibiting no recovery is a challenging problem. Nerve crossover procedures using a hypoglossal to seventh nerve anastomosis are recommended for restoring resting facial tone. For traumatic lesions, interposition grafting of

the damaged segment with a greater auricular or sural nerve graft may be efficacious. Occasionally the nerve is long enough that an end-to-end anastomosis is possible. Finally, neuromuscular transfer techniques utilizing either temporalis or masseter muscle pedicles are effective for facial reanimation in selected cases.

Nose & Paranasal Sinuses

Nasal foreign bodies are common in children, who frequently place pebbles, beads, seeds, buttons, or paper into the nares. A severe inflammatory reaciton ensures, especially with organic matter, and this is associated with a foul-smelling unilateral nasal discharge. Removal of a foreign body requires topical vasoconstrictors such as phenylephrine and topical anesthesia with lidocaine or cocaine. General anesthesia may be needed in uncooperative children.

Nasal Vestibulitis

Inflammation of the nasal vestibule may assume 2 forms: a localized acute furunculitis or a chronic diffuse dermatitis. Acute staphylococcal furunculitis of the pilosebaceous follicles in the vestibule may develop into a spreading cellulitis of the tip of the nose. Treatment includes hot soaks and systemic penicillin. Incision and drainage of a localized abscess is rarely necessary, as the majority of cases will drain spontaneously. Diffuse nasal vestibulitis is treated with antibiotic ointment containing polymyxin B, bacitracin, and neomycin. Early treatment of all acute infections of the nose, paranasal sinuses, and face is important to prevent retrograde thrombophlebitis and cavernous sinus thrombosis.

Acute Rhinitis

Acute rhinitis (coryza, common cold) is often secondary to infection with respiratory viruses, including rhinoviruses and coronaviruses. It is associated with sneezing, watery rhinorrhea, tearing, malaise, and headache. Examination of the nasal mucous membrane reveals hyperemia, edema, and watery mucosal discharge. Later, the secretions may become thick and yellow-green in color. Tenderness to palpation over the paranasal sinuses may be found. Nonnarcotic anaglesics such as aspirin, decongestants, and antihistamines as well as fluids and rest will alleviate symptoms. Antibiotic therapy is not necessary unless secondary bacterial invasion occurs. The condition usually resolves within 5-10 days. Ultimately, antiviral therapy may be needed for control of the common cold.

Allergic Rhinitis

Antigens inhaled and deposited on the mucous membrane of the nasal cavities of hypersensitive individuals elicit and IgE-mediated rhinitis. This perennial or seasonal disorder is often associated with additional respiratory allergies such as asthma, chronic laryngitis, or tracheobronchitis. Allergens such as animal danders, molds, dust, and pollens are commonly implicated, and sensitivity to them may be confirmed by skin testing. Allergic rhinitis is characterized by sneezing, watery rhinorrhea, tearing, anosmia, and nasal obstruction. The

mucous membrane appears edematous and pale, with a thin discharge. Individuals with chronic allergic rhinitis commonly develop nasal polyps and acute or chronic sinusitis. Polyps may arise from the middle meatal region at the sinus ostia and appear as pale gray, glistening edematous masses within the nasal cavity. Occasionally, a large antro-choanal polyp arises from the maxillary sinus ostia in conjunction with chronic maxillary sinusitis and presents as a long pedunculated mass in the nasopharynx. Treatment requires removal of the polyp and drainage of the maxillary sinus.

The best management of allergic rhinitis is avoidance of known allergens. If the patient cannot avoid allergens or be desnsitized, then treatment with an antihistamine (eg, chlorpheniramine, brompheniramine, triprolidine) with or without a sympathomimetic amine (eg, pseudoephedrine, phenylephrine, or phenylopropanolamine) is indicated. Topical corticosteroids in the form of beclomethasone dipropionate (400 microg daily) delivered intranasally are effective in reducing or even eliminating nasal polyps in some patients. Alternatively, intranasal dexamethasone or injection of nasal polyps with a suspension of triamcinolone acetonide may be used. Surgical removal of polyps combined with surgical drainage of the involved sinuses should be done for severe nasal obstruction or chronic sinusitis unresponsive to medical therapy.

Rhinitis Medicamentosa

Misuse or abuse of intranasal vasoconstrictors (eg, phenylephrione, cocaine) may lead to mucosal edema, hyperemia, and watery rhinorrhea. The resulting nasal obstruction is severe, prompting the individual to increase the use of topical decongestants and thus perpetuate the cycle. Successful treatment requires complete cessation of intranasal medications for 2-3 weeks, and an oral decongestant such as pseudoephedrine must be used.

Vasomotor Rhinitis

Vasomotor rhinitis results from hyperreactivity of parasympathetic control of the nasal vasculature and glands. The vasomotor reaction is characterized by vascular engorgement, mediated by the release of acetylcholine (a powerful vasodilator) at parasympathetic nerve endings. This commonly occurs in response to changes in external temperature and humidity and is *not* caused by allergens. Exposure to inhalant irritants such as tobacco smoke and industrial pollutants may provoke parasympathetic activity. There is often a history of trauma. The patient complains of nasal obstruction, sneezing, and watery rhinorrhea. Systemic decongestants such as pseudoephedrine and phenylpropanolamine give some relief. Antihistamines help combat the problem through their anticholinergic action. Corticosteroid nasal spray is occasionally effective, while the use of intranasal cromolyn sodium may benefit the patient by preventing the release of mediators from mast cells. Surgical correction of the underlying traumatic deformity, such as a deviated nasal septum or nasal collapse, is a useful adjunct.

Paranasal Sinusitis

Inflammation of the paranasal sinuses is commonly precipitated by an acute upper respiratory tract infection of viral origin. Edema of the nasal mucosa produces obstruction of the sinus ostia, resulting in secondary bacterial invasion and localized pain, tenderness, and headache exacerbated by changes in barometric pressure. The microorganisms responsible for acute sinusitis in children and adults are most often *S pneumoniae* and *H influenzae.* In children, *Branhamella catarrhalis* may be playing an increasing etiologic role. In general, ampicillin, minocycline, Augmentin (amoxicillin and clavulanic acid), or amoxicillin combined with systemic and topical vasoconstrictors are the drugs of choice. In communities with a high incidence of ampicillin-resistant *H influenzae,* trimethoprim-sulfamethoxazole or a cephalosporin such as cefaclor is used. In penicillin-allergic individuals, erythromycin combined with sulfisoxazole or cefaclor may be used.

If pus remains in the maxillary sinus after medical treatment, the antrum should be irrigated with saline via a trocar passed either through the canine fossa or inferior meatus. This promotes drainage through the natural ostium. One must be careful not to pierce the orbital floor with the trocar.

Complications of acute sinusitis are rare except in infancy and childhood. Acute maxillary sinusitis and, more commonly, ethmoiditis may be complicated by orbital cellulitis and abscess formation. The progressive development of chemosis, scleral erythema, proptosis, and ophthalmoplegia points to orbital infection and potential intracranial invasion. CT scan with contrast enhancement can usually detect an orbital abscess and eliminate other causes of unilateral orbital proptosis. Treatment consists of intravenous ampicillin and chloramphenicol (or cefuroxime) along with sinusotomy and orbital drainage. Drainage of the maxillary sinus is established by a Caldwell-Luc approach in the gingivobuccal canine sulcus. A Lynch incision, midway between the dorsum of the nose and the medial canthal ligament of the eye, is used for ethmoidal disease. Drainage must be performed early to avoid serious intracranial complications such as meningitis, epidural or subdural abscess, cerebral abscess, and cavernous sinus thrombosis.

