# The Merck Manual of Diagnosis and Therapy

## Section 7. Ear, Nose, And Throat Disorders

## Chapter 82. Approach To The Patient With Ear Problems

Hearing loss, tinnitus, vertigo, earache, and otorrhea are the principal symptoms of ear problems. When a patient has complaints referable to the ears, a thorough history should be taken, and a physical examination performed with emphasis on the ears, nose, nasopharynx, and paranasal sinuses. In addition, the teeth, tongue, tonsils, hypopharynx, larynx, salivary glands, and temporomandibular joint should be examined because pain and discomfort may be referred from them to the ears. X rays or CT of the temporal bones is often indicated for trauma to the ear, possible basal skull fracture, perforation of the tympanic membrane, hearing loss, vertigo, facial paralysis, and otalgia of obscure origin. Measurements of auditory and vestibular functions are very important in the diagnosis of complaints referable to the ears.

### Hearing Loss

Hearing loss caused by a lesion in the ear canal (external auditory canal) or middle ear is **conductive;** hearing loss caused by a lesion in the inner ear or 8th nerve is **sensorineural.** Conductive and sensorineural hearing loss can be differentiated by comparing the threshold of hearing by air conduction with that by bone conduction.

Sensorineural hearing loss can be further differentiated as sensory (cochlea) or neural (8th nerve). This distinction is important because neural hearing loss is often caused by potentially curable tumors (see Differentiation of Sensory and Neural Hearing Losses, below).

### Clinical Measurement of Hearing

The minimum comprehensive audiologic assessment includes measurement of pure-tone air and bone conduction thresholds, speech reception threshold, and speech discrimination; tympanometry; and acoustic reflex testing, including reflex decay testing. Information gained from these procedures helps determine whether more definitive differentiation of sensory from neural hearing loss is indicated.

**Hearing by air conduction** is tested by presenting an acoustic stimulus through earphones or a loudspeaker to the ear. A hearing loss or an elevation of the hearing threshold detected this way can be caused by a defect in any part of the hearing apparatus--ear canal, middle ear, inner ear, 8th nerve, or central auditory pathways.

**Hearing by bone conduction** is tested by placing a sound source (eg, the oscillator of an audiometer or the stem of a tuning fork) in contact with the head. The sound causes vibration throughout the skull, including the walls of the bony cochlea, and stimulates the inner ear directly. Hearing by bone conduction bypasses the external and middle ear and tests the integrity of the inner ear, 8th nerve, and central auditory pathways.

If the air conduction threshold is elevated and the bone conduction threshold is normal, the hearing loss is conductive. If air and bone conduction thresholds are elevated equally, the

hearing loss is sensorineural. Occasionally, hearing loss is **composite** or **mixed,** with conductive and sensorineural components. In such cases, both bone and air conduction thresholds are elevated, with the air conduction threshold more elevated than the bone.

Weber's test for hearing and Rinne's test are used to differentiate conductive from sensorineural hearing loss. For these tests, tuning forks with frequencies of 256, 512, 1024, and 2048 Hz are used. In Weber's test, the stem of a vibrating tuning fork is placed on the midline of the head, and the patient indicates in which ear the tone is heard louder. A patient with a unilateral conductive hearing loss hears the tone louder in the ear with the conductive hearing loss, for reasons that are unclear. In contrast, a patient with a unilateral sensorineural hearing loss hears the tone louder in the normal ear because the tuning fork stimulates both inner ears equally and the patient perceives the stimulus with the more sensitive, unaffected end organ and nerve.

Rinne's test compares hearing by air conduction with that by bone conduction. The stem of a vibrating tuning fork is placed in contact with the mastoid process (for bone conduction); then the tines of the still vibrating fork are held near the pinna (for air conduction), and the patient is asked to indicate which stimulus is louder. Normally, the stimulus is heard louder by air conduction (AC) than by bone conduction (BC), so the relationship is AC > BC. With a conductive hearing loss, the relationship is reversed; the bone conduction stimulus is perceived louder than the air conduction stimulus (BC > AC). With a sensorineural hearing loss, both air and bone conduction perceptions are reduced, but the ratio remains the same as that for normal hearing (AC > BC).

In audiometry, hearing loss is quantitated. An audiometer delivers acoustic stimuli of specific frequencies (pure tones) at specific intensities so that the patient's hearing threshold for each frequency can be determined. Hearing in each ear is measured from 125 or 250 to 8000 Hz by air conduction (using earphones) and by bone conduction (using an oscillator in contact with the mastoid process or forehead). Hearing loss is measured in decibels (dB). A decibel is a logarithmic unit that is a ratio between a reference level and a measured level. Because various references are used, the reference must be stated when referring to decibels. The amount of sound energy is increased 10 times for each 20 dB. Test results are plotted on graphs called audiograms (see Figs. 82-1 and 82-2). The audiogram is a logarithmic representation of the sound energy needed to achieve threshold. When hearing differs between ears or between air and bone conduction, intense tones presented to one ear may be heard in the other ear. In such cases, a masking sound, usually noise, is presented to the ear not being tested, so that responses obtained more accurately reflect hearing in the ear being tested.

Speech audiometry includes the speech reception threshold (SRT) and speech discrimination. The SRT, the intensity at which speech is recognized as a meaningful symbol, is determined by presenting a list of words, usually words with two equally accented syllables (spondees), such as railroad, staircase, baseball, at specific intensities and noting the intensity at which the patient repeats 50% of the words correctly. The SRT should approximate the average hearing level at speech frequencies of 500, 1000, and 2000 Hz.

Speech discrimination, the ability to discriminate among the various speech sounds or phonemes, is determined by presenting 50 phonetically balanced one-syllable words, containing the phonemes in the same relative frequency as in conversational English, at an

intensity of 25 to 40 dB above the SRT. The percentage of words correctly repeated by the patient is the speech discrimination score, normally 90 to 100%, and is a good indication of a person's ability to understand speech under ideal listening conditions. This score remains in the normal range with conductive hearing loss but is reduced with sensorineural hearing loss because analysis of speech sounds by the inner ear and 8th nerve is impaired. Discrimination tends to be poorer in neural than in sensory hearing loss (see below).

Tympanometry measures the impedance of the middle ear to acoustic energy. While the patient remains quiet, a probe containing a sound source and microphone is placed in the ear canal to measure how much acoustic energy is absorbed (passes through) or is reflected by the middle ear. Normally, maximal compliance of the middle ear occurs when the pressure in the ear canal equals atmospheric pressure. Increasing or decreasing pressure in the ear canal demonstrates various patterns of compliance. When pressure in the middle ear is relatively negative, as in eustachian tube obstruction and middle ear effusion, maximal compliance occurs with a negative pressure in the ear canal and very little movement of the tympanic membrane. When the ossicular chain is disrupted, as in necrosis or dislocation of the long process of the incus, the middle ear is excessively compliant. When the ossicular chain is fixated, as in stapedial ankylosis in otosclerosis, compliance may be normal or reduced. Tympanometry is used to screen children for middle ear effusions (serous or secretory otitis media) and to provide diagnostic clues about conductive hearing loss.

Acoustic reflex testing can detect changes in compliance produced by reflex contraction of the stapedius muscle; the acoustic reflex is initiated by presenting a tone of varying intensities to the ear being evaluated or to the opposite ear. The presence or absence of this reflex is important in the topographic diagnosis of middle ear functioning and of facial nerve paralysis. The reflex adapts or decays in neural hearing loss, and determining whether it adapts or decays, especially below 2000 Hz, aids in differential diagnosis of sensory and neural hearing loss. This test can confirm voluntary threshold responses and may indicate that a patient is malingering.

The patient who cannot or will not respond voluntarily to acoustic stimuli may be evaluated by measuring the following: the cochlear microphonic response and action potentials of the 8th nerve with electrocochleography; evoked responses from the brain stem and auditory cortex (auditory brain stem response) to acoustic stimuli; and spontaneous or evoked otoacoustic emissions, sounds produced by the outer hair cells of the cochlea in response to sound stimulation in the ear (see below). These techniques are useful in evaluating infants and children suspected of having profound hearing loss (see also Measurement of Hearing in Children in Ch. 256), persons suspected of feigning or exaggerating a hearing loss (psychogenic hypoacusis), and patients with sensorineural hearing loss of unknown etiology; another use is evaluation of the neurologic auditory system. Seven sequential waveforms that occur in the 8th nerve and central auditory pathways in response to acoustic stimuli have been identified. Lesions of the 8th nerve and brain stem auditory pathways change the configuration and latency of the waveforms; changes in latency often have diagnostic value. Auditory brain stem responses are used in coma to determine the functional integrity of the brain stem and in intracranial operations to monitor the integrity of the 8th nerve and central auditory pathways. Auditory brain stem response testing cannot be performed on patients with severe hearing loss.

## Differentiation of Sensory and Neural Hearing Losses

The term sensorineural indicates uncertainty as to whether the hearing loss is due to a lesion in the inner ear (cochlea) or in the 8th nerve. The differentiation between sensory (cochlear) and neural (8th nerve) hearing loss is clinically important. Sensory hearing loss results from end-organ lesions (acoustic trauma, viral labyrinthitis, ototoxic drugs, Meniere's disease), which usually are not life threatening. Conversely, neural hearing loss is frequently due to potentially fatal cerebellopontine angle tumors (see Ch. 177) and a wide variety of other neurologic disorders.

Sensory and neural hearing losses may be differentiated on the basis of tests for speech discrimination, performance-intensity function for phonetically balanced words, recruitment, acoustic reflex decay, pathologic adaptation, otoacoustic emissions, cochlear potentials, and auditory brain stem responses (see Table 82-1).

In speech discrimination tests for phonetically balanced words (see above), the decrement is moderate when hearing loss is sensory and severe when it is neural.

Performance-intensity function for phonetically balanced words is tested at increments of 5 or 10 dB--starting from 20 to 30 dB above the speech reception threshold. With sensory hearing loss, discrimination usually improves at higher intensities. With neural hearing loss, discrimination characteristically deteriorates at higher intensities. The plotted word recognition score as a function of intensity is called articulation function. "Rollover," which refers to a decrement in discrimination as intensity increases, is characteristic of 8th nerve lesions, such as vestibular schwannoma.

Recruitment (an abnormal increase in the perception of loudness or the ability to hear loud sounds normally despite a hearing loss) can be demonstrated by having the patient compare the loudness of sounds in the affected ear with the loudness of sounds in the normal ear. In sensory hearing loss, the sensation of loudness in the affected ear increases more with each increment in intensity than it does in the normal ear. In neural hearing loss, the sensation of loudness in the affected ear increases no more (no recruitment) or increases less (decruitment) with each increment in intensity than it does in the normal ear.

The acoustic reflex (see above) adapts or decays over time with continuous presentation of a tone (particularly below 2000 Hz). Decay is absent or mild in sensory hearing loss and severe in neural hearing loss.

Pathologic adaptation (tone decay) is demonstrated when a patient cannot continue to perceive a constant tone above the hearing threshold. Tone decay is absent or mild with sensory lesions and severe with neural lesions.

Electrocochleography measures stimulus-related electrophysiologic potentials from the most peripheral portion of the auditory system; they include the cochlear microphonic, summating potential, and action potential. Surface electrodes, such as those used in auditory brain stem response, cannot record these potentials; electrodes must be placed on or through the tympanic membrane. Electrocochleography may be valuable in the assessment and

monitoring of patients with dizziness, in intraoperative monitoring, and in the enhancement of waveform I for patients with profound hearing loss.

The cochlear microphonic, probably generated by outer hair cells in the basal turn of the cochlea, is a response to alternating current that mirrors the waveform of low to moderately intense sounds. It is thought to reflect the time displacement pattern of the cochlear partition. It is recorded via a needle electrode placed through or an electrode placed on the tympanic membrane. Magnitude of the cochlear microphonic depends on the activity of the hair cells. Its usefulness has been questioned because of the difficulty in interpreting responses.

Auditory brain stem response is a powerful technique for differentiating sensory from neural hearing loss. Five distinct electric waveforms generated in the 8th nerve, brain stem, and other regions in response to acoustic stimulation are examined. They can be recorded using a computer to average responses to many stimuli. Each waveform probably emanates from a distinct structure in the auditory pathway, such as the 8th nerve, cochlear nuclei, superior olivary complex, lateral lemniscus, and inferior colliculus. With lesions of the 8th nerve, one or more waveforms may be lost, the latency of the waveforms from the onset of the acoustic stimuli may be increased, and the interwave latencies may be prolonged. With cochlear lesions, the waveforms are easily recognized, and the latency relationships remain normal.