Acute frontal sinusitis is more common in adults and frequently occurs following nasal trauma involving the nasofrontal duct. In the adolescent, it is a major source of orbital infection. In cases refractory to medical management, treatment consists of trephination of the anterior floor of the frontal sinus inthe medial part of the eyebrow. Chronic frontal sinusitis unresponsive to medical treatment or development of a mucocele are other indications for operative treatment. A mucocele results from mucosal membrane duplication and obstruction of the sinus ostia secondary to chronic infection or trauma. It gradually enlarges to destroy the frontal bone and encroach upon the orbit or anterior cranial fossa. A bicoronal incision and osteoplastic flap are made and the frontal sinus is obliterated with fat or muscle after excision of the mucocele. Because of the frequent invasion by gram-negative bacteria (eg, *E coli, K pneumoniae,* and *P mirabilis*), *S aureus,* and anaerobic mouth organisms, an antibiotic with broader spectrum such as a cephalosporin or ampicillin is used to treat chronic sinusitis. If antibiotic therapy fails, chronic ethmoid and maxillary sinusitis in patients with pain, headache, and nasal polyposis are treated with external ethmoidectomy (Lynch incision) or the Caldwell-Luc procedure. Because a small percentage of chronic maxillary sinusitis are secondary to dental infection, radiographs of the apexes of the roots of the teeth should be obtained to rule out a periapical abscess in need of surgical drainage.

Epistaxis

The nasal cavity is a common site of spontaneous hemorrhage. The blood supply to the nose is derived from both the external and internal carotid artery systems. In 90% of cases, the epistaxis originates in the anterior nasal septum in the rich vascular plexus (Kiesselbach's plexus) in Little's area. The terminal septal branches of the anterior and posterior ethmoidal arteries arising from the internal carotid artery (via the ophthalmic artery) anastomose in this area, along with branches from the superior labial artery (via the external facial) and the sphenopalatine artery (via the internal maxillary artery). Both originate from the external carotid artery system. Because of their location, vessels on the anterior septal mucosa are readily susceptible to trauma from nasal picking, drying, crusting, and infection. Severe caudal septal deformities may lead to mucosal drying over the point of deflection, causing spontaneous hemorrhage.

Mild epistaxis from the anterior septum is readily controlled with digital pressure for 5-10 minutes. Persistent hemorrhage requires topical cocainization and cauterization with a silver nitrate applicator or electrocautery. If hemorrhage is still not controlled, 1/4-inch gauze packing impregnated with petrolatum should be placed atraumatically in the nasal cavity. Bleeding associated with leukemia, uremia, hepatic failure, coagulopathies, or hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber syndrome) should be treated with absorbable gelatin sponge (Gelfoam) soaked in topical thrombin, oxidized regenerated cellulose (Surgicel), or a wedge of pork fat inserted into the nasal cavity for 4-5 days. Treatment of the underlying coagulopathy, leukemia, uremia, or liver disorder is obviously important.

Posterior epistaxis from the termial branches of the sphenopalatine and internal maxillary arteries is more serious and is frequently associated with hypertension, diabetes, or major systemic vascular disorders. Successful treatment requires the use of both anterior and posterior packing, inserted with topical 4% cocaine anesthesia. The posterior pack is made by tying folded 4 x 4 inch gauze squares to the end of a catheter that has been passed transnasally and brought out through the mouth. Two strings are tied to the pharyngeal end of the catheter and brought out through the nares. They are then tied over an anterior nasal pack and bolster. The remaining third string is brought out through the mouth and taped to the cheek, where it can be grasped and removed in 4-5 days. This technique, however, is extremely uncomfortable, lowers arterial oxygen saturation, and may induce arrhythmias or an acute myocardial infarction in patients with severe cardiovascular disease. Therefore, transantral ligation of the internal maxillary artery, sphenopalatine artery, and descending palatine artery via a Caldwell-Luc approach is advocated in selected patients. Ligation of the internal maxillary artery may also be necessary in some situations where adequate nasal packing fails to control hemorrhage.

Nasal Trauma

Nasal fractures are the most common fractures of the maxillofacial skeleton and frequently are associated with septal fractures and epistaxis. Clinical findings commonly include periorbital edema and ecchymosis, displacement of the bony dorsum to the right with depression of the left nasal bone (secondary to a right hook), crepitus, and, occasionally, laceration of the dorsum. Because the other facial bones are often broken, the entire facial skeleton should be x-rayed.

Early reduction under local anesthesia before significant swelling appears produces an excellent result. Elevation with a periosteal elevator, combined with laterally applied digital pressure, is usually effective. An external plaster of Paris splint is applied, and antibiotic-impregnated packing is inserted into the nasal cavity. The packing is removed in 2-3 days and the external splint in 1 week. It may be necessary to reduce severely impacted nasal bones with Walsham forceps, one blade placed intranasally and the other extranasally. If the nasal fracture is encountered after severe edema has developed, it is better to postpone reduction for several days to allow resolution of edema. In children, the facial skeleton heals so fast that the fracture must be reduced within 4-5 days to avoid malunion. Manunion in the adult is treated by rhinoplasty and, in many cases, concomitant septoplasty to repair the deviated nasal septum.

Complications of nasal trauma include septal hematoma and abscess formation, septal perforation, septal deviation, and cerebrospinal fluid rhinorrhea secondary to fracture of the cribriform plate, the roof of the ethmoid sinus, the posterior table of the frontal sinus, or the sphenoid sinus.

A septal hematoma is a collection of blood underneath the mucoperichondrium or mucoperiosteum of the septum. Physical examination discloses a bulging red septum, and nasal obstruction is usually complete and bilateral. Unless immediately incised and drained, a staphylococcal abscess may develop that results in cartilaginous necrosis and saddle nose deformity. Intravenous nafcillin or oxacillin should be given to prevent cavernous sinus thrombosis and meningitis.

A septal deviation, especially along the nasal floor, produces varying degrees of nasal obstruction, depending upon the severity of deflection into the nasal cavity. The caudal end of the septum may be deflected into the nasal vestibule, causing obstruction or external deformity. Nasal septoplasty through a caudal submucoperichondrial incision is used to reconstruct and straighten the septum.

Septal perforations are repaired only if complicated by persistent epistaxis, crusting with nasal obstruction, or, rarely, whistling. The repair involves use of a fascia temporalis graft and advancement of 2 bipedicled mucoperichondrial flaps to cover the defect. Alternatively, a pedicle buccal mucosal flap or septal alloplastic button may be used.

Fractures of the nasal bones, nasoethmoidal region, and frontal region may occur in association with a dural defect and cerebrospinal fluid rhinorrhea. This provides a potential route for ascending infection and meningitis. The dural defect may communicate with the nasal cavity via the ethmoidal, frontal, or sphenoidal sinuses or the cribriform plate. A basilar skull fracture with an intact tympanic membrane may also present with cerebrospinal fluid rhinorrhea.

The diagnosis should be suspected by finding watery rhinorrhea with an increased glucose contant and may be confirmed by CT scan following subarachnoid instillation of metrizamide. The source of cerebrospinal fluid leak may be demonstrated in many cases by placing fluorescein dye (1 mL of 5% solution) in the lumbar subarachnoid space. Cocaine-impregnated neurosurgical cottonoids are inserted in the middle meatus, superior meatus, cribriform plate region, and auditory tube orifice. One hour later, the pledgets are removed

and examined for fluorescence under ultraviolet light. If a pledget placed in the nasal roof fluoresces, for example, the defect is confined to the cribriform plate region. If this fails to demonstrate the leak, CT scan combined with metrizamide introduced into the subarachnoid space may be useful. Five milliliters of isosmolar metrizamide is inserted into the lumbar space with the patient positioned head down. After 2 mionutes, coronal CT sections can be obtained with the patient in prone position.