Otoacoustic emissions are sounds generated by the outer hair cells in a healthy cochlea. They can be measured by placing in the ear canal a sound source to present a stimulus and a microphone to record the response. Information obtained is frequency specific and can be used in combination with other tests to characterize the type of hearing loss. Absence of otoacoustic emissions indicates damage in the cochlea. If otoacoustic emissions are present, the cochlea is intact. If the loss is sensorineural and otoacoustic emissions are present, the damage is in the 8th nerve. Middle ear diseases, such as otitis media, eliminate otoacoustic emissions. Most commonly, otoacoustic emissions are measured to screen hearing in infants and young children.

Patients with complaints referable to one cranial nerve, such as the 8th nerve, require a thorough neurologic evaluation. Further evaluation should include vestibular testing (see below) and MRI of the head with gadolinium enhancement to detect lesions of the 7th or 8th cranial nerve.

## Tests of Central Auditory Imperception

Lesions of the central auditory pathways may occur in the cochlear nuclei, brain stem pathways crossing the midline, superior olivary complex, lateral lemniscus, inferior colliculus, medial geniculate body, auditory radiation, or auditory cortex. Characteristically, such lesions do not result in elevation of pure-tone and spondee thresholds or in decreased discrimination of single words. Special tests are required to assess the deficit in auditory function caused by these lesions. These tests measure discrimination of degraded or distorted speech and discrimination in the presence of a competing message in the other ear, and they evaluate the ability to fuse incomplete or partial messages delivered to each ear into a meaningful message

and to localize sound in space (median plane localization) when acoustic stimuli are delivered simultaneously to both ears.

Speech may be degraded or distorted with low-frequency or high-frequency filters, periodic interruptions, or time compression. In the ear contralateral to a cortical lesion, discrimination of degraded or distorted connected speech is lost, as is discrimination in the presence of a competing message in the ipsilateral ear. Brain stem lesions cause a loss of ability to fuse incomplete messages presented to each ear into a meaningful message and impair the ability to localize sound accurately in space.

## Hearing Aids

Amplification of sound with hearing aids helps almost all persons with mild to profound conductive or sensorineural hearing losses, including those with predominantly high-frequency sensorineural hearing loss and those with unilateral hearing loss. All hearing aids have a microphone, amplifier, speaker, earpiece, and volume control. With many models, the microphone can be switched off and a magnetic coil used to enhance clarity when talking on a telephone. The best models are adjusted to the particular pattern of hearing loss, selectively amplifying the most poorly heard frequencies.

When a patient is evaluated for a hearing aid, professional advice, usually from an audiologist, is required. Selecting the proper hearing aid requires matching the electroacoustic characteristics of the aid with the type of hearing loss on the basis of gain (amplification), saturation level, frequency response, and listening requirement. Gain refers to the difference between the input and the output of the hearing aid. The more severe the hearing loss, the more gain is generally required. Saturation level, the maximum output of the hearing aid regardless of input, is an important consideration for patients with reduced tolerance to sound (as in recruitment). For severe tolerance problems, special circuitry (automatic gain control) is available to keep the output of the aid at a tolerable level. Frequency response refers to the gain of the aid as a function of frequency. As a general rule, the frequency response should be selected to provide gain consistent with the patient's audiometric configuration. High-frequency accentuation can also be achieved by venting the ear mold, which benefits many persons with a sensorineural hearing loss greater in high frequencies than in low frequencies.

Air conduction hearing aids, usually coupled to the ear canal with an airtight seal or open tube, are generally superior to bone conduction hearing aids and are usually used except when contraindicated. The body aid type, appropriate for profound hearing loss, is the most powerful. It is worn in a shirt pocket or a body harness and connected by a wire to the earpiece (the receiver), which is coupled to the ear canal by a plastic insert (ear mold). For moderate to severe hearing loss, a postauricular (ear-level) aid, which fits behind the pinna and is coupled to the ear mold with flexible tubing, is appropriate. An in-the-ear aid is contained entirely within the ear mold and fits less conspicuously into the concha and ear canal; it is appropriate for mild to moderate hearing loss. Canal aids are contained entirely within the ear canal and are cosmetically acceptable to many persons who would otherwise refuse to use a hearing aid, but they are difficult for some persons (especially the elderly) to manipulate. The CROS aid (Contralateral Routing Of Signals) is used by persons with severe unilateral hearing loss; a hearing-aid microphone is placed in the nonfunctioning ear, and sound is routed to the functioning ear through a wire or miniature radio transmitter. This

device enables the wearer to hear sounds from the nonfunctioning side and to develop a limited ability to localize sound. If the better ear also has some hearing loss, the sound from both sides can be amplified with the BICROS aid.

A bone conduction aid may be used when an ear mold or tube cannot be used, as in atresia of the ear canal or persistent otorrhea. An oscillator is placed in contact with the head, usually over the mastoid, with a spring band over the head, and sound is conducted through the bone of the skull to the cochlea. Bone conduction hearing aids require more power, introduce more distortion, and are less comfortable to wear than air conduction hearing aids. Some bone conduction aids can be implanted in the mastoid process, avoiding the discomfort and prominence of the spring band.

## Cochlear Implants

Profoundly deaf persons who cannot be helped by hearing aids to read speech (read lips) or to hear environmental sounds (eg, doorbells, ringing telephones, alarms) may benefit from a cochlear implant. This electronic device consists of a battery-powered processor that converts sound into modulations of an electric current, an internal and external induction coil system that transmits the electrical impulses through the skin, and an array of electrodes connected to the internal induction coil, which stimulates the remaining fibers of the auditory division of the 8th cranial nerve. During mastoid surgery, the electrode array is inserted into the scala tympani of the basal turn in the inner ear. The internal induction coil is implanted into the bone of the skull posterior and superior to the ear; the external conduction coil is held in place on the skin over the induction coil by magnets in the two coils. Multichannel implants are usually more effective than single-channel ones.

Cochlear implants help with speech reading by providing information about the intonation of words, the rhythm of speech, and some speech percepts. Some persons who have a cochlear implant can discriminate words without visual clues and can talk on the telephone. Cochlear implants enable deaf persons to hear and distinguish environmental sounds and warning signals. They also help deaf persons modulate their voices to make their speech more intelligible.

## Tinnitus

Perception of sound in the absence of an acoustic stimulus.

Tinnitus, a subjective experience of the patient, is distinguished from bruit, noise that may be heard by the examiner and often also by the patient.

Tinnitus may be a buzzing, ringing, roaring, whistling, or hissing, or it may involve more complex sounds that vary over time. It may be intermittent, continuous, or pulsatile (synchronous with the heartbeat). An associated hearing loss is usually present.

The mechanism causing tinnitus is obscure. Tinnitus may occur as a symptom of nearly all ear disorders, including obstruction of the ear canal by cerumen or a foreign body, infections (external otitis, myringitis, otitis media, labyrinthitis, petrositis, syphilis, meningitis), eustachian tube obstruction, otosclerosis, middle ear neoplasms (eg, glomus

tympanicum and glomus jugulare tumors), Meniere's disease, arachnoiditis, cerebellopontine angle tumors, ototoxicity (eg, due to salicylates, quinine and its synthetic analogs, aminoglycoside antibiotics, certain diuretics, carbon monoxide, heavy metals, alcohol), cardiovascular diseases (eg, hypertension, arteriosclerosis, aneurysms), anemia, hypothyroidism, hereditary sensorineural or noise-induced hearing loss, acoustic trauma (blast injury), and head trauma.

Evaluation of a patient with tinnitus requires the minimum comprehensive audiologic assessment (see above) as well as CT of the temporal bone and MRI of the head. If a sensorineural hearing loss is found, tests to differentiate sensory and neural hearing losses (see above) are indicated. Pulsatile tinnitus requires investigation of the vascular system with carotid and vertebral arteriograms to exclude arterial obstruction, aneurysms, and vascular neoplasms.

## Treatment

The ability to tolerate tinnitus varies among patients. Treatment should be directed toward the underlying disease, because its amelioration may lessen the tinnitus. Correcting the associated hearing loss usually relieves the tinnitus; a hearing aid often suppresses the tinnitus. Although there is no specific medical or surgical therapy for tinnitus, many patients find relief by playing background music to mask the tinnitus and may go to sleep with the radio playing. Some patients benefit from using a tinnitus masker, a device worn like a hearing aid that presents a sound more pleasant than the tinnitus. Electrical stimulation of the inner ear, as with a cochlear implant, occasionally reduces the tinnitus but is appropriate only for the profoundly deaf.

## Vertigo

An abnormal sensation of rotary movement associated with difficulty in balance, gait, and navigation of the environment.

The sensation may be subjective: the patient feels as if he is moving in relation to his environment; or it may be objective: he feels as if the environment is moving in relation to him. Vertigo results from lesions or disturbances in the inner ear, 8th nerve, or vestibular nuclei and their pathways in the brain stem and cerebellum.

## Clinical Evaluation of the Vestibular Apparatus

Vestibular function should be evaluated if a patient has vertigo, difficulty in balance, or a sensorineural hearing loss of unknown etiology. Evaluation centers on thorough history taking and specific tests that include rapidly alternating movement, finger-to-nose, and heel-to-shin tests; Romberg's sign; gait testing; and electronystagmography with caloric testing (see Fig. 82-3). With caloric tests, results for the two ears can be compared, so these tests are more useful clinically than are rotational, torsion swing, and lateral swing tests using stimulation with acceleration or deceleration.

Artificial stimulation of the vestibular apparatus produces nystagmus, past-pointing, falling, and autonomic responses, such as sweating, vomiting, hypotension, and bradycardia.

Nystagmus, the most useful response, can be monitored by observation or, more reliably, by electronystagmography, which records changes in the corneoretinal potential. Vestibular nystagmus is a rhythmic movement of the eyes. It has a quick and a slow component and may be rotary, vertical, or horizontal. The direction of the nystagmus is defined by the direction of the quick component because it is easier to see. However, the slow component is the more fundamental response to vestibular stimulation; the quick component is compensatory. The slow component moves in the direction of the movement of the endolymph; past-pointing and falling are also in this direction. The hallucination of movement of the environment is in the direction of endolymphatic flow, and the hallucination of movement of the subject is in the opposite direction.

Electronystagmography electronically detects spontaneous, gaze, or positional nystagmus that might not be visually detectable. Typically, it records responses to a battery of stimuli. Eye tracking of a moving target and the response to optokinetic stimulation with a rotating striped drum are conveniently recorded electronically at the time of caloric testing. Different components of the vestibular system can be tested by varying head and body position or visual stimuli.

Caloric stimulation is produced by irrigating the ears with warm and cool water, which causes convection currents in the endolymph. The currents cause movement of the cupula in the ampulla of the horizontal semicircular canal; the movement is in one direction during cooling and in the opposite direction during warming.

The bithermal caloric test, an accurate and reproducible measure of vestibular sensitivity, is performed with the patient supine and the head elevated 30° so that the horizontal semicircular canal is vertical. Each ear is irrigated with 240 mL of water delivered in 40 sec, first at 30° C (86° F), then at 44° C (111° F). The resulting nystagmus is monitored with the patient gazing straight ahead. Irrigating the ear with cool water produces nystagmus to the opposite side; warm water produces nystagmus to the same side. A mnemonic device is COWS (Cold to the Opposite and Warm to the Same).

The duration and frequency of nystagmus and the velocity of the slow component may be measured. Canal paresis, a unilateral reduction or absence of sensitivity, and directional preponderance, a relative exaggeration of the nystagmic response in one direction, can be detected. Various combinations of canal paresis and directional preponderance may coexist. The presence of canal paresis, directional preponderance, or combinations of the two indicates an organic lesion--end organ, 8th nerve, brain stem, or cerebellar--but does not necessarily indicate on which side the lesion is. Occasionally, an important differential point relies on caloric testing. Acoustic neuromas frequently cause canal paresis or no response on the side with the neoplasm.

Patients with vertigo should also have a minimum comprehensive audiologic assessment and MRI of the head with gadolinium enhancement to look for a lesion of the 8th cranial nerve.

## Earache

Earache (otalgia) occurs with infections and neoplasms in the external or middle ear (see Table 82-2) or is referred to the ear from remote disease processes (see Table 82-3). Even mild inflammation in the ear canal produces severe pain; perichondritis of the pinna produces severe pain and tenderness. Eustachian tube obstruction causes abrupt changes in middle ear pressure relative to atmospheric pressure, which may result in painful retraction of the tympanic membrane. Infection in the middle ear produces painful inflammation of its mucous membrane and pain from increased pressure in the middle ear, with bulging of the tympanic membrane. The most common cause of earache in children, acute otitis media, requires prompt examination by a physician and antibiotic therapy to prevent serious sequelae.

If there is no disease in the ear, the source of referred pain should be sought in areas receiving sensory supply from the cranial nerves that subserve sensation in the external and middle ear--ie, the 5th (trigeminal), 9th (glossopharyngeal), and 10th (vagus) nerves. Specifically, the cause of obscure otalgia should be sought in the nose, paranasal sinuses, nasopharynx, teeth, gingiva, temporomandibular joint, mandible, parotid glands, tongue, palatine tonsils, pharynx, larynx, trachea, and esophagus. Occult neoplasms (most often, carcinoma of the nasopharynx) in these locations are sometimes first manifested by pain referred to the ear. Otalgia commonly occurs after a tonsillectomy.

Treatment involves identifying the cause of the pain and providing appropriate therapy (see Chs. 83 and 84).

# Chapter 83. External Ear

Examination of the external ear focuses on the pinna and the ear canal (external auditory canal).

## Obstructions

Cerumen (earwax) may obstruct the ear canal and cause itching, pain, and a temporary conductive hearing loss. It may be removed by irrigation, but rolling the cerumen out of the ear canal with a blunt curet or loop or removing it with a vacuum through a tube is quicker, less messy, generally safer, and more comfortable for the patient. Irrigation is contraindicated if the patient has a history of otorrhea or perforation of the tympanic membrane. Water entering the middle ear through a perforation may exacerbate chronic otitis media. Cerumen solvents are not recommended because they often do not dissolve the mass and frequently cause maceration of the canal skin and allergic reactions.

Children insert all types of objects, particularly beads, erasers, and beans, into their ear canals. A foreign body in the ear canal is best removed by raking it out with a blunt hook. Forceps tend to push smooth objects deeper into the canal. A foreign body lying medial to the isthmus is difficult to remove without injuring the tympanic membrane and ossicular chain. Metal and glass beads can sometimes be removed by irrigation, but a hygroscopic foreign body (eg, a bean) swells when water is added to it, complicating its removal. A general anesthetic should be used when a child is uncooperative or when a mechanical problem could make removal difficult, possibly injuring the tympanic membrane or ossicles.

Insects in the canal are most annoying while alive. Filling the canal with mineral oil kills the insect, giving some immediate relief, and facilitates its removal with forceps.

## External Otitis

Infection of the ear canal.

External otitis may be localized (furuncle) or diffuse, affecting the entire canal (generalized or diffuse external otitis). It is more common during the summer swimming season and is often called swimmer's ear.

### Etiology

Diffuse external otitis may be caused by a gram-negative rod, such as Escherichia coli, Pseudomonas aeruginosa, or Proteus vulgaris; by Staphylococcus aureus; or, rarely, by a fungus. Furuncles are usually due to S. aureus. Certain persons (eg, those with allergies, psoriasis, eczema, or seborrheic dermatitis) are particularly prone to external otitis. Predisposing factors include getting water or various irritants (eg, hair spray, hair dye) in the ear canal and injuring the canal while cleaning it. The ear canal cleanses itself by moving desquamated epithelium, as on a conveyor belt, from the tympanic membrane outward. The patient's attempts to clean the canal with cotton applicators interrupt the self-cleansing mechanism and promote accumulation of debris by pushing it in the direction opposite the movement of the desquamated epithelium. Debris and cerumen tend to trap water allowed into the canal; the resulting skin maceration sets the stage for invasion of pathogenic bacteria.

### Symptoms and Signs

Patients with diffuse external otitis complain of itching, pain, a foul-smelling discharge, and hearing loss if the canal becomes swollen or filled with purulent debris. Tenderness on traction of the pinna and on pressure over the tragus tends to distinguish external otitis from otitis media. The skin of the ear canal is red, swollen, and littered with moist, purulent debris.

Furuncles cause severe pain and, when draining, brief sanguineous purulent otorrhea.

### Treatment

Swimmer's ear can often be prevented by irrigating the ears with a 1:1 mixture of rubbing alcohol and vinegar immediately after swimming. The alcohol helps remove water, and the vinegar alters the pH of the canal. Systemic antibiotics are seldom necessary unless there is a spreading cellulitis or other evidence of infection spreading outside the canal skin.

In diffuse external otitis, topical antibiotics and corticosteroids are effective. First, the infected debris is gently and thoroughly removed from the canal with suction or dry cotton wipes. A solution or suspension containing neomycin sulfate 0.5% and polymyxin B sulfate 10,000 U/mL is effective against the usual gram-negative rods. The addition of a topical corticosteroid, such as 1% hydrocortisone, reduces the swelling and allows the antibiotic to penetrate deep in the canal; 5 drops are instilled tid for 7 days. External otitis also responds

to alteration of the canal's pH with topical 2% acetic acid 5 drops tid for 7 days; the addition of 1% hydrocortisone reduces swelling and enhances the effectiveness of the acetic acid. An analgesic, such as codeine 30 mg po q 4 h, is usually necessary for the first 24 to 48 h. If cellulitis is present and extends beyond the ear canal, penicillin V 500 mg po q 6 h for 7 days is indicated. If the patient is allergic to penicillin, erythromycin in the same dose can be used.

Furuncles should be allowed to drain spontaneously, because incision may lead to spreading perichondritis of the pinna. Oral antistaphylococcal antibiotics are used. Topical antibiotics are ineffective. Analgesics, such as codeine 30 mg po q 4 h, are necessary to relieve the pain. Dry heat also helps relieve pain and hastens resolution.

## Perichondritis

Infection of the perichondrium of the pinna.

Trauma, insect bites, and incision of superficial infections of the pinna may initiate perichondritis, in which pus accumulates between the cartilage and the perichondrium. The blood supply to the cartilage is provided by the perichondrium, so that if the perichondrium is separated from both sides of the cartilage, the resulting avascular necrosis leads to a deformed pinna. Septic necrosis also plays a role. Perichondritis tends to be indolent, long-lasting, and destructive. It is usually caused by a gram-negative rod.

## Treatment

Wide incision and suction drainage are used to approximate the blood supply to the cartilage. Systemic antibiotic therapy is indicated and should be guided by culture and sensitivity studies; often, IV therapy with an aminoglycoside antibiotic and a semisynthetic penicillin is required.

## Aural Eczematoid Dermatitis

Inflammation of the skin of the pinna or ear canal.

Eczema--characterized by itching, redness, discharge, desquamation, and even fissuring leading to secondary infection--frequently affects the pinna and ear canal. Recurrences are common.

## Treatment

Dilute aluminum acetate solution (Burow's solution) is applied as often as required. Itching and inflammation can be reduced with topical corticosteroids. Topical antibiotic therapy as described above for diffuse external otitis may be needed occasionally. An underlying middle ear infection, if present, must be controlled with cleaning and use of appropriate topical and systemic antibiotics.

## Malignant External Otitis

Pseudomonas osteomyelitis of the temporal bone.

Malignant external otitis occurs mainly in elderly diabetics, beginning as external otitis caused by Pseudomonas aeruginosa, but also occurs in AIDS patients. It is characterized by persistent and severe earache, foul-smelling purulent otorrhea, and granulation tissue in the ear canal. Varying degrees of conductive hearing loss may occur. Frequently, facial nerve paralysis occurs in severe cases. CT scan of the temporal bone may show increased radiodensity in the air-cell system and middle ear radiolucency (demineralization) in some areas. Biopsy of the ear canal is necessary to differentiate the granulation tissue typical of this condition from a malignant neoplasm. Osteomyelitis spreads along the base of the skull and may cross the midline.

## Treatment

Surgery is usually not helpful or necessary. Control of the diabetes and prolonged (6 wk) IV therapy with a fluoroquinolone or with a combination of an aminoglycoside and a semisynthetic penicillin results in complete resolution in most cases. Longer therapy is required for extensive bone disease.

## Trauma

Hematoma: A subperichondrial hematoma may result from blunt trauma to the pinna. The pinna becomes a shapeless, reddish purple mass when blood collects between the perichondrium and the cartilage. Because the perichondrium carries the blood supply to the cartilage, avascular necrosis of the cartilage may occur. The "cauliflower ear" characteristic of wrestlers and boxers results from an organized, calcified hematoma. Treatment consists of evacuating the clot through an incision and approximating the skin and perichondrium to the cartilage with suction drainage to keep the cartilage close to its blood supply.

Lacerations: For lacerations of the pinna that penetrate the cartilage and skin on both sides, the skin margins are sutured, the cartilage is splinted externally with benzoin-impregnated cotton, and a protective dressing is applied. Sutures should not extend into the cartilage. Oral penicillin V 250 mg qid is given, particularly if there is any evidence of contamination.

Fractures: Forceful blows to the mandible may be transmitted to the anterior wall of the ear canal (posterior wall of the glenoid fossa). Displaced fragments from a fractured anterior wall may cause stenosis of the canal and must be reduced or removed using a general anesthetic.

## Tumors

Sebaceous cysts, osteomas, and keloids may arise in and occlude the ear canal, causing retention of cerumen and a conductive hearing loss. Excision is the treatment of choice.

Ceruminomas arise in the outer third of the ear canal. Although these neoplasms appear benign histologically, they behave in a malignant manner and should be excised widely.

Basal cell and squamous cell carcinomas frequently develop on the pinna after regular exposure to the sun. Early lesions can be successfully treated with cautery and curettage or radiation therapy. More advanced lesions affecting the cartilage require surgical excision of V-shaped wedges or of larger portions of the pinna. Invasion of cartilage makes radiation therapy less effective and surgery the preferred treatment. Basal cell and squamous cell carcinomas may also arise in or secondarily invade the ear canal. Persistent inflammation in chronic otitis media may predispose to the development of squamous cell carcinoma. Extensive resection is indicated, followed by radiation therapy. En bloc resection of the ear canal with sparing of the facial nerve is performed when lesions are limited to the canal and have not invaded the middle ear.

## Chapter 84. Tympanic Membrane And Middle Ear

A patient with a middle ear disorder may present with one or more of the following complaints: a feeling of fullness or pressure in the ear; constant or intermittent mild to excruciating pain; otorrhea; diminished hearing; tinnitus; and vertigo. In acute otitis media, systemic symptoms (eg, fever) are also common. Symptoms may begin with a feeling of fullness and progress serially and additively. Infants and children, especially, may be febrile and present with other prominent systemic manifestations (eg, anorexia, vomiting, diarrhea, lethargy).

Symptoms may result from infection, trauma, or disturbed pressure relationships secondary to eustachian tube obstruction. In determining the cause, a physician should elicit information about antecedent and associated symptoms (eg, rhinorrhea, nasal obstruction, sore throat, URI, allergic manifestations, headache, systemic symptoms). The appearance of the ear canal (external auditory canal) and tympanic membrane (see Fig. 84-1) often yields a diagnosis. The nose, nasopharynx, and oropharynx should be examined for signs of infection and allergy and for evidence of an underlying disorder--eg, a mass in the nasopharynx.

The function of the middle ear should be evaluated with pneumatic otoscopy, Weber's and Rinne's tests, tympanometry, and audiologic assessment (see Ch. 82).

### Trauma

The tympanic membrane (eardrum) may be punctured and the tympanum penetrated by objects placed in the ear canal (eg, cotton applicators) or entering the canal accidentally (eg, twigs on a tree or missiles, such as pencils or hot slag). Sudden overpressure (as in an explosion, a slap, or a swimming or diving accident) or sudden negative pressure (as in strong suction applied to the ear canal) can perforate the tympanic membrane. Penetration of the tympanic membrane may cause dislocations of the ossicular chain, fracture of the stapedial footplate, displacement of fragments of the ossicles, a perilymph fistula from the oval or round window, or facial nerve injury.

### Symptoms and Signs

Traumatic perforation of the tympanic membrane results in sudden severe pain followed by bleeding from the ear. Hearing loss and tinnitus occur. The hearing loss is more severe if the ossicular chain is disrupted or the inner ear injured. Vertigo suggests injury to

the inner ear. Purulent otorrhea may begin in 24 to 48 h, particularly if water enters the middle ear.

### Treatment

Many perforations can be monitored without the need for medical treatment. If infection is present or considered likely to occur, oral penicillin V 250 mg q 6 h should be given for 7 days. Aseptic technique is used when examining the ear. If possible, the displaced flaps of tympanic membrane are placed in their original positions, using local anesthetics and microscopic control, to facilitate healing. The ear is kept dry. Topical drugs with 2% acetic acid (5 drops tid) may be administered if the ear becomes infected, but no eardrops should be used prophylactically. Spontaneous closure of the perforation is usual, but if it does not occur within 2 mo, repair of the tympanic membrane is indicated. If a conductive hearing loss persists, suggesting discontinuity of the ossicular chain, the middle ear should be explored surgically and repaired. A sensorineural hearing loss or vertigo that persists for hours or longer after the injury may result from inner ear concussion but may also indicate penetration of the inner ear, which requires a prompt exploratory tympanotomy to assess and repair the damage as soon as possible.