Acute posttraumatic cerebrospinal fluid rhinorrhea is treated conservatively, with bed rest in the semisitting position, fluid restriction, diuretics, and penicillin. The patient should avoid straining, blowing the nose, sneezing, or vigorous coughing. Indications for surgery are persistent cerebrospinal fluid leakage of more than 6 weeks' duration, recurrent meningitis, pneumoencephalos, or intermittent leakage.

Small defects of the cribriform plate, fovea ethmoidalis, and sphenoid sinus are successfully repaired through an external ethmoidectomy incision using a variety of septal or middle turbinate mucoperiosteal flaps. Small defects of the posterior table of the frontal sinus are best managed by a bicoronal incision, osteoplastic flap approach, and fascia lata or temporalis fascia repair, accompanied by abdominal fat obliteration. Large defects will necessitate anterior fossa craniotomy and repair.

Other soft tissue facial injuries and fractures of the zygoma, maxilla, orbit, and mandible are discussed in Chapter 45.

Congenital Nasal Malformations

Congenital malformations of the nose and its appendages are unusual, Facial clefts, such as cleft lip and palate or a bifid nose, commonly result from genetic or teratogenic factors operating in the second month of fetal life. Although atresia and stenosis of the anterior nares is rare, it should be suspected in any infant who has difficulty breathing. Bilateral bony posterior choanal atresia is more commonly the cause of congenital neonatal respiratory impairment. Because they are obligatory nasal breathers during the first several weeks of life, newborns develop apnea and cyanosis when crying stops and the mouth is closed. The definitive diagnosis is confirmed by inability to pass a catheter transnasally.

Initial treatment includes either an oral endotracheal tube or McGovern nipple, followed by early transnasal or transpalatal correction of the atresia. More recently, the CO_2 laser has been used successfully in transnasally resecting the bony atresia plate. The surgically created posterior choana is kept patent with a 16F or 18F polyvinylchloride (Portex) endotracheal tube that is removed in 6-8 weeks. Unilateral choanal atresia, on the other hand, is usually not diagnosed until later in childhood or early adulthood and is associated with unilateral nasal obstruction or rhinorrhea. Repair is best performed when the nasal cavities and hard palate have reached adult size.

Other congenital lesions that may produce nasal obstruction include nasal gliomas, encephaloceles, meningoceles, and teratomas (dermoids, teratoids, true teratomas, and epignathi). Nasal gliomas are composed of neural and glial elements that may have intracranial connections through the cribriform plate, fovea ethmoidalis, or sphenoid bone. Similarly, meningoceles and encephaloceles may present as a nasal mass that may be

mistaken for a nasal polyp. Not infrequently, these heterotopic brain elements are seen as a mass on the nasal dorsum that is frequently confused with a midline dermoid cyst. Polycycloidal tomography and CT scans of the anterior cranial fossa and cribriform plate must be obtained to rule out intracranial connections. Treatment of heterotopic brain elements with an intracranial connection requires a combined craniotomy and transfacial approach. If the diagnosis remains in doubt, frontal craniotomy is performed before transfacial excision to avoid development of cerebrospinal fluid rhinorrhea and meningitis.

Neoplasms of the Nose & Paranasal Sinuses

Benign neoplasms of the nasal cavities and paranasal sinuses are rare. The most common benign lesions are of epithelial origin: the exophytic squamous papilloma and inverting papilloma. **Exophytic papillomas** arise primarily at the mucocutaneous junction within the nasal vestibule and should be excised with a small margin of normal tissue. **Inverting papillomas,** on the other hand, emerge almost exclusively from the lateral nasal wall as bulky vascular lesions with a marked tendency to invade and destroy bone. In 10-15% of cases, squamous cell carcinoma arising in the same anatomic area has been found. Wide surgical excision through a lateral rhinotomy approach is necessary to prevent local recurrence.

Malignant neoplasms of the nasal cavity and paranasal sinuses represent 0.2-0.3% of all cancers and 3% of all malignant tumors of the upper aerodigestive tract. Squamous cell carcinomas are the most common malignant neoplasms of the nasal cavities and paranasal sinuses. On the sun-exposed skin of the nasal tip and dorsum, basal cell carcinoma is more common. Early lesions are removed by local excision, using a Mohs chemosurgical technique. More advanced carcinomas necessitate wide local excision of underlying bone and cartilage with flap reconstruction.

Squamous cell carcinoma most frequently arises in the maxillary sinus and presents with unilateral nasal obstruction; foul-smelling, bloody rhinorrhea; and headache. Treatment consists of combined external beam radiation therapy and partial or total maxillectomy with or without orbital exenteration, depending upon the presence or absence of orbital invasion. Other neoplasms arising in the paranasal sinuses include lymphoma, adenoid cystic carcinoma, mucoepidermoid carcinoma, a variety of mesenchymal cancers, and adenocarcinoma. Adenocarcinomas tend to occur in the ethmoid air cells and may follow long-term exposure to wood dust or prior radiation therapy for bilateral retinoblastomas. Treatment requires combined radiation therapy and radical pansinusectomy for resectable lesions.

Oral Cavity

White Lesions of the Oral Cavity

The mucous membrane resembles the skin in that individual cells arise from the germinal layer and mature, but they do not keratinize. Mechanical, thermal, and chemical (eg, alcohol, nicotine) trauma may lead to thickening of the germinal layer and later development of a nonnucleated keratinized layer that appears as gray-white nonulcerated plaques on the oral cavity mucosa (leukoplakia). Because the histologic appearance varies considerably and 15% of these lesions may be premalignant, biopsy is indicated to rule out early carcinoma.

The treatment is surgical excision of small lesions with a knife or CO_2 laser and avoidance of further exposure to irritants. Larger lesions may require multiple excisions, cryosurgery, or wide-field laser excision.

Lichen planus, although primarily a dermatologic disorder, may involve any mucous membrane exposed to trauma; eg, the bucall mucosa frequently develops white papules or striae from repetitive biting. Hyperkeratosis, acanthosis, and subepithelial edema complete the histologic picture. Topical triamcinolone is often useful for associated submucosal inflammation. In some cases, this may undergo malignant degeneration into squamous cell carcinoma.

Finally, ill-fitting dentures may elicit white, raised folds of tissue in the gingivolabial sulcus that histologically consist of fibrous tissue proliferation and overlying epithelial hyperplasia. An inflammatory papillary hyperplasia on the hard palate may occur as a result of improperly fitting dentures. These polypoid lesions are hyperemic, soft, and mobile. Surgical removal is necessary followed by denture readjustment.

1. Inflammatory Diseases of the Gingiva

Inflammation of the gums (gingivitis) frequently develops as a result of poor oral hygiene, heavy smoking, and lowered resistance. **Acute necrotizing ulcerative gingivitis** (Vincent's gingivitis) is due to an overgrowth of the normal oral symbionts, a treponeme *(Borrelia vincentii),* and a fusiform bacillus. Clinically, it is characterized by painful hemorrhagic gums, ulceration, and a yellow-gray gingival pseudomembrane. Treatment consists of topical hydrogen peroxide, proper oral hygiene with removal of plaque and tartar at the teeth margin, and oral penicillin.

Although not limited to the gingivae, recurrent **aphthous stomatitis** (canker sores) frequently presents on the gingivae as round or ovoid, discrete, erythematous macules 2-20 mm in diameter that rapidly indurate but do not vesiculate (unlike herpetic lesions). They may elicit enough pain to interfere with mastication and speaking. Tetracycline compresses for 20 minutes 4-6 times a day and an analgesic (dyclonine, 0.5%) is effective treatment. The cause is uncertain, but the evidence suggests that the sores are viral in origin.

Trauma to a tooth may provoke inflammation of the periodontal membrane **(acute periodontitis),** rendering it tender to touch. If the traumatic stimulus is removed, the inflammation resolves. Recurrent gingivitis coupled with poor dental hygiene may cause chronic periodontitis, pyorrhea, and regression of the periodontal ligament from the neck of the tooth. The gingival sulcus deepens, pockets form between the roots of the teeth and the surrounding gingivae, and debris and tartar accumulate. Mild periodontitis is characterized by gingival erythema, edema, tenderness, and hemorrhage. Severe periodontitis is associated with gingival necrosis, halitosis, and loss of unstable teeth. Unless the cycle is broken, periapical abscess formation and tooth devitalization will continue. Frequent flossing and regular professional dental care are recommended.

2. Herpetic Stomatitis

Herpetic lesions of the oral cavity are divided into primary and recurrent labial herpes. Both are caused by herpes simplex virus and present as small vesicles that rupture, yielding a yellow-white superficial ulcer surrounded by a red halo. They are usually located on the labial and buccal mucosa, gingivae, and tongue. In severe cases, the gingivae are edematous and bleed readily. Although the primary disease is self-limiting, the virus remains dormant waiting to be activated by physical trauma and endogenous stress. Treatment is supportive, including topical anesthetics in solution or troche form. If herpetic stomatitis occurs as part of a disseminated infection, parenteral acyclovir is indicated.

3. Candidiasis

Oral candidiasis (thrush) is caused by the yeastlike fungus *C albicans* and characterized by a white membranous lesion closely adherent to the mucous membrane that bleeds and ulcerates when it sloughs. *Candida* is normally a part of the oral biota but may become pathogenic after prolonged administration of antibiotics; radiation therapy to the oral cavity or pharynx; immunotherapy for carcinoma, leukemia, or lymphoma; or immune deficiency associated with corticosteroid therapy, diabetes, hepatic disease, etc. In addition, candidiasis along with oral leukoplakia is identified in patients with AIDS. Oral nystatin is effective treatment. For immunocompromised hosts who develop systemic candidal infections, amphotericin B is recommended. Ketoconazole administered orally is the drug of choice for chronic mucocutaneous candidiasis.

Congenital Oral Cavity Malformations

1. Torus Palatinus & Torus Mandibularis

Torus palatinus, a common developmental abnormality of the oral cavity, consists of a bony exostosis of varying size and shape in the midline of the palate. Clinically, it may interfer with the proper fitting of dentures and require surgical removal. It must be distinguished from tumors of the minor salivary glands and fissural cysts of the palate.

Torus mandibularis, like its palatal counterpart, is a bony exostosis usually situated on the lingual surface of the mandible, adjacent to the cuspid and first bicuspid teeth. It is asymptomatic until an attempt is made at fitting a denture.

2. Macroglossia

Isolated macroglossia is rare and may be seen in cretinism, Down's syndrome, and acromegaly. It may also be caused by lymphangiomatous invasion of the tongue. Relative macroglossia is encountered in the Pierre Robin syndrome (micrognathia and cleft palate). The relatively large tongue may obstruct the upper airway, necessitating insertion of an oral airway or tongue-lip adhesion. The tongue base is sutured over an anterior neck button to assist in anterior displacement of the tongue. These measures are only necessary until the oral cavity enlarges enough to accommodate the tongue. Rarely is tracheostomy necessary.

3. Ankyloglossia

Tongue-tie or partial ankyloglossia is manifested by an abnormally short and thick lingual frenulum. Various degrees of ankyloglossia occur, ranging from mild restriction with only a mucous membrane band to thise in which both the frenulum and the underlying fibers of the genioglossus muscle are markedly fibrosed. Rarely, complete ankyloglossia with fusion of the tongue to the floor of the mouth may be encountered. Limitation of movement of the tongue tip results in malocclusion with an anterior "open bite" deformity, early prognathism, and swallowing and speech difficulties. Children with severe ankyloglossia meeting any of these criteria require frenulectomy, genioglossus myotomy, and mucous membrane closure with multiple Z-plasties.

4. Ranula

A **ranula** is a retention cyst in the floor of the mouth arising from the submandibular or sublingual ducts or from minor salivary glands. The cyst enlarges gradually, penetrating the deep structures of the floor of the mouth above the mylohyoid muscle. It should be excised if it is small; marsupialization is necessary for large cysts, owing to their multiple ramifications.

Salivary Glands

Chronic Sialadenitis

Chronic or recurrent bacterial parotitis may develop as a result of antecedent acute suppuration or viral inflammation. More commonly, however, there is a history of ductal obstruction. Recurrent bacterial invasion of the parotid gland leads to destruction and fibrosis of acini with ductal ectasia. The subsequent decrease in salivary flow creates a cycle of ascending sialadenitis, ductal ectasia, acinar atrophy, and obstructive fibrosis. Clinically, the patient complains of recurrent parotid pain and swelling, typically while eating. Sialography confirms sialectasia and areas of ductal dilatation. Initial treatment should be conservative, utilizing sialogogues (lemon ballws or chewing gum) and adequate oral hydration to stimulate salivary flow. Superficial parotidectomy is recommended if prolonged conservative management fails.

Sialolithiasis

Salivary gland stones are both a cause and a consequence of chronic sialadenitis. In addition, they may produce acute suppurative sialadenitis. The stones are composed of inorganic calcium and sodium phosphate salts that are deposited in the duct on an organic nidus of mucus or cellular debris. Eighty to ninety percent of salivary calculi occur in the submandibular gland and may lead to complete acute obstruction of the gland. The patient complains of painful swelling, especially with meals, and may report extrusion of gravel from the duct.

The diagnosis is confirmed by palpation of a stone or by demonstration of decreased salivary flow from the duct. Soft tissue films may reveal a radiodense stone, and sialography may disclose complete or partial filling defects in the ductal system with retention of dye on evacuation films. Treatment consists of intraoral removal of stones that are close to the duct

orifice by ductal dilatation and massage. Stones in the hilum of the submandibular gland necessitate excision of the gland; parotid sialoliths are managed in a similar fashion.

Salivary Gland Trauma

Injuries to the salivary glands may be intraoral or extraoral, blunt or lacerating. The parotid gland is most commonly injured along with associated structures, including the facial nerve, Stensen's duct, soft tissues, mandible, or zygoma. Laceration of the parotid duct may occur with a facial laceration posterior to the anterior edge of the masseter muscle and results in simultaneous injury to the buccal branch of the facial nerve. The severed duct is repaired over a small polyethylene catheter using fine interrupted sutures. The associated seventh nerve injury is repaired by anastomosing the cut ends with 10-0 monofilament suture. All lacerations in the parotid gland region must be examined for seventh nerve injury. Injury may occur either to the main trunk at the pes anserinus or to one of the branches within the parotid gland. Facial injuries anterior to a vertical line from the lateral canthus of the eye do not require exploration, and the nerve will regenerate spontaneously. If injury to the nerve is suspected, each of the 5 major branches is assessed by observing voluntary movements and responses to nerve excitability testing. Early repair is preferable, since the severed distal ends of the nerve will respond to electrical stimulation up to 72 hours after injury. Precise repair using the operating microscope is crucial. The greater auricular nerve may be used as a cable graft for avulsed segments of the facial nerve.