## Barotitis Media (Aerotitis Media)

Damage to the middle ear due to ambient pressure changes.

During a sudden increase in ambient pressure--as during an airplane's descent or a deep-sea dive (see Chs. 283 and 285)--gas must move from the nasopharynx into the middle ear to maintain equal pressure on the two sides of the tympanic membrane. If the eustachian tube is not functioning properly, as in a URI or an allergy, the pressure in the middle ear is lower than the ambient pressure, resulting in retraction of the tympanic membrane. A transudate of blood from the vessels in the lamina propria of the mucous membrane forms in the middle ear. If the difference in pressure becomes great, ecchymosis and subepithelial hematoma may develop in the mucous membrane of the middle ear and in the tympanic membrane. A very large pressure differential causes bleeding into the middle ear and rupture of the tympanic membrane. A perilymph fistula through the oval or round window may develop. A pressure differential usually produces severe pain and a conductive hearing loss. A sensorineural hearing loss or vertigo during a descent suggests the possibility of a perilymph fistula; the same symptoms during ascent from a deep-sea dive suggest bubble formation in the inner ear.

A person with an acute URI or allergic reaction should be advised not to fly or dive. However, if these activities are undertaken, a nasal vasoconstrictor, such as phenylephrine 0.25% to 1.0% applied topically 30 to 60 min before descent, has prophylactic value.

## Infectious Myringitis (Bullous Myringitis)

Inflammation of the tympanic membrane secondary to viral or bacterial infections.

Vesicles develop on the tympanic membrane during viral infections or during acute bacterial (particularly Streptococcus [Diplococcus] pneumoniae) or mycoplasmal otitis media.

Pain is sudden in onset and persists for 24 to 48 h. Hearing loss and fever suggest bacterial otitis media.

### Treatment

Because differentiating viral from bacterial or mycoplasmal otitis is difficult, antibiotic therapy as for acute otitis media is indicated. Pain may be relieved by rupturing the vesicles with a myringotomy knife or by giving a narcotic, such as codeine 30 to 60 mg po q 4 h prn.

## Acute Otitis Media

A bacterial or viral infection in the middle ear, usually secondary to a URI.

Although acute otitis media can occur at any age, it is most common in young children, particularly from age 3 mo to 3 yr. Microorganisms may migrate from the nasopharynx to the middle ear by moving over the surface of the eustachian tube's mucous membrane or by propagating in the lamina propria of the mucous membrane as a spreading cellulitis or thrombophlebitis. Secondhand smoke has been implicated as a risk factor.

### Etiology

In newborns, gram-negative enteric bacilli, particularly Escherichia coli, and Staphylococcus aureus cause suppurative otitis media. After the neonatal period, E. coli rarely causes acute otitis media. In older infants and children < 14 yr, Streptococcus pneumoniae, Haemophilus influenzae, group A [beta]-hemolytic streptococci, Moraxella (Branhamella) catarrhalis, and S. aureus are the causative microorganisms. Viral otitis media is usually complicated by secondary invasion by one of these bacteria. In those > 14 yr, S. pneumoniae, group A [beta]-hemolytic streptococci, and S. aureus are the causative organisms; H. influenzae is less common. The relative frequency of the microorganisms identified as causing acute otitis media varies according to which are epidemic in a community at a given time. The frequency of otitis media caused by multidrug-resistant S. pneumoniae has increased in many communities. Klebsiella pneumoniae and Bacteroides sp rarely cause acute otitis media.

### Symptoms, Signs, and Complications

The first complaint usually is persistent severe earache. Hearing loss may occur. Fever (up to 40.5° C [105° F]), nausea, vomiting, and diarrhea may occur in young children. The tympanic membrane is erythematous and may bulge; landmarks become indistinct, and the light reflex is displaced. Bloody, then serosanguineous, and finally purulent otorrhea may follow spontaneous perforation of the tympanic membrane.

Serious complications include acute mastoiditis, petrositis, labyrinthitis, facial paralysis, conductive and sensorineural hearing loss, epidural abscess, meningitis (the most common intracranial complication), brain abscess, lateral sinus thrombosis, subdural empyema, and otitic hydrocephalus. Symptoms of an impending complication include headache, sudden profound hearing loss, vertigo, chills, and fever.

## Diagnosis and Treatment

Diagnosis is usually made clinically. If myringotomy is performed, exudate obtained during the procedure should be cultured, as should spontaneous otorrhea. Nasopharyngeal cultures may be helpful but do not correlate well with the causative agent.

Antibiotic therapy is generally indicated to relieve symptoms, hasten resolution of the infection, and reduce the chance of labyrinthine and intracranial infectious complications and of residual damage to the hearing mechanism in the middle ear.

Penicillin V 250 mg po q 6 h for 12 days is the drug of choice in patients > 14 yr. Amoxicillin 35 to 70 mg/kg/day po in three equal doses q 8 h for 7 to 12 days is preferred for those < 14 yr because of the frequency of H. influenzae infections. Treatment is continued for 12 to 14 days to ensure resolution and prevent sequelae. Subsequent therapy depends on cultures, sensitivities, and the clinical course. In penicillin allergy, erythromycin 250 mg po q 6 h for older children and adults or a combination of erythromycin 30 to 50 mg/kg/day po and sulfisoxazole 150 mg/kg/day po, both in equally divided doses q 6 h, for children < 14 yr may be given for 12 to 14 days. Sulfonamides are contraindicated in infants < 2 mo of age. Alternatively, trimethoprim and sulfamethoxazole (TMP-SMX) may be used: in infants > 2 mo and children, TMP-SMX 8/40 mg/kg/day in two divided doses q 12 h for 10 days; and in adults, TMP-SMX 160/800 mg q 12 h for 12 days. For children, another choice is a single IM dose of ceftriaxone (maximum, 50 mg/kg).

In resistant cases, a cephalosporin may be given for 12 days--eg, cefaclor (in children, 40 mg/kg/day in divided doses q 8 h; in adults, 250 mg q 8 h), cefuroxime (in children < 2 yr, 125 mg q 12 h; in children 2 to 12 yr, 250 mg q 12 h; in adults, 500 mg q 12 h), amoxicillin-clavulanate (in children, 40 mg/kg/day in three divided doses), clarithromycin (in children, 15 mg/kg/day in two divided doses), or cefixime (in children, 8 mg/kg/day usually in two divided doses; in adults, 200 mg q 12 h).

For improving eustachian tube function, topical vasoconstrictors, such as phenylephrine 0.25% 3 drops q 3 h, may be instilled into each nasal cavity while the patient is supine with the neck extended. Such therapy should not exceed 3 to 4 days. Systemic sympathomimetic amines, such as ephedrine sulfate, pseudoephedrine, or phenylpropanolamine 30 mg po (for adults) q 4 to 6 h for 7 to 10 days (removed from the US market in 2000), may also be helpful but generally are not recommended for children. If allergy is considered a significant factor, antihistamines, such as chlorpheniramine 4 mg (for adults) po q 4 to 6 h for 7 to 10 days, may improve eustachian tube function, but they are not indicated for persons without allergies.

Myringotomy should be considered if the tympanic membrane is bulging or if pain, fever, vomiting, and diarrhea are severe or persistent. The patient's hearing, tympanometry, and the appearance and movement of the tympanic membrane should be monitored until resolution is complete.

## Secretory Otitis Media (Serous Otitis Media)

An effusion in the middle ear resulting from incomplete resolution of acute otitis media or obstruction of the eustachian tube.

Secretory otitis media is common in children. The effusion may be sterile but usually contains pathogenic bacteria. Eustachian tube obstruction may be due to inflammatory processes in the nasopharynx, allergic manifestations, hypertrophic adenoids, or benign or malignant neoplasms.

Normally, the middle ear is ventilated 3 to 4 times/min as the eustachian tube opens during swallowing, and O2 is absorbed by the blood in the vessels of the middle ear mucous membrane. If the patency of the eustachian tube is impaired, a relative negative pressure develops within the middle ear.

### Symptoms and Signs

At first, the tympanic membrane retracts mildly, with displacement of the light reflex and accentuation of the landmarks. Then a transudate from the blood vessels in the mucous membrane develops in the middle ear, recognizable by the amber or gray appearance it gives the tympanic membrane and the immobility of the tympanic membrane. An air-fluid level or bubbles of air may be seen through the tympanic membrane. Conductive hearing loss occurs. Tympanometry demonstrates maximal compliance with negative pressures in the ear canal.

### Treatment

Because pathogenic bacteria may have a role in middle ear effusions, a trial of antibiotic therapy as for acute otitis media (see above) is often beneficial and is the first step to consider in therapy. It is effective in relieving eustachian tube obstruction due to bacterial infection and in sterilizing the middle ear.

Systemic sympathomimetic amines, such as ephedrine sulfate, pseudoephedrine, or phenylpropanolamine 30 mg po tid (for adults; removed from the US market in 2000), may encourage the eustachian tube to open by their vasoconstrictive effect. Antihistamines, such as loratadine 10 mg/day (for adults and children >= 12 yr old) po, may relieve eustachian tube obstruction in patients with allergies. Myringotomy may be necessary for aspiration of the fluid and for insertion of a tympanostomy tube, which allows ventilation of the middle ear and temporarily ameliorates the eustachian tube obstruction, regardless of the cause. Occasionally, the middle ear may be temporarily ventilated with the Valsalva maneuver or politzerization.

Correction of any underlying condition in the nasopharynx is required. Children may require adenoidectomy, removing lymphoid aggregations on the torus of the eustachian tube and in Rosenmüller's fossa as well as the central adenoid tissue mass, to eradicate persistent, recurrent secretory otitis media. Antibiotics should be given to treat bacterial rhinitis, sinusitis, and nasopharyngitis. Immunologic investigation is occasionally indicated. Any demonstrated allergen should be eliminated from the patient's environment, or immunotherapy should be tried.

## Acute Mastoiditis

Bacterial infection in the mastoid process resulting in coalescence of the mastoid air cells.

In acute purulent otitis media, the infection extends into the mastoid antrum and cells, but progression and destruction of the bony portions of the mastoid process may be aborted by appropriate antibiotic therapy. The responsible bacteria are the same as those causing acute otitis media (see above). Characteristically, streptococcal mastoiditis is preceded by early perforation of the tympanic membrane and profuse otorrhea. Pneumococcal mastoiditis is likely to be less symptomatic but just as destructive; advanced coalescence of the mastoid air cells may precede perforation of the tympanic membrane.

### Symptoms and Signs

Acute mastoiditis becomes clinically apparent a few days to >= 2 wk after the onset of untreated acute otitis media, as one of the cortices of the mastoid process is destroyed. A postauricular subperiosteal abscess may develop as the lateral mastoid cortex is destroyed. Redness, swelling, tenderness, and fluctuation develop over the mastoid process; the pinna is displaced laterally and inferiorly. An exacerbation of aural pain, fever, and otorrhea usually occurs. The pain tends to be persistent and throbbing; a creamy, profuse discharge is common.

In acute otitis media, the mastoid air cells are filled with fluid, and CT scans may show a soft tissue density due to purulent fluid, swollen mucous membrane, and granulation tissue in the air cells. In coalescent mastoiditis, cell partitions become indistinct. The individual septum can no longer be seen as the fluid- and tissue-filled air cells coalesce.

### Treatment

The initial antibiotic given should provide coverage for the common pathogens and be stable to [beta]-lactamase. Antibiotic penetration into the CNS is desirable if a complication seems imminent. A sample of the otorrhea is taken for culture and for determination of antibiotic sensitivities. Subsequent IV therapy depends on cultures, sensitivities, and the clinical course. Antibiotic therapy should be continued for at least 2 wk.

A subperiosteal abscess requires complete exenteration of mastoid air cells (mastoidectomy).

## Chronic Otitis Media

A permanent perforation of the tympanic membrane, with or without permanent changes in the middle ear.

Chronic otitis media can result from acute otitis media, eustachian tube obstruction, mechanical trauma, thermal or chemical burns, or blast injuries. It can be divided into two major categories, depending on the type of perforation: (1) that caused by central perforations of the pars tensa, and (2) that caused by the more dangerous attic perforations of the pars flaccida or marginal perforations of the pars tensa.

In central perforations, some substance of the tympanic membrane remains between the rim of the perforation and the bony sulcus tympanicus (see Fig. 84-2). These perforations result in a conductive hearing loss. Exacerbations of chronic otitis media may follow a URI or occur when water enters the middle ear during bathing or swimming. They are often caused by gram-negative rods or Staphylococcus aureus, resulting in painless, purulent otorrhea, which may be foul-smelling. Persistent exacerbations may produce aural polyps (granulation tissue that prolapses from the middle ear through the perforation into the ear canal) and destructive changes in the middle ear, such as necrosis of the long process of the incus. Aural polyps are a serious sign, almost invariably associated with cholesteatoma, a benign tumor.

Attic perforations of the pars flaccida lead into the epitympanum (see Fig. 84-2). Marginal perforations usually occur in the posterior-superior portion of the pars tensa, with no substance of the tympanic membrane between the edge of the perforation and the bony sulcus tympanicus (see Fig. 84-2). Marginal perforations result from an acute necrotizing otitis media that destroys large areas of the tympanic membrane, including the annulus tympanicus and the mucous membrane of the middle ear. These perforations, like central perforations, may cause a conductive hearing loss and exacerbations of otorrhea. Complications, such as labyrinthitis, facial paralysis, and intracranial suppuration, are more likely to occur with marginal than with central perforations.

Attic and marginal perforations are frequently associated with cholesteatomas. During the healing of acute necrotizing otitis media, the remaining epithelium of the mucous membrane and the stratified squamous epithelium of the ear canal migrate to cover the denuded areas. Once the stratified squamous epithelium is established in the middle ear, it begins to desquamate and accumulate, resulting in a cholesteatoma. Cholesteatomas may also develop from hyperplasia of the basal layer of the stratified squamous epithelium of the pars flaccida, from progressive retraction of the pars flaccida or the pars tensa, and from squamous metaplasia in the middle ear due to long-standing infection. The desquamated epithelium accumulates in ever-enlarging concentric layers, and collagenases in the epithelium destroy adjacent bone.

Cholesteatomas may be recognized during otoscopic examination by the white debris in the middle ear and the destruction of the ear canal bone adjacent to the perforation. Bone destruction due to an otherwise unsuspected cholesteatoma may be demonstrated on a CT scan. Aural polyps are usually associated with cholesteatomas. A cholesteatoma, particularly with an attic perforation, greatly increases the probability of a serious complication (eg, purulent labyrinthitis, facial paralysis, intracranial suppuration).

## Treatment

For exacerbations of both types of chronic otitis media, the ear canal and middle ear are thoroughly cleaned with suction and dry cotton wipes; then a solution of 2% acetic acid with hydrocortisone 1% is instilled into the ear, 5 to 10 drops tid for 7 to 10 days. Severe exacerbations require systemic therapy with a broad-spectrum antibiotic, such as amoxicillin 250 to 500 mg po q 8 h for 10 days. Subsequent treatment should be guided by cultures and sensitivities of the isolated microorganisms and by the patient's clinical response.

The middle ear can generally be repaired. A tympanoplasty restores the two major functions of the tympanic membrane: sound protection for the round window and sound pressure transformation through the ossicular chain to the oval window and inner ear. If the ossicular chain is disrupted, it may also be repaired during tympanoplasty. Patients with marginal or attic perforations and cholesteatomas require surgical removal of the cholesteatoma. Preservation and reconstruction of the middle ear mechanism is less likely when a cholesteatoma is present.

### Otosclerosis

A disease of the bone of the otic capsule and a common cause of progressive conductive hearing loss in an adult with a normal tympanic membrane.

Histologically, foci of otosclerosis are irregularly arranged, new, immature bone interspersed with numerous vascular channels. These foci enlarge, causing ankylosis of the stapedial footplate and a consequent conductive hearing loss. Otosclerosis also may produce a sensory hearing loss, particularly when the foci of otosclerotic bone are adjacent to the scala media.

Otosclerosis tends to be hereditary (probably autosomal dominant). About 10% of white adults have foci of otosclerosis, but only about 10% of affected persons develop conductive hearing loss. Otosclerosis becomes clinically evident in the late teenage and early adult years, with slowly progressive, asymmetric hearing loss. Fixation of the stapes may progress rapidly during pregnancy.

Treatment involves a trial with a hearing aid or microsurgical techniques. The latter consists of removing the stapes or a portion of it and replacing it with a prosthesis; the hearing loss is corrected in most cases.

### Neoplasms

Rarely, squamous cell carcinoma originates in the middle ear. The persistent otorrhea of chronic otitis media may be a predisposing factor. Radiation therapy and resection of the temporal bone are necessary.

Nonchromaffin paragangliomas (chemodectomas) arise in the temporal bone from glomus bodies in the jugular bulb (globus jugulare tumors) or the medial wall of the middle ear (globus tympanicum tumors). They produce a pulsatile red mass in the middle ear. The first symptom is often tinnitus that is synchronous with the pulse. Hearing loss develops, followed by vertigo. Excision is the treatment of choice. For tumors too large to excise, palliation is achieved with radiation therapy.

## Chapter 85. Inner Ear

(See also Hearing Loss and Vertigo in Ch. 82 and Hearing Deficits in Children in Ch. 260.)

The inner ear consists of the auditory portion (cochlea, saccule, acoustic nerve) and the vestibular portion (semicircular canals, utricle, superior and inferior vestibular nerves).

## Meniere's Disease

A disorder characterized by recurrent prostrating vertigo, sensory hearing loss, tinnitus, and a feeling of fullness in the ear associated with generalized dilation of the membranous labyrinth (endolymphatic hydrops).

The cause of Meniere's disease is unknown, and the pathophysiology is poorly understood. Attacks of vertigo appear suddenly, last from a few to 24 h, and subside gradually. The attacks are associated with nausea and vomiting. The patient may have a recurrent feeling of fullness or pressure in the affected ear; hearing in that ear tends to fluctuate but progressively worsens over the years. Tinnitus may be constant or intermittent and may be worse before, after, or during an attack of vertigo. Although usually only one ear is affected, both ears are affected in 10 to 15% of patients.

In Lermoyez's syndrome (a variant of Meniere's disease), hearing loss and tinnitus precede the first attack of vertigo by months or years, and the hearing may improve with the onset of vertigo.

## Treatment

Treatment is empirical. A number of surgical procedures are advocated for patients who are disabled by frequent vertiginous attacks. Vestibular neurectomy relieves the vertigo and usually preserves the hearing. A labyrinthectomy can be performed if the vertigo is sufficiently disabling and hearing has degenerated to a useless level.

Symptomatic relief of vertigo may be obtained with anticholinergic drugs (eg, oral scopolamine and atropine in OTC preparations, transdermal scopolamine, glycopyrrolate 1 or 2 mg po bid or tid, prochlorperazine 25 mg rectally q 12 h or 10 mg po tid or qid) to minimize vagal-mediated GI symptoms; antihistamines (eg, diphenhydramine, meclizine, or cyclizine 50 mg po or IM q 6 h) to sedate the vestibular system; or barbiturates (eg, pentobarbital 100 mg po or IM q 8 h) to provide general sedation. Diazepam 2 to 5 mg po q 6 to 8 h is particularly effective in relieving the distress of severe vertigo by sedating the vestibular system. Intratympanic gentamicin (chemical labyrinthectomy) in a series of applications is used for selected patients. The typical dose is > 1 mL (strength 30 mg/mL, made by diluting the commercially available preparation at 40 mg/mL) delivered to the middle ear through a myringotomy.

## Vestibular Neuronitis

A benign disorder characterized by sudden onset of severe vertigo that is persistent at first, then paroxysmal.

The disease is thought to be a neuronitis affecting the vestibular division of the 8th nerve and to be viral in origin because of its frequent epidemic occurrence, particularly among adolescents and young adults.

The first attack of vertigo is severe, is associated with nausea and vomiting, and lasts for 7 to 10 days. There is persistent nystagmus toward the affected side. The condition is self-limited. It may occur as a single episode or as several attacks over 12 to 18 mo, with each subsequent attack being less severe and shorter. There is no associated hearing loss or tinnitus.

The diagnostic evaluation should include an audiologic assessment, electronystagmography with caloric testing, and MRI of the head with gadolinium enhancement, giving particular attention to the internal auditory canals to exclude other diagnostic possibilities, such as cerebellopontine angle tumor and brain stem hemorrhage or infarction.

### Treatment

Acute attacks of vertigo may be suppressed symptomatically as in Meniere's disease (see above). If vomiting is prolonged, IV fluids and electrolytes may be required for replacement and maintenance.

## Benign Paroxysmal Positional Vertigo (Benign Postural Or Positional Vertigo)

Violent vertigo lasting < 30 sec and induced by certain head positions.

### Etiology and Symptoms

Granular basophilic masses in the cupula of the posterior semicircular canal have been demonstrated. The cupular deposits (cupulolithiasis) may be composed of calcium carbonate derived from the otoliths, which are calcium carbonate crystals normally embedded in the saccule and utricle, different parts of the inner ear. Etiologic factors appear to be spontaneous degeneration of the utricular otolithic membranes, labyrinthine concussion, otitis media, ear surgery, and occlusion of the anterior vestibular artery.

Benign paroxysmal positional vertigo occurs when the patient lies on one ear or the other or when he tips his head backward to look up. Nystagmus also occurs, but there is no associated hearing loss or tinnitus. Benign paroxysmal positional vertigo usually subsides in several weeks or months but may recur after months or years.

### Diagnosis

A provocative test for positional nystagmus may be performed. The patient is first seated on an examining table; then with his head turned to the side, he quickly assumes the supine position with his head dependent over the end of the table. After a latent period of several seconds, severe vertigo occurs; it is likely to last 15 to 20 sec and is accompanied by rotary nystagmus. If the left ear is affected, the nystagmus is clockwise when the head is turned to the left; if the right ear is affected, the nystagmus is counterclockwise. When the patient sits up, the response recurs, but the nystagmus is rotary in the opposite direction and is milder. The response fatigues, so that if the test is repeated immediately, the response is diminished.

Positional nystagmus may occur with end-organ or CNS lesions. The latency and fatigability of the response, the severe subjective sensation, the limited duration, and the direction of the rotary nystagmus distinguish benign paroxysmal positional vertigo from a CNS lesion. The positional nystagmus of CNS lesions lacks latency, fatigability, and severe subjective sensation and may continue for as long as the position is maintained. Nystagmus due to a CNS lesion may be vertical or change direction, and if rotary, it is likely to be perverted (ie, not in the anticipated direction).

The diagnostic evaluation should include an audiologic assessment, electronystagmography with caloric testing, and MRI of the head with gadolinium enhancement, giving particular attention to the internal auditory canals to exclude other conditions, such as acoustic neuroma.

### Treatment

The patient is instructed to avoid the provocative position. If benign positional paroxysmal vertigo lasts for as long as a year, it usually can be relieved by dividing the nerve to the posterior semicircular canal of the affected ear at tympanotomy. In some cases, this procedure results in hearing loss.

## Herpes Zoster Oticus (Ramsay Hunt's Syndrome, Viral Neuronitis And Ganglionitis, Geniculate Herpes)

Invasion of the 8th nerve ganglia and the geniculate ganglion of the facial nerve by the herpes zoster virus, producing severe ear pain, hearing loss, vertigo, and paralysis of the facial nerve.

Vesicles can be seen on the pinna and in the ear canal (external auditory canal) along the distribution of the sensory branch of the facial nerve. Other cranial nerves are often involved, and some degree of meningeal inflammation is common. Lymphocytes may be present in the CSF, and its protein content is often increased. Evidence of a mild generalized encephalitis can be found in many patients. Hearing may be lost permanently or recovered partially or completely. Vertigo lasts for days to several weeks. Facial paralysis may be transient or permanent.

### Treatment

Although there is no reliable evidence that corticosteroids, antiviral drugs, or decompression makes a difference, they are the only possibly useful treatments. Corticosteroid therapy is the treatment of choice and should be started promptly: eg, prednisone 40 mg/day po for 2 days, then 30 mg/day po for 7 to 10 days, followed by gradual tapering of the dose. Acyclovir 1 g/day po in five divided doses for 10 days may shorten the clinical course. Pain is relieved with codeine 30 to 60 mg po q 3 to 4 h prn; the vertigo is effectively suppressed with diazepam 2 to 5 mg po q 4 to 6 h. Decompression of the fallopian canal, indicated when the nerve excitability declines or when electroneurography demonstrates a 90% decrement, occasionally relieves the facial paralysis.

## Purulent Labyrinthitis

Invasion of the inner ear by bacteria.