Pharynx

Pharyngitis

Acute pharyngitis is usually caused by viruses (especially the adenoviruses and rhinoviruses) or group A beta-hemolytic *Streptococcus.* Clinically, erythema, edema, and occasional membrane formation are present, and the patient complains of pain on swallowing. In bacterial pharyngitis, the white count and fever are higher and cervical adenopathy is more marked. *Pneumococci, H influenzae,* and coagulase-positive *staphylococci* are occasionally the primary pathogens. Pharyngeal cultures are obtained and penicillin started pending the results.

One must differentiate acute bacterial pharyngitis from diphtheric pharyngitis and infectious mononucleosis. *Corynebacterium diphtheriae* is a potentially lethal bacterium in a nonimmunized host. Characteristic features are odynophagia, fever, and the development of a gray pseudomembrane on the oropharynx associated with a fetid odor. The membrane bleeds easily when removed and may gradually involve the larynx, producing acute upper airway obstruction. Infectious mononucleosis may mimic diphtheria, exhibiting faucial arch edema, a pharyngeal pseudomembrane, and laryngeal edema. A positive heterophil agglutination (Monospot) test, absolute lymphocytosis, generalized lymphadenopathy, and hepatosplenomegaly are features that help differentiate this disease entity from diphtheria. Tonsilloadenoidal hyperplasia may be so marked as to obstruct the upper airway, necessitating intubation and tonsilloadenoidectomy.

Acute tonsillitis secondary to group A beta-hemolytic streptococcal infection commonly occurs in children under 10 years of age and may occasionally present in epidemic

form. Clinically, pyrexia, odynophagia, referred otalgia, and malaise predominate. The tonsils appear hyperaemic and edematous, with or without a purulent exudate filling the tonsillar crypts that coalesce to form a yellow-white pseudomembrane. A 7- to 10-day course of penicillin is adequate therapy. An alternative choice is erythromycin or clindamycin.

Chronic tonsillitis may follow acute or subacute episodes of tonsillitis, especially in older children, and is associated with recurrent odynophagia, cough, and findings of tonsillar enlargement, debris in tonsillar crypts, and cervical lymphadenitis. Acute attacks should be treated with antibiotics. Tonsillectomy is indicated for 5 or more documented episodes of acute tonsillitis per year.

Acute tonsillitis may in some instances extend beyond the tonsillar tissue into the space between the anterior and posterior tonsillar pillars or into the soft palate, producing a **peritonsillar abscess.** Physical examination reveals an edematous, bulging, anterior tonsillar pillar with medial displacement of the soft palate and uvula. Immediate therapy includes incision and drainage of the abscess through the anterior tonsillar pillar, intravenous penicillin, hydration, and antipyretics followed by either immediate tonsillectomy or an interval tonsillectomy in 6 weeks. The advantages of immediate tonsillectomy in carefully selected patients include complete abscess drainage without recurrence, rapid relief of symptoms, greater technical simplicity with less hemorrhage, decreased morbidity, and shorter hospitalization.

Pulmonary hypertension and congestive heart failure secondary to chronic hypoxia have been reported in young children with hyperplasia of Waldeyer's ring. More recently, chronic hypersomnolence and periodic apnea has been recognized in patients with upper airway obstruction secondary to tonsilloadenoidal hyperplasia. During sleep, these patients experience periods of cessation of nasal-oral air flow greater than 10 seconds, persistent chest wall movement, and subsequent hypoxia and hypercapnia. The obstructive sleep apnea syndrome is characterized by frequent arousals during sleep and more subtle clinical findings such as weight loss, behavioral disturbances, and enuresis. Tonsillectomy and adenoidectomy are indicated to relieve upper respiratory tract obstruction.

Isolated adenoidal hypertrophy secondary to physiologic enlargement of the adenoids or chronic viral nasopharyngitis may produce nasal airway obstruction. These children exhibit chronic rhinorrhea, mouth breathing, dental abnormalities, and snoring during sleep. Adenoidectomy is curative.

Benign Neoplasms of the Pharynx

The most common benign tumor of the nasopharynx is the juvenile angiofibroma, a highly vascular, nonencapsulated, invasive neoplasm with a propensity to occur in adolescent males. Onset of clinical signs and symptoms may be at any time for 7 to 21 years of age. Epistaxis occurs in 75% of cases in addition to nasal obstruction and rhinorrhea. Preoperative carotid angiography and CT scanning should be performed to evaluate the blood supply and anatomic extent of the tumor. The major blood supply is from the internal maxillary artery. Preoperative embolization of the tumor and estrogentherapy markedly decrease blood loss at surgical resection. For lesions confined to the nasopharynx, a transpalatal approach is satisfactory. Larger tumors with involvement of the nasal cavity, maxillary, ethmoid, or

sphenoid sinus require a lateral rhinotomy or Caldwell-Luc approach for complete excision. Although these neoplasms are only moderately responsive to radiation therapy, such therapy is often the treatment of choice for orbital or intracranial invasion.

Pharyngeal Foreign Bodies

Irregular foreign bodies entering the pharynx are likely to lodge in the lingual or palatine tonsils, valleculae, or piriform sinuses. Smooth round or ovoid objects commonly lodge at the opening to the esophagus or cricopharyngeus muscle, especially in children. Dysphagia, odynophagia, or aphagia may result. In a young child or infant, drooling is a characteristic sign. Dyspnea, wheezing, or persistent cough may develop secondary to compression of the larynx or trachea. If the esophagus is penetrated by a sharp object such as a pin or fish bone, subcutaneous emphysema can be palpated in the neck. Foreign bodies lodging more distally in the esophagus such as at the level of the aortic arch, left main bronchus, or gastroesophageal junction generally do not produce early symptoms.

Chest x-ray, anteroposterior and lateral neck films, and occasionally a barium swallow are necessary to delineate the site of a foreign body. Foreign bodies of the palatine and lingual tonsils are removed directly with a curved hemostat; objects located in the hypopharynx or esophagus require direct laryngoscopy or esophagoscopy under general anesthesia.

Larynx

Clinical Assessment

Abnormalities such as aspiration, weak cry, hoarseness, stridor, and poor cough point to laryngeal dysfunction and should prompt an inspection of the larynx with a laryngeal mirror (indirect laryngoscopy) during phonation and deep inspiration. Cinefluoroscopy with barium sulfate allows assessment of the competency of the larynx during swallowing. Neoplasms and traumatic lesions of the larynx are effectively evaluated by CT scan. In any individual with hoarseness of more than 2-3 weeks' duration, the larynx should be inspected with a mirror. If the larynx cannot be inspected in this way, trasnasal fiberoptic laryngoscopy or direct laryngoscopy under general anesthesia is required. A specimen of larynx is obtained at direct laryngoscopy for biopsy examination for suspected neoplasms.

Foreign Bodies of the Larynx & Tracheobronchial Tree

In children, a variety of objects, including seeds, beans, pins, and tiny toys, may be aspirated into the tracheobronchial tree. In adults, meat is the most common cause of obstruction and is associated with a number of factors: (1) large, poorly chewed pieces of food; (2) elevated blood alcohol; and (3) upper and lower dentures. The development of a "café coronary" is frequently confused with a myocardial infarction.

Foreign bodies entering the tracheobronchial tree must pass (1) the epiglottis, (2) the upper laryngeal inlet, (3) the false cords (ventricular bands), (4) the true vocal cords, and (5) the cough reflex. If a foreign body lodges in the larynx, there is immediate pain and

laryngospasm, dyspnea, and inspiratory stridor proportionate to the degree of upper airway obstruction. The voice may be hoarse or aphonic.

If partial airway obstruction is present and the victim can exchange air and cough, no attempt should be made to move the foreign body at that time. If the victim is aphonic, unable to cough or exchange air, and is clutching his or her neck, complete airway obstruction is present. If equipment is not at hand for emergency tracheostomy or cricothyrotomy, 2 manual maneuvers are recommended for relieving foreign body airway obstruction: (1) a series of 4 back blows are delivered with the heel of the hand over the spine betweenthe shoulder blades, and (2) a series of 4 manual thrusts are administered to the upper abdomen or lower chest. Finally, if the foreign body remains in the larynx or pharynx after these maneuvers, manual removal with the finger probe may be successful.

In the conscious adult patient with adequate air exchange, indirect laryngoscopy supplemented with anteroposterior and lateral x-rays of the neck and chest will confirm the position of the foreign body. Removal of a laryngeal foreign body necessitates general anesthesia and a laryngoscope and alligator forceps. Foreign bodies in the tracheobronchial tree also require general anesthesia and open bronchoscopy with forceps removal.

The reaction of the tracheobronchial tree to a foreign body depends upon the degree of obstruction and physical nature of the foreign body. For example, a bean acts as a ball valve, rising with expiration and occluding the distal airway on inspiration. Vegetable matter produces a violent bronchitis that may be associated with chronic suppurative pneumonitis; nonobstructive metallic objects may remain within the tracheobronchial tree for an extended period with little tissue damage.

Tracheal foreign bodies produce inspiratory and expiratory wheezing. With distally located objects, 3 different patterns may occur: (1) partial (bypass valve) bronchial obstruciton, in which the foreignbody permits the passage of air during both inspiration and expiration; (2) expiratory check valve obstruction, where ingress of air is minimally impeded but egress is checked, resulting in obstructive emphysema; and (3) stop valve obstruction, in which no air enters the subjacent lung, resulting in atelectasis. Currently, open (rigid) bronchoscopy is the standard way to remove foreign bodies of the tracheobronchial tree. However, flexible fiberoptic bronchoscopy is a better method of finding distal objects, facilitating earlier diagnosis of carcinoma of the lung and permitting transbronchial drainage of pulmonary abscesses.

Laryngeal Trauma

Trauma to the larynx and trachea may occur from iatrogenic causes (prolonged endotracheal intubation or inappropriate tracheostomy, laryngotomy or cricothyrotomy) or extrinsic injuries (automobile accidents, neck blows, strangulation, etc). The passenger in the front seat of an automobile is particularly vulnerable to hyperextension injury of the neck. This results in compression of the larynx, hyoid bone, and upper trachea between the dashboard and the cervical spine. Injuries of the larynx are less common in children because of the higher position of the larynx in the neck and the resulting protection provided by the mandible. Severe laryngotracheal injury may occur, however, in children riding bicycles, motor bikes, snowmobiles, etc, who strike a horizontal cable or fall against the handlebars.

The most common injury to the larynx is vertical fracture of the thyroid cartilage with or without fracture of the cricoid cartilage. Because the cricoid cartilage is the only complete cartilaginous ring in the respiratory tract, its functional integrity is critical in maintaining a patent airway. An unreduced fracture of the cricoid may result in subglottic stenosis. Associated injuries of the pharynx, trachea, esophagus, soft tissues, and neurovascular structures of the neck are common. Escape of air into the mediastinum may produce tension pneumothorax.

Clinical findings in laryngotracheal trauma include (1) subcutaneous emphysema or crepitus, (2) dysphonia, (3) loss of the laryngeal prominence (Adam's apple), (4) dysphagia, (5) odynophagia, (6) stridor, (7) hemorrhage, and (8) cough.

Conservative treatment with cool mist, intravenous fluids, penicillin, and parenteral corticosteroids will suffice for laryngeal soft tissue edema without significant airway obstruction or impaired vocal cord mobility. More severe laryngotracheal injury requires endotracheal intubation or tracheostomy. In an emergency, an endotracheal or tracheostomy tube may be introduced through an open laryngotracheal wound. Ideally, tracheostomy should be performed after the airway is controlled by intubation or open bronchoscopy. However, this may not be possible in cases of complete laryngotracheal separation. If a high tracheostomy or a cricothyrotomy has been performed, it should be revised as soon as possible to the third or fourth tracheal ring to prevent vocal cord paralysis and subglottic stenosis. Open reduction and stabilization of all cartilaginous, mucosal, and soft tissue defects with internal fixation and a soft stent is immediately done if the patient's general condition permits. They may be delayed for 3-5 days to permit easier identification of the laryngeal landmarks. Ultimately, the following factors affect wound healing within the larynx and trachea and determine the success or failure of therapy: (1) mechanical loss of lumen-supporting structures, (2) loss of blood supply to cartilaginous structures, (3) presence of chondritis, and (4) degree of progressive fibrosis and stenosis.

Pediatric Airway Obstruction

Airway obstruction at birth or in the first several months of life is commonly secondary to congenital and neoplastic disorders. At birth, immediate differentiation must be made between respiratory depression with cyanosis, shallow and slow respirations, and respiratory tract obstruction producing tachypnea, stridor, and suprasternal and subcostal retractions.

Stridor (noisy breathing) is the most prominent symptom and is an expression of partial respiratory tract obstruction secondary to external compression or partial occlusion within the airway. The character and intensity of stridor depend upon the site and degree of obstruction and the airflow velocity and pressure gradient across the point of obstruction. Obstruction at the level of the true vocal cords produces high-pitched inspiratory stridor. By contrast, stridor that occurs chiefly during expiration and is lower in pitch is commonly associated with tracheal obstruction. The quality of the cry remains normal in most infants with airway obstruction who do not have a laryngeal lesion. A weak or absent cry at birth suggests neurogenic vocal cord impairment. In addition to evaluating the cry and breathing patterns, swallowing function should be assessed in all infants with stridor. Mediastinal tumors and vascular rings producing extrinsic esophageal and tracheal compression cause

feeding difficulties and failure to thrive. The presence of recurrent pneumonitis and aspiration suggests a laryngeal lesion or tracheoesophageal fistula. All infants with stridor should have an anteroposterior and lateral chest fild and barium swallow followed by endoscopy.

Newborns & Small Infants

The most common cause of **infantile stridor** is laryngomalacia, or congenital flaccid larynx. During inspiration, there is extreme infolding of the omega-shaped epiglottis and aryepiglottic folds owing to inadequate cartilaginous support. The supine position or head flexion aggravates the stridor, whereas patency of the airway is improved by the prone position and head extension. The stridor gradually resolves in most infants within 2-3 monhts. Endoscopic inspection is necessary in infants with persistent or progressive stridor.

Congenital subglottic stenosis is the second most frequently encountered laryngeal lesion and may become evident several weeks or more after birth, following an upper respiratory tract infection. Because the subglottic region is the narrowest point in the upper respiratory tract, a small amount of edema will critically narrow this conduit. In those instances where stenosis is severe, a tracheostomy followed by either expectant waiting or dilatation may be necessary. Although controversy exists concerning the surgical correction of subglottic stenosis, a variety of techniques are available including autogenous auricular nasoseptal or costal cartilage grafts along with thyroid cartilage or pedicled hyoid bone interposition grafts.

Progressive laryngeal stridor and a crouplike illness in the first several months of life suggest a lesion simulating subglottic stenosis - the **subglottic hemangioma.** The neoplasm is a soft compressible bluish tumor below the level of the true vocal cords that is frequently poorly delineated from surrounding tissue. There is a 2:1 female to male preponderance, and 50% of the lesions are associated with cutaneous hemangiomas. The lateral neck film confirms the presence of a localized subglottic soft tissue mass.