Purulent (suppurative) labyrinthitis may be secondary to acute otitis media or purulent meningitis. In acute otitis media, microorganisms may enter the inner ear through the oval and round windows; in purulent meningitis, they may enter through the cochlear aqueduct. Purulent labyrinthitis is frequently followed by meningitis as the microorganisms gain access to the subarachnoid space through the cochlear aqueduct.

Purulent labyrinthitis is characterized by severe vertigo and nystagmus. It invariably results in complete hearing loss and, in chronic otitis media and cholesteatoma, is often followed by facial paralysis.

## Treatment

Treatment with IV antibiotics appropriate for meningitis is usually adequate. Rarely, labyrinthectomy to drain the inner ear or radical mastoidectomy is required.

## Sudden Deafness

Severe sensorineural hearing loss that usually occurs in only one ear and develops over a period of a few hours or less.

Sudden deafness occurs in about 1 of 5000 persons every year (see also Hearing Loss in Ch. 82). Although the sudden onset suggests a vascular cause (embolism, thrombosis, or hemorrhage) by analogy with vascular accidents in the CNS, the evidence supports a viral cause in most cases. Sudden deafness tends to occur in children and in young or middle-aged adults who have no evidence of vascular disease. The histopathologic findings in the temporal bone of persons with sudden deafness are unlike those seen in the inner ear of animals with experimental vascular occlusion or embolization but are similar to those seen in human viral infections of the inner ear (viral endolymphatic labyrinthitis) that result in sudden deafness--eg, mumps and measles. The viruses that cause influenza, chickenpox, and mononucleosis; adenoviruses; and others may cause sudden deafness.

The pathologic findings in persons with persistent hearing loss due to viral endolymphatic labyrinthitis are similar, regardless of the causative virus. The organ of Corti is missing, and ganglion cell populations are reduced in the basal turn. Individual hair cells tend to be missing. The stria vascularis atrophies. The tectorial membrane is often rolled up and ensheathed in a syncytium. Reissner's (vestibular) membrane may be collapsed and adhere to the basilar membrane.

Perilymph fistulas between the inner and middle ears occasionally result from large ambient pressure changes or strenuous activities, such as weight lifting. Fistulas in the oval or round window result in a sudden or fluctuating sensory hearing loss and vertigo. The patient may hear an explosive sound in the affected ear when the fistula occurs. The fistula can be demonstrated by combining the pressure changes in the ear canal used in

tympanometry with electronystagmography. Nystagmus resulting from pressure changes in the ear canal can be detected by electronystagmography and suggests a perilymph fistula.

### Symptoms and Signs

Hearing loss is usually profound, but hearing returns to normal in most patients and is partially recovered in others. If hearing is going to return, it is likely to do so in 10 to 14 days. Tinnitus and vertigo may be present initially, although the vertigo usually subsides in several days.

### Treatment

Although vasodilators, anticoagulants, low molecular weight dextran, corticosteroids, and vitamins have been advocated, no form of treatment is of proven value. Because micropetechiae and extravasation of blood are characteristic of virus-induced inflammatory reactions, vasodilation and anticoagulation may not be indicated. Furthermore, in an inflammatory reaction, the cochlear blood flow is already increased as much as is beneficial. The use of corticosteroids appears rational--eg, prednisone 60 mg/day po for 2 days, then 40 mg/day po for 5 to 7 days, followed by tapering the dose. Bed rest also seems advisable.

Generally, surgical exploration of the middle ear should be performed if a perilymph fistula is suspected, and the fistula should be repaired with an autogenous graft of fascia.

## Noise-Induced Hearing Loss

Any source of intense noise, such as woodworking equipment, chain saws, internal combustion engines, heavy machinery, gunfire, or aircraft, may damage the inner ear. Such activities as shooting, snowmobiling, flying, and attending rock concerts are associated with noise-induced hearing loss. Exposure to intense noise results in loss of hair cells in the organ of Corti. Although persons vary greatly in susceptibility to noise-induced hearing loss, nearly everyone loses some hearing if exposed to sufficiently intense noise for an adequate time. Any noise > 85 dB is damaging. High-frequency tinnitus usually accompanies the hearing loss. Loss occurs first at 4 kHz and gradually occurs in the lower and higher frequencies as exposure continues. In contrast to most sensorineural hearing losses, loss is less at 8 kHz than at 4 kHz. Blast injury (acoustic trauma) produces the same kind of sensory hearing loss.

Prevention depends on limiting the length of exposure, reducing the noise at its source, and isolating the person from the sound source. As the intensity of the noise increases, the duration of exposure must be reduced to prevent damage to the inner ear. Noise may be attenuated by wearing ear protectors, eg, plastic plugs in the ear canals or glycerin-filled muffs over the ears.

When noise-induced hearing loss interferes with communication, a hearing aid is usually helpful (see Hearing Aids in Ch. 82).

## Presbycusis

Sensorineural hearing loss that occurs in people as they age and that may be affected by genetic or acquired factors.

(See also Hearing Loss in Ch. 82.)

Presbycusis begins after age 20 but is usually significant only in persons over 65. Men are affected more often and more severely than women. Stiffening of the basilar membrane and deterioration of the hair cells, stria vascularis, ganglion cells, and cochlear nuclei may play a role in pathogenesis, and presbycusis appears to be related in part to noise exposure. It first affects the highest frequencies (18 to 20 kHz) and gradually affects the lower frequencies; it usually begins to affect the 4- to 8-kHz range by age 55 to 65, although variation is considerable. Some persons are severely handicapped by age 60, and some are essentially untouched at age 90. The loss of high-frequency hearing makes discrimination of speech particularly difficult. Thus, many persons who have this type of loss have difficulty understanding conversation, particularly when background noise is present, and complain that others mumble.

Speech reading (lipreading), auditory training for making maximum use of nonauditory clues, and amplification with a hearing aid are helpful.

## Drug-Induced Ototoxicity

Aminoglycoside antibiotics, salicylates, quinine and its synthetic substitutes, and diuretics (ethacrynic acid and furosemide) can be ototoxic. Although these drugs affect both the auditory and vestibular portions of the inner ear, they are particularly toxic to the organ of Corti (cochleotoxic). Nearly all ototoxic drugs are eliminated through the kidneys, and renal impairment predisposes to the accumulation of toxic levels. Ototoxic drugs should not be prescribed as topical medication for the ear when the tympanic membrane is perforated because they can be absorbed into the inner ear fluids through the secondary tympanic membrane at the round window.

Streptomycin damages the vestibular portion of the inner ear more readily than the auditory portion. Although vertigo and difficulty in maintaining balance tend to be temporary and eventually completely compensated, severe loss of vestibular sensitivity may persist, sometimes permanently, causing difficulty when walking, especially in the dark, and Dandy's syndrome (bouncing of the environment with each step). About 4 to 15% of patients who receive 1 g/day for > 1 wk develop a measurable hearing loss, which usually appears after a short latent period (7 to 10 days) and slowly worsens if treatment is continued. Complete, permanent deafness may follow.

Neomycin has the greatest cochleotoxic effect of all antibiotics. When large doses are given orally or by colonic irrigation for intestinal sterilization, enough of the drug may be absorbed to affect hearing, particularly if GI ulceration or other mucosal lesions are present. Neomycin should not be used for wound irrigation or for intrapleural or intraperitoneal irrigation because massive amounts of the drug may be retained and absorbed, causing deafness. Kanamycin and amikacin are close to neomycin in cochleotoxic potential.

Viomycin has both cochlear and vestibular toxicity. Vancomycin causes hearing loss, especially in the presence of renal insufficiency. Gentamicin and tobramycin have vestibular and cochlear toxicity.

Ethacrynic acid given IV has caused profound, permanent hearing loss in gravely ill patients with renal failure who were receiving concomitant aminoglycoside antibiotics. Similarly, IV furosemide has caused transient and permanent hearing loss in patients with renal failure or who were receiving concomitant aminoglycoside antibiotics.

In very high doses, salicylates produce hearing loss and tinnitus that are usually reversible. Quinine and its synthetic substitutes can cause permanent hearing loss.

## Precautions

Ototoxic antibiotics should be avoided in pregnancy. Elderly persons and persons with a preexisting hearing loss should not be treated with ototoxic drugs if other effective drugs are available. If possible, before treatment with an ototoxic drug (especially an ototoxic antibiotic) is begun, hearing should be measured to document preexisting hearing loss. Hearing should be monitored audiometrically during treatment. The highest frequencies are usually affected first, and high-pitched tinnitus or vertigo may develop, although they are not reliable warning symptoms. If renal function is impaired, the dosage of renally eliminated ototoxic drugs should be adjusted so that the blood levels do not exceed those required therapeutically. Peak and trough serum levels of the drug should be monitored to ensure that adequate therapeutic levels have been achieved but not exceeded. Although susceptibility to ototoxic drugs varies somewhat among persons, hearing can usually be conserved by not exceeding the recommended blood level.

## Temporal Bone Fractures

Ecchymosis in the postauricular skin (Battle's sign) suggests a fracture of the temporal bone. Bleeding from the ear after a skull injury strongly suggests such a fracture. The bleeding may be medial to an intact tympanic membrane, or it may come from the middle ear through a ruptured tympanic membrane or from a fracture line in the ear canal. A hematotympanum makes the tympanic membrane appear blue-black. CSF otorrhea indicates a communication between the middle ear and the subarachnoid space. Longitudinal fractures, which are parallel to the petrous pyramid in 80% of cases, extend through the middle ear and rupture the tympanic membrane; they produce facial paralysis in 15% of cases and a profound sensorineural hearing loss in 35%. Middle ear damage may include disruption of the ossicular chain. Transverse fractures (20% of cases) cross the fallopian canal and the cochlea and nearly always produce facial paralysis and permanent hearing loss. Hearing can be assessed initially with Weber's and Rinne's tests and subsequently with audiometry (see Clinical Measurement of Hearing in Ch. 82). The fracture can usually be demonstrated on a CT scan of the head with special attention to the temporal bone.

## Treatment

Penicillin G 1.6 million U IV q 6 h can be given for 7 to 10 days in an attempt to prevent meningitis. However, this treatment increases the risk of organisms becoming

resistant. Persistent facial paralysis requires nerve decompression. Tympanoplasty with repair of the ossicular chain is performed weeks or months later. Exploratory tympanotomy to search for a perilymph fistula may be indicated when a patient has fluctuating hearing loss or other clinical evidence suggesting a fistula.

### Acoustic Neuroma
### (Acoustic Schwannomas, Acoustic Neurinomas, 8th Nerve Tumors)

Acoustic neuromas are derived from Schwann cells (see also Neurofibromatosis in Ch. 183). They arise twice as often from the vestibular division of the 8th nerve as from the auditory division and account for about 7% of all intracranial tumors.

As the tumor grows larger, it projects from the internal auditory meatus into the cerebellopontine angle and begins to compress the cerebellum and brain stem. The 5th cranial nerve and later the 7th are affected.

#### Symptoms, Signs, and Diagnosis

Hearing loss and tinnitus are early symptoms. Although the patient complains of dizziness and unsteadiness, true vertigo is not usually present. The sensorineural hearing loss (see Differentiation of Sensory and Neural Hearing Losses in Ch. 82) is characterized by greater impairment of speech discrimination than would be expected with a cochlear lesion. Recruitment is absent, and tone decay is marked. Acoustic reflex decay, the absence of waveforms, and increased latency of the 5th waveform in auditory brain stem response testing are further evidence of a neural lesion. As a rule, caloric testing demonstrates marked vestibular hypoactivity (canal paresis). Early diagnosis is based on the audiologic assessment, particularly auditory brain stem response and MRI with gadolinium enhancement.

#### Treatment

Small tumors may be removed with microsurgical techniques that preserve the facial nerve, using a middle cranial fossa route to preserve the remaining hearing or a translabyrinthine route if no useful hearing remains. Large tumors are removed by a combined translabyrinthine and suboccipital approach. Alternatively, radiation therapy or gamma knife radiosurgery may be used.

## Chapter 86. Nose And Paranasal Sinuses

(See also Foreign Bodies in Ch. 272.)

The nose, including the nasal septum that divides the nasal cavity into two passages, is made of bone and cartilage. The paranasal sinuses--maxillary, frontal, ethmoid, and sphenoid--open into the nasal cavity.

### Fractures Of The Nose

The nasal bones are fractured more frequently than are other facial bones. A nasal fracture may affect the ascending processes of the maxilla and the septum, and the torn

mucous membrane results in nosebleed. Soft tissue swelling develops promptly and may obscure the break. Septal hematomas may occur between the perichondrium and the quadrilateral cartilage and may become infected; abscess formation leads to avascular and septic necrosis of the cartilage, with a saddle deformity of the nose.

### Diagnosis and Treatment

A fracture should be suspected if blunt injury causes bleeding from the nose. The diagnosis can ordinarily be established by gently palpating the dorsum (bridge) of the nose for deformity, instability, crepitus, and point tenderness and is confirmed by x-ray. The most common deformity is deviation of the dorsum to one side and depression of the nasal bone and ascending process of the maxilla on the other side.

Nasal fractures in adults may be reduced after using a local anesthetic; children, however, require a general anesthetic. Then the fracture is manipulated into a good position by internal and external traction: A blunt elevator is placed under the depressed nasal bone, which is lifted anteriorly and laterally while pressure is applied to the other side of the nose to bring the nasal dorsum to the midline. The position of the nose may be stabilized with internal packing and external splinting. Septal hematomas must be immediately incised and drained to prevent infection and cartilage necrosis. Septal fractures are difficult to hold in position and often require septal surgery later.

### Septal Deviation And Perforation

Deviations of the nasal septum due to developmental abnormalities or trauma are common but often are asymptomatic and require no treatment. Septal deviation may cause nasal obstruction and predispose the patient to sinusitis (particularly if the deviation obstructs the ostium of a paranasal sinus) and to epistaxis as a result of drying air currents. Treatment of symptomatic septal deviation consists of septoplasty (septal reconstruction).

Septal ulcers and perforations may result from nasal surgery; repeated trauma, such as that from picking the nose; cocaine use; or conditions such as TB, syphilis, leprosy, and Wegener's granulomatosis. Crusting around the margins and repeated epistaxis may result. Small perforations may whistle. Topically applied bacitracin 500 U/g in a petrolatum base reduces crusting. Symptomatic perforations of the nasal septum are occasionally repaired using buccal or septal mucous membrane flaps; closing the perforation with a Silastic septal button is a reliable option.

### Epistaxis (Nosebleed)

Epistaxis occurs secondary to local infections, such as vestibulitis, rhinitis, and sinusitis; systemic infections, such as scarlet fever, malaria, and typhoid fever; drying of the nasal mucous membrane; trauma (digital, as in picking the nose, or blunt, as in nasal fractures); arteriosclerosis; hypertension; a tumor in a paranasal sinus or the nasopharynx; septal perforations; and bleeding tendencies associated with aplastic anemia, leukemia, thrombocytopenia, liver disease, hereditary coagulopathies, and the Rendu-Osler-Weber syndrome (hereditary hemorrhagic telangiectasia--see Ch. 134).

## Treatment

Most nasal bleeding originates from a plexus of vessels in the anteroinferior septum (Kiesselbach's area). Bleeding may be controlled by pinching the nasal alae together for 5 to 10 min. If this maneuver fails, the bleeding site must be found. Bleeding can be controlled temporarily by applying pressure with a cotton pledget impregnated with a vasoconstrictor, such as phenylephrine 0.25%, and a topical anesthetic, such as lidocaine 2%, until the site is anesthetized. The bleeding point may be cauterized, using electrocautery or silver nitrate in a 75% applicator bead, which may control bleeding without burning mucous membranes too deeply.

For epistaxis due to a bleeding tendency, petrolatum gauze is used to apply pressure as atraumatically as possible to the bleeding point. Cautery is not used because the periphery of a cauterized area might begin to bleed. The bleeding disorder should be identified and corrected, if possible.

In arteriosclerosis and hypertension, bleeding is likely to occur far posterior in the inferior meatus and may be difficult to control. Ligating the internal maxillary artery and its branches or packing the posterior part of the nasal cavity is required to control the bleeding. The arteries may be ligated with clips using microscopic guidance and a surgical approach through the maxillary sinus. A postnasal pack is placed in the posterior part of the nasal cavity to obstruct the choana. The pack consists of 4-in gauze squares folded, rolled, and tied into a tight bundle with two strands of heavy silk suture. The ends of one suture are tied to a catheter that has been introduced through the nasal cavity on the side of the bleeding and brought out through the mouth. As the catheter is withdrawn from the nose, the postnasal pack is placed behind the soft palate in the nasopharynx. The second suture is trimmed below the level of the soft palate so that it can be used to remove the pack. (Alternatively, the balloon of a Foley catheter may be inflated in the nasopharynx to obstruct the choana.) The nasal cavity, particularly the posterior part of the inferior meatus, is firmly packed with petrolatum gauze, and the first suture is tied over a roll of gauze at the anterior nares to secure the postnasal pack. The packing remains in place for 4 days. An antibiotic is given to prevent sinusitis and otitis media. Optimally, the choice of an antibiotic is based on knowledge of locally prevalent patterns of bacteria and antibiotic-resistant strains. Postnasal packing lowers the arterial PO2, and supplementary O2 should be given while the packing is in place.

In the Rendu-Osler-Weber syndrome, multiple severe nosebleeds may result from arteriovenous aneurysms in the mucous membrane, causing severe and persistent anemia that is not easily corrected with administration of iron. A split-thickness skin graft (septal dermatoplasty) reduces the number of nosebleeds and allows the anemia to be corrected.

Severe epistaxis is often caused by liver disease. Blood may be swallowed in large amounts and should be eliminated as promptly as possible with enemas and cathartics; the GI tract should be sterilized with nonabsorbable antibiotics (eg, neomycin 1 g po qid) to prevent the breakdown of blood and the absorption of ammonia.

Need for blood replacement is determined by the Hb level, vital signs, and central venous pressure.

## Nasal Vestibulitis

Infection of the nasal vestibule.

Low-grade infections and folliculitis produce annoying crusts, and bleeding occurs as the crusts come off. Bacitracin ointment 500 U/g applied topically bid for 14 days is effective.

Furuncles of the nasal vestibule are usually staphylococcal; they may develop into spreading cellulitis of the tip of the nose. Systemic antibiotics should be given along with hot soaks; penicillin V is the drug of choice, except when antibiotic-resistant bacteria are known to be locally prevalent. Furuncles of the nose and surrounding tissues should be allowed to drain spontaneously. Because incision and drainage increase the risk of retrograde thrombophlebitis and subsequent cavernous sinus thrombosis, they are contraindicated.

## Rhinitis

Edema and vasodilation of the nasal mucous membrane, nasal discharge, and obstruction.

(See also Allergic Rhinitis in Ch. 148.)

Acute rhinitis is the usual manifestation of a common cold (see The Common Cold in Ch. 162); it may also be caused by streptococcal, pneumococcal, or staphylococcal infections. Chronic rhinitis is generally a prolongation of subacute inflammatory or infectious rhinitis but may also occur in syphilis, TB, rhinoscleroma, rhinosporidiosis, leishmaniasis, blastomycosis, histoplasmosis, and leprosy--all of which are characterized by granuloma formation and destruction of soft tissue, cartilage, and bone. These conditions produce nasal obstruction, purulent rhinorrhea, and frequent bleeding. Rhinoscleroma causes progressive nasal obstruction from indurated inflammatory tissue in the lamina propria. Rhinosporidiosis is characterized by bleeding polyps.

## Diagnosis and Treatment

Diagnosis and treatment of acute bacterial rhinitis are based on pathogen identification and antibiotic sensitivities. Topical vasoconstriction with a sympathomimetic amine, such as phenylephrine 0.25%, given q 3 to 4 h for not more than 7 days relieves stuffiness, as do systemic sympathomimetic amines, such as pseudoephedrine 30 mg po q 4 to 6 h.

Diagnosis in chronic rhinitis is based on demonstrating the causative microorganism by culture or biopsy. Treatment consists of appropriate antimicrobial drugs.

## Atrophic Rhinitis

A chronic rhinitis characterized by an atrophic and sclerotic mucous membrane, abnormal patency of the nasal cavities, crust formation, and a foul odor.

The cause is unknown, although bacterial infection frequently plays a role. The mucous membrane changes from ciliated pseudostratified columnar epithelium to stratified

squamous epithelium, and the lamina propria is reduced in amount and vascularity. Anosmia results, and epistaxis may be recurrent and severe. Atrophic rhinitis is differentiated from other forms of chronic rhinitis by the abnormal patency of the nasal cavities, caused by atrophy of the blood vessels and the seromucinous glands in the lamina propria.

### Treatment

Treatment is directed at reducing the crusting and eliminating the odor. Topical antibiotics (such as bacitracin 500 U/g in a petrolatum base), topical or systemic estrogens, and vitamins A and D may be effective. Occluding or reducing the patency of the nasal cavities, surgically or with a pledget of lamb's wool, decreases the crusting caused by the drying effect of air flowing over the atrophic mucous membrane.

## Vasomotor Rhinitis

A chronic rhinitis characterized by intermittent vascular engorgement of the nasal mucous membrane, sneezing, and watery rhinorrhea.

The etiology is uncertain, and no allergy can be identified. The turgescent mucous membrane varies from bright red to purple. The condition is marked by periods of remission and exacerbation. It appears to be aggravated by a dry atmosphere. Vasomotor rhinitis is differentiated from specific viral and bacterial infections of the nose by the lack of purulent exudate and crusting. It is differentiated from allergic rhinitis by the absence of an identifiable allergen.

### Treatment

Treatment is empirical and not always satisfactory. Patients benefit from humidified air, eg, from a humidified central heating system or a vaporizer in the workroom and bedroom. Systemic sympathomimetic amines (eg, for adults, pseudoephedrine 30 mg po q 4 to 6 h prn) relieve symptoms but are not recommended for regular long-term use. Topical vasoconstrictors should be avoided because they cause the vasculature of the nasal mucous membrane to lose its sensitivity to other vasoconstrictive stimuli--eg, the humidity and temperature of inspired air. Vasodilation results, except after application of a strong stimulus, such as a topical sympathomimetic amine.

## Polyps

Fleshy outgrowths of the mucous membrane of the nose.

Allergic rhinitis predisposes to polyp formation. Polyps may also occur in acute and chronic infections and in cystic fibrosis (see Ch. 267); they may regress after an acute infection resolves. Nasal polyps form at the site of massive dependent edema in the lamina propria of the mucous membrane, usually around the ostia of the maxillary sinuses. A developing polyp is teardrop-shaped; when mature, it resembles a peeled seedless grape. Bleeding polyps occur in rhinosporidiosis. Unilateral polyps occasionally occur in association with or represent benign or malignant neoplasms of the nose or paranasal sinuses. Persons with nasal polyps are more likely to be allergic to aspirin.

## Treatment

Corticosteroids, such as beclomethasone dipropionate (42 μg/spray) or flunisolide (25 μg/spray) aerosols, given as one or two sprays in each nasal cavity bid, sometimes reduce or eliminate polyps, although surgical removal is often still required. Polyps that obstruct the airway or promote sinusitis should be removed, as should unilateral polyps that may be obscuring benign or malignant neoplasms. However, polyps tend to recur unless the underlying allergy or infection is controlled. After removal of nasal polyps, topical beclomethasone, flunisolide, or cromolyn therapy tends to retard recurrence. In severe, recurrent cases, maxillary sinusotomy or ethmoidectomy may be indicated.

## Wegener's Granulomatosis

This vasculitis of unknown etiology is characterized by granulomas of the nose and lungs and by glomerulitis; it is discussed fully in Ch. 50. However, most destructive lesions of the bone, cartilage, and soft tissue in the nose and paranasal sinuses are ultimately identified on biopsy as malignant neoplasms, such as lymphoma or carcinoma.

## Disorders of Smell and Taste

Because distinct flavors depend on aromas to stimulate the olfactory chemoreceptors, taste and smell are physiologically interdependent, and dysfunction of one often disturbs the other. Disorders of smell and taste are rarely incapacitating or life threatening, so they often do not receive close medical attention. However, the inability to detect certain odors, such as gas, may be dangerous, and several systemic and intracranial disorders should be eliminated before dismissing symptoms as harmless. Whether brain stem disease (involvement of the nucleus solitarius) can cause disorders of smell and taste is uncertain because other neurologic manifestations are usually overshadowing.

Anosmia (loss of the sense of smell) is probably the most common abnormality (see below). Hyperosmia (increased sensitivity to odors) usually reflects a neurotic or histrionic personality. Dysosmia (disagreeable or distorted sense of smell) may occur with infection of the nasal sinuses, partial damage to the olfactory bulbs, or psychologic depression. Some cases, accompanied by a disagreeable taste, result from poor dental hygiene. Uncinate epilepsy can produce brief, vivid, unpleasant olfactory hallucinations. Hyposmia (diminished sense of smell) and hypogeusia (diminished sense of taste) can follow acute influenza, usually temporarily.