Mechanical airway obstruction is treated with tracheostomy; however, early therapy with systemic corticosteroids may decrease the need for tracheostomy. Hemangiomas producing severe airway obstruction that do not respond to corticosteroids or regress spontaneously should be surgically removed. Because they are so vascular, these lesions are best controlled by the CO_2 or neodymium:YAG laser.

Larger Infants & Children

Supraglottitis is an acute inflammatory disorder of the larynx secondary to infection with *H influenzae* type B that affects the epiglottis, aryepiglottic folds, arytenoids, and ventricular bands (Table 41-2). There is usually no prodromal phase, and dysphagia, odynophagia, and shortness of breath rapidly progress to drooling, inspiratory stridor, and a muffled but clear voice. The disease affects principally children 2-6 years of age. Most children are extremely toxic, with fever, tachycardia, and tachypnea. The child sits erect, anxious and increasingly exhausted, drooling, and hungry for air. Lateral neck films confirm the diagnosis and reveal massive edema of the epiglottis.

Immediate control of the airway is mandatory and lifesaving. Children are given 100% humidifed oxygen and taken immediately to the operating room, where rapid halothane and oxygen anesthetic induction is followed by atraumatic peroral endotracheal intubation. Pharyngeal blood cultures are obtained, and an intravenous line is started. A course of antibiotics consisting of (1) ampicillin and chloramphenicol or (2) cefamandole is initiated pending culture and sensitivity reports. Direct laryngoscopy is perfored to rule out other potential causes of acute laryngeal obstruction. Direct inspection of the larynx reveals a cherry-red swollen epiglottis. At this time, the endotracheal tube is changed to a nasotracheal tube (1-2 sizes smaller than normal). Within 36-48 hours, the infant is generally afebrile and coughing around the tube during suctioning and may be successfully extubated.

Table 41-2. Laryngotracheobronchitis and supraglottitis

Laryngotracheobronchitis	**Supraglottitis**
Onset and history	
Relatively slow in onset as the terminal event of a 4- or 5-day respiratory tract infection	Rapid in onset and progression, advancing to severe airway obstruction within 6-8 hours. Usually no antecedent respiratory infection.
Etiology	
Usually viral but may be bacterial	Usually bacterial *(Haemophilus influenzae)* but may be viral.
Symptoms	
Stridor, barking cough, sometimes hoarseness	Stridor preceded by severe sore throat and dysphagia (drooling).
X-ray findings	
Narrowing of the subglottic airway ("steeple sign")	Enlarged epiglottis on soft tissue lateral to the pharynx and larynx (thumbprint sign).
Treatment	
Early: Moist oxygen, corticosteroids, antibiotics, and nebulised epinephrine Late: Endoscopic intubation with or without tracheostomy	Immediate moist oxygen and early establishment of an airway by endoscopic intubation. This is followed by administration of intravenous antibiotics.

By contrast, **acute laryngotracheobronchitis** is a viral illness that is far more common than acute supraglottitis (Table 41-2). This illness occurs chiefly in late autumn and winter, with parainfluenza and influenza A and B viruses accounting for most cases. The principal lesion is subglottic edema, with a variable component of tracheobronchial inflammation. Infants 3 months to 3 years of age are principally affected, exhibiting a 2:1 female to male ratio. The symptoms of barking cough, hoarseness, inspiratory and expiratory stridor, and substernal retraction are frequently preceded by an insidious upper respiratory tract illness lasting 1-7 days. In contrast to children with supraglottitis, the infant appears sick

but not toxic. Anteroposterior neck films confirm the clinical impression of marked subglottic narrowing and assing in excluding aerodigestive tract foreign bodies, mediastinal tumors, laryngotracheal neoplasms, and vascular compression of the trachea. Initial treatment includes high humidification, oxygenation, hydration, ampicillin, and parenteral corticosteroids. Some physicians recommend nebulized racemic epinephrine. Patients with progressive hypoxia, cyanosis, hypercapnia, and increasing tachypnea and tachycardia who do not respond to medical management should be intubated. Tracheostomy is performed after 3-4 days of intubation if significant subglottic edema persists, thus avoiding the high risk of subglottic stenosis associated with prolonged intubation with an inflamed larynx. Tracheostomy technique is discussed in Chapter 51. The only difference between adult and pediatric tracheostomy is that in children a vertical incision of the trachea is used and no cartilage is excised.

Laryngeal Papillomas

Laryngeal papillomas are thought to be caused by the human papovavirus. They may occur as early as 1 year of age but are more common in the second or third year. They present initially with hoarseness and may multiply rapidly to obstruct the airway. Papillomas recur promptly after surgical excision and may be implanted in the trachea or distal bronchi by mechanical trauma. Laser excision of papillomas with the operating microscope appears to be associated with less risk of laryngeal stenosis and perhaps a decrease in the rate of recurrence. Interferon is currently being studied as a treatment modality.

Laryngitis

Acute laryngitis often occurs in association with a general viral upper respiratory tract infection; however, bacterial (particularly streptococcal) infections may cause laryngitis. Hoarseness, cough, and odynophagia are often quite marked, with minimal edema or erythema of the true vocal cords. Treatment includes voice rest, humidification, and penicillin if streptococcal infection is suspected.

Chronic laryngitis, on the other hand, is related to many factors, including voice misuse, inhalation of irritants, and chronic allergies. Pathologically, fluid accumulates in the subepithelial space of the vocal cords (Reinke's space). In some individuals, large sessile **polyps** may develop and occupy the entire vocalcord or a portin thereof. The voice is severely compromised, having a hoarse and breathy character. In adults, polyps and chronic laryngitis are managed by voice rest, avoidance of chronic irritants, and speech therapy. Microdirect laryngoscopy and surgical excision with microforceps or the CO_2 laser is necessary for polyps not responding to conservative management.

Vocal Nodules

Misuse of the voice, particularly shouting or roaring in a very high or very low tone of voice, will result in condensation of hyaline connective tissue at the junction of the anterior and middle thirds of the true vocal cords. Vocal nodules occur in both children and adults and produce a hoarse and breathy voice. In children, most nodules regress with voice therapy, and

surgical removal is unnecessary. In both children and adults, however, voice therapy must be instituted for persistent nodules before they are endoscopically removed with the laser or microforceps.

Vocal Cord Paralysis

The recurrent laryngeal nerves of the vagus nerves are the primary innervators of the abductors and adductors of the vocal folds. Isolated injury of the recurrent laryngeal nerve results in paralysis of the vocal cord in the paramedian position on one side, 2-3 mm lateral to the laryngeal midline. Combined injury of the recurrent and superior laryngeal nerves paralyzes the vocal cord in the intermediate position, several millimeters lateral to the paramedian position.

Vocal cord paralysis may be unilateral or bilateral, central or peripheral. Unilateral left vocal cord paralysis is most common. Less than 20% of cases are bilateral. Thyroidectomy is by far the most common cause of bilateral vocal cord paralysis. Central causes include brain stem and supranuclear lesions and account for only 5% of all cases. Supranuclear or cortical causes of vocal cord paralysis are exceedingly rare, owing to the bilateral crossed neural innervation to the brain stem medullary centers in the nucleus ambiguus. The most frequent centralcause is vascular insufficiency or a stroke affecting the brain stem. Congenital central lesions are usually secondary to Arnold-Chiari malformation or brain stem dysgenesis and are often associated with additional cranial neuropathies.

Most cases of peripheral vocal cord paralysis are secondary to thyroidectomy or nonlaryngeal neoplasms, including bronchogenic, esophageal, and thyroic carcinoma. Other less common lesions causing paralysis of the vocal cord include tumors of the deep lobe of the parotid gland, carotid body tumors, glomus jugulare and vagale tumors, and neurogenic neoplasms of the tenth nerve and jugular foramen. External penetrating wounds to the neck or prolonged endotracheal intubation may also traumatize the recurrent laryngeal nerve, producing vocal cord paralysis. Finally, toxic neuropathy and idiopathic causes account for a few cases.

In adults, unilateral recurrent laryngeal nerve paralysis generally produces hoarseness and a weak, breathy voice with varying amounts of aspiration. The normal vocal cord may cross the midline to approximate the paralyzed vocal cord in the paramedian position. In children, varying degrees of inspiratory stridor may also be present. Bilateral vocal cord paralysis is commonly associated with inspiratory stridor, shortness of breath, and dyspnea on exertion.

Diagnostic assessment of vocal cord paralysis includes indirect laryngoscopy, examination of the head and neck for neoplasms, chest x-ray, base of skull films, thyroid scan, upper gastrointestinal series, and endoscopic evaluation of the aerodigestive tract.

Management of unilateral vocal cord paralysis due to lesions of the recurrent laryngeal nerve includes the injection of Teflon paste under local anesthesia into the paralyzed vocal cord, mobilizing it medially. Medialization is valuable in the therapy of aspiration and results in dramatic improvement in voice quality. In the past, bilateral paramedian vocal cord paralysis was commonly managed by permanent tracheostomy. More recently,

arytenoidectomy through an endolaryngeal or extralaryngeal approach has been used. This procedure may be complicated by loss of adequate voice production and exacerbation of aspiration. Currently, attempts to treat bilateral abductor vocal cord paralysis with nerve-muscle transposition of the ansa hypoglossi nerve and the omohyoid muscle to the posterior cricoarytenoid muscle have met with qualified success. This reinnervation technique attempts to provide inspiratory neuronal input tothe sole abductor of the vocal cord, the posterior cricoarytenoid muscle. In some patients, successful reinnervation has allowed decannulation of the tracheostomy tube.

Laryngeal Neoplasms

Most malignant epithelial neoplasms of the larynx are squamous cell carcinomas, ranging from well-differentiated to undifferentiated cell types. Alcohol and tobacco abuse are common predisposing factors. Cancer of the larynx affects chiefly men in a 9:1 ratio to women. Any patient who complains of persistent hoarseness, odynophagia, "a lump in the throat", or a change in voice quality should be examined promptly by indirect laryngoscopy. Precancerous laryngeal lesions appearing as leukoplakia or erythroplasia often evolve into carcinoma and should be biopsied to rule out carcinoma in situ or invasive carcinoma. Additional findings that arouse suspicion of laryngeal carcinoma include persistent localized edema or ulceration, irregular epithelium, and a paralyzed vocal cord.

Suspicious epithelial lesions of the true vocal cords are treated surgically by removing the entire vocal cord epithelium. Fortunately, true vocal cord carcinomas tend to be well-differentiated, to grow slowly, and to metastasize late, because of the limited lymphatic drainage of the cords. As a result, cervical metastases are infrequent, and fixation of the vocal cord is an unusual early clinical finding. Glottic cancers without vocal cord fixation are successfully treated by irradiation (5500-6500 rads in 5-7 weeks through a limited field). Total laryngectomy is necessary for vocal cord fixation and should include radical neck dissection followed by postoperative radiation therapy if cervical nodes are palpable.

Supraglottic carcinomas more often manifest local invasion and lymph node metastasis. As many as 50% of patients present with palpable metastases in jugular lymph nodes owing to the rich lymphatic drainage from this region. Unfortunately, the patients seek care late, because the tumor does not interfere with phonation and breathing until it is relatively large. Spread to the base of the tongue and hypopharynx is common. For tumors that stop 5 mm above the anterior commissure, that do not involve the true vocal cords, and that do not extend 5 mm above the vallecula, a horizontal supraglottic laryngectomy combined with pre- or postoperative radiation therapy may preserve the vocal cords and phonation. The objectives of this operation and other procedures that preserve the larynx are to provide an adequate surgical margin, prevent aspiration, and conserve speech and the airway. This procedure is contraindicated in old and debilitated patients or those with severe chronic obstructive pulmonary disease. Radiation therapy to the neck is given routinely after surgery to treat clinically inapparent lymph node metastases or as an adjunct to radical neck dissection for palpable metastases.

True subglottic carcinomas are uncommon (less than 5% of all laryngeal carcinomas). However, subglottic extension across the true vocal cord from a transglottic carcinoma is not unusual. When this occurs, lymphatic spread to the jugular chain, paratracheal, and

tracheoesophageal lymph nodes may be rapid. Total laryngectomy is necessary for these tumors and other far-advanced laryngeal carcinomas. Radiation is used as primary therapy for patients who reject laryngectomy. A method of speech rehabilitation is preoperatively discussed with each patient and may include esophageal speech, artificial larynx, or surgical restoration. Currently, encouraging results are being achieved with a small alloplastic button placed in a fistula between the posterior tracheal wall and the anterior esophageal wall. The patient wears a tracheostomy tube, which is occluded manually during speech, diverting air into the hypopharynx. The intact pharyngeal resonators and oral articulators assist in speech formation. Periodic follow-up examinations of the remainder of the aerodigestive tract are mandatory for the rest of the patient's life, because of the high incidence of second or even third primary carcinomas.

Inflammatory Neck Masses

Acute suppurative lymphadenitis usually occurs in infants and children with viral upper respiratory tract infections. Bacterial lymphadenitis commonly develops secondary to infection with streptococci, *S aureus,* or mouth anaerobes and may evolve into a deep neck abscess forming a lateral neck mass. High fever and leukocytosis characterize this complication. Deep neck abscesses may develop in the prevertebral, sublingual, submandibular, submental, or retropharyngeal spaces as well as in the lateral neck region. Abscesses of the neck are compartmentalized by 2 of the 3 envelopes of the deep cervical fascia: the superficial, middle, and deep layers. Infections may spread from one space to another or extend downward into the mediastinum. In addition, cellulitis or abscess formation in the retropharyngeal space or sublingual space (Ludwig's angina) can obstruct the airway. Infection around the carotid sheath may also produce serious hemorrhage by necrosis of the great vessels and their branches.

Patients with deep neck infections should be hospitalized immediately. Intravenous penicillin in high doses is the drug of choice, reserving clindamycin for recalcitrant cases. The airway should be controlled with an endotracheal tube or tracheostomy before the abscess is incised and drained. The surgical approach to a deep neck abscess depends on the space involved. Proximal control of the carotid artery should be obtained. The lateral pharyngeal space is approached through an incision parallel to the anterior border of the sternocleidomastoid muscle. Most retropharyngeal abscesses are drained intraorally. Submandibular abscesses are approached through an incision 2 cm below the inferior border of the mandible.

Chronic granulomatous infections, which may involve the cervical lymph nodes, include tuberculosis, atypical mycobacteria, and occasionally actinomycosis. Tuberculous adenitis commonly develops following pulmonary tuberculosis and usually responds to triple-drug chemotherapy. Persistently enlarged or suppurative nodes should be excised. Atypical mycobacterial adenitis, on the other hand, is seldom associated with pulmonary disease, and routine tuberculin skin tests are either negative or weakly positive. In contrast to tuberculous adenitis, these atypical infections do not respond well to chemotherapy alone and frequently must be excised.

Finally, actinomycosis may present as an abscess with multiple draining sinuses near the angle of the mandible, discharging pus with characteristic sulfur granules. Often there is

underlying dental disease or osteomyelitis of the adjacent bone. Long-term (3-4 weeks) intravenous penicillin in high doses is necessary, reserving surgical excision for persistent disease.