Drying of the oral mucosa from heavy smoking, Sjögren's syndrome, radiation therapy of the head and neck, or desquamation of the tongue can impair taste, and various drugs (eg, amitriptyline, vincristine) alter taste. In all instances, the gustatory receptors are diffusely involved. When limited to one side of the tongue (eg, in Bell's palsy), ageusia (loss of the sense of taste) is rarely noticed.

Rarely, idiopathic dysgeusia (distorted sense of taste), hypogeusia, and dysosmia respond to zinc supplementation.

## Anosmia

Loss of the sense of smell.

Anosmia requires thorough evaluation for intranasal and intracranial diseases. Loss of smell occurs when intranasal swelling or other obstruction prevents odors from gaining access to the olfactory area; when the olfactory neuroepithelium is destroyed, as in viral infections, atrophic rhinitis, or the chronic rhinitis of granulomatous diseases and neoplasms; or when the olfactory nerve fila, bulbs, tracts, or central connections are destroyed, eg, by head trauma, intracranial surgery, infections, or neoplasms. Head trauma is a major cause of anosmia in young adults. Viral infections are a major cause in older adults. Anosmia occurs congenitally in male hypogonadism (Kallmann's syndrome). Most patients with anosmia have normal perception of salty, sweet, sour, and bitter substances, but they lack flavor discrimination, which is largely dependent on olfaction; therefore, they often complain of losing the sense of taste (ageusia). If unilateral, anosmia is often unrecognized.

Diagnostic evaluation requires examination of the cranial nerves (see Neurologic Examination in Ch. 165) and of the upper respiratory tract (particularly the nose and nasopharynx), psychophysical assessment of odor and taste identification and threshold detection, and enhanced CT of the head to rule out neoplasms and unsuspected fractures of the floor of the anterior cranial fossa.

Treatment of allergic or bacterial rhinitis and sinusitis or removal of nasal polyps and benign neoplasms may result in recovery of the sense of smell. Conditions causing destruction of the olfactory neuroepithelium or its central pathways do not lend themselves to effective treatment, although spontaneous recovery may follow regeneration of these tissues.

## Sinusitis

Inflammation of the paranasal sinuses due to viral, bacterial, or fungal infections or allergic reactions.

Acute sinusitis is caused by streptococci, pneumococci, Haemophilus influenzae, or staphylococci and is usually precipitated by an acute viral respiratory tract infection. Chronic sinusitis may be exacerbated by a gram-negative rod or anaerobic microorganisms. In a minority of cases, chronic maxillary sinusitis is secondary to dental infection.

In a URI, the swollen nasal mucous membrane obstructs the ostium of a paranasal sinus, and the O2 in the sinus is absorbed into the blood vessels of the mucous membrane. The resulting relative negative pressure in the sinus (vacuum sinusitis) is painful. If the vacuum is maintained, a transudate from the mucous membrane develops and fills the sinus; the transudate serves as a medium for bacteria that enter the sinus through the ostium or via a spreading cellulitis or thrombophlebitis in the lamina propria of the mucous membrane. An outpouring of serum and leukocytes to combat the infection results, and painful positive pressure develops in the obstructed sinus. The mucous membrane becomes hyperemic and edematous.

## Symptoms, Signs, and Diagnosis

Acute sinusitis and chronic sinusitis produce similar symptoms and signs. The area over the affected sinus may be tender and swollen. Maxillary sinusitis causes pain in the maxillary area, toothache, and frontal headache. Frontal sinusitis produces pain in the frontal area and frontal headache. Ethmoid sinusitis causes pain behind and between the eyes and a frontal headache often described as splitting. Pain from sphenoid sinusitis is less well localized and is referred to the frontal or occipital area. Malaise may be present. Fever and chills suggest an extension of the infection beyond the sinuses.

The nasal mucous membrane is red and turgescent; yellow or green purulent rhinorrhea may be present. Seropurulent or mucopurulent exudate may be seen in the middle meatus with maxillary, anterior ethmoid, or frontal sinusitis and in the area medial to the middle turbinate with posterior ethmoid or sphenoid sinusitis (see Fig. 86-1).

In acute and chronic sinusitis, the swollen mucous membrane and retained exudate cause the affected sinus to appear opaque on x-rays. CT provides better definition of the extent and degree of sinusitis. X-rays of the apices of the teeth may be required in chronic maxillary sinusitis to exclude a periapical abscess.

## Treatment

In acute sinusitis, improved drainage and control of infection are the aims of therapy. Steam inhalation effectively produces nasal vasoconstriction and promotes drainage. Saline nasal washes may promote drainage. Topical vasoconstrictors, such as phenylephrine 0.25% spray q 3 h, are effective but should be used for a maximum of 7 days; systemic vasoconstrictors, such as pseudoephedrine 30 mg po (for adults) q 4 to 6 h, are less effective.

In acute and chronic sinusitis, antibiotics should be given for at least 10 to 12 days. In acute sinusitis, penicillin V 250 mg po q 6 h is the initial antibiotic of choice, and erythromycin 250 mg po q 6 h is the second choice. In exacerbations of chronic sinusitis, a broad-spectrum antibiotic, such as ampicillin 250 or 500 mg or tetracycline 250 mg po q 6 h, is better. In chronic sinusitis, prolonged antibiotic therapy for 4 to 6 wk often results in complete resolution. The sensitivities of pathogens isolated from the sinus exudate and the patient's response guide subsequent therapy. Sinusitis not responsive to antibiotic therapy may require an operation (maxillary sinusotomy, ethmoidectomy, or sphenoid sinusotomy) to improve ventilation and drainage and to remove inspissated mucopurulent material, epithelial debris, and hypertrophic mucous membrane. These operations are usually performed intranasally with the aid of an endoscope (functional endoscopic sinus surgery). Chronic frontal sinusitis is managed with osteoplastic obliteration of the frontal sinuses but may be treated endoscopically in selected patients.

## Sinusitis in Metabolically or Immunologically Compromised Patients

In patients with poorly controlled diabetes or with immunodeficiency, aggressive and even fatal fungal or bacterial sinusitis can occur.

Mucormycosis (phycomycosis)--a mycosis due to fungi of the order Mucorales, including species of Mucor, Absidia, and Rhizopus--may develop in patients with poorly controlled diabetes. It is characterized by black, devitalized tissue in the nasal cavity and neurologic signs secondary to retrograde thromboarteritis in the carotid arterial system. Diagnosis is made by the histopathologic demonstration of mycelia in the avascularized tissue, and treatment requires control of the diabetes and IV administration of amphotericin B.

Aspergillosis and candidiasis of the paranasal sinuses may occur in a patient who is immunologically compromised as a result of therapy with cytotoxic drugs or the underlying disease process in leukemia, lymphoma, multiple myeloma, AIDS, or other immunosuppressive diseases. Aspergillosis is characterized by polypoid tissue in the nose and paranasal sinuses. Biopsy and culture of this tissue are required for diagnosis; aggressive paranasal sinus surgery and IV amphotericin B therapy are advocated as attempts to control these often fatal infections.

### Neoplasms

Unilateral bloody nasal discharge and obstruction, facial swelling, and numbness indicate cancer of the nose or paranasal sinuses until proved otherwise.

Exophytic papillomas are squamous cell papillomas that have a branching, vascular connective tissue stalk with fingerlike projections on the surface. In the nasal cavity, they often require repeated excision but have a benign course. Inverted papillomas are squamous cell papillomas in which the epithelium is invaginated into the vascular connective tissue stroma. They are invasive and act in a locally malignant manner; excision must involve a large margin of normal tissue, including the bone of the lateral wall of the nasal cavity, in a procedure called a lateral rhinotomy.

Fibromas, hemangiomas, and neurofibromas are benign tumors that occur in the nasal cavity. Fibromas, neurilemomas, and ossifying fibromas occur in the paranasal sinuses.

Squamous cell carcinoma is the most common malignant tumor in the nose and paranasal sinuses. Other tumors include adenoid cystic and mucoepidermoid carcinomas, malignant mixed tumors, adenocarcinomas, lymphomas, fibrosarcomas, osteosarcomas, chondrosarcomas, and melanomas. Hypernephroma is the most common metastatic tumor in the paranasal sinuses. Combined radiation therapy and radical resection result in the best survival rates for patients with primary neoplasms.

## Chapter 87. Pharynx

The pharynx, which can be divided into the nasopharynx, oropharynx, and hypopharynx, may be affected by inflammation, infection, and carcinoma. Disorders of the nasopharynx, located above the soft palate, include adenoid hypertrophy (see under Bacterial Infections in Ch. 265) and juvenile angiofibroma (see Ch. 272). In the oropharynx, located behind the mouth, the main structures to be examined are the palatine and lingual tonsils, tongue base, and posterior pharyngeal wall. Retropharyngeal abscess is discussed under Bacterial Infections in Ch. 265, and Zenker's (pharyngoesophageal) diverticulum under Esophageal Diverticula in Ch. 20.

## Tornwaldt's Cyst (Pharyngeal Bursa)

A rare cyst located in the midline of the nasopharynx that may become infected.

Tornwaldt's cyst is superficial to the superior constrictor muscle of the pharynx and is covered by the mucous membrane of the nasopharynx. If infected, it may cause persistent purulent drainage with a foul taste and odor, eustachian tube obstruction, and sore throat. Purulent exudate may be seen at the opening of the cyst. Treatment consists of marsupialization or excision.

## Pharyngitis

### Acute inflammation of the pharynx.

Although usually viral in origin, pharyngitis may be due to a group A [beta]-hemolytic streptococcus, Mycoplasma pneumoniae, Chlamydia pneumoniae, or other bacteria. It is characterized by sore throat and pain when swallowing. (Pharyngitis in gonorrhea and in other sexually transmitted diseases is discussed in Ch. 164.)

Differentiating viral from bacterial pharyngitis on the basis of physical examination alone is difficult. In both, the pharyngeal mucous membrane may be mildly injected or severely inflamed and may be covered by a membrane and a purulent exudate. Fever, cervical adenopathy, and leukocytosis are present in both viral and streptococcal pharyngitis but may be more marked in the latter.

### Treatment

Treatment includes acetaminophen to relieve discomfort and rest. In patients with clinical evidence suggesting bacterial infection, antibiotic therapy is usually administered while awaiting results of cultures for group A [beta]-hemolytic streptococcus. Penicillin V 250 mg po q 6 h for 10 days is indicated for group A streptococcal pharyngitis, primarily to prevent rheumatic fever. Alternatively, parenteral penicillin G benzathine, oral erythromycin, or a first-generation cephalosporin may be used.

## Tonsillitis

Acute inflammation of the palatine tonsils, usually due to streptococcal or, less commonly, to viral infection.

Epidemics of viral tonsillitis occur among military recruits. Tonsillitis is characterized by sore throat and pain, most marked when swallowing and often referred to the ears. Very young children may not complain of sore throat, but they refuse to eat. High fever, malaise, headache, and vomiting are common.

### Diagnosis

The tonsils are edematous and hyperemic. There may be a purulent exudate from the crypts and a membrane--white, thin, nonconfluent, and confined to the tonsil--that peels away

without bleeding. Differential diagnosis includes diphtheria, Vincent's angina (trench mouth), and infectious mononucleosis. In diphtheria, the membrane is dirty gray, thick, and tough; it bleeds if peeled away, and smear and culture show Corynebacterium diphtheriae. Vincent's angina, characterized by superficial, painful ulcers with erythematous borders, is caused by a fusiform bacillus and a spirochete that are demonstrable on smear. Inflamed tonsils in infectious mononucleosis are characteristically associated with micropetechiae of the soft palate; atypical lymphocytes on smear and a positive monospot test confirm the diagnosis of mononucleosis.

### Treatment

For viral tonsillitis, symptomatic therapy is the same as that for pharyngitis (see above). Penicillin V 250 mg po q 6 h or, for children < 6 yr, penicillin V 125 mg po q 8 h is the treatment of choice for streptococcal tonsillitis and should be continued for 10 days. If possible, another throat culture should be performed 5 to 6 days later. Throat cultures of family members should be performed initially so that carriers may be treated at the same time. Tonsillectomy should be considered if acute tonsillitis returns repeatedly after adequate treatment or if chronic tonsillitis and sore throat are relieved only temporarily by antibiotic therapy.

## Peritonsillar Cellulitis And Abscess

An acute infection located between the tonsil and the superior pharyngeal constrictor muscle.

Peritonsillar abscess (quinsy) is rare in children but more common in young adults. Although usually due to a group A [beta]-hemolytic streptococcus, peritonsillar infection can also be caused by anaerobic microorganisms, such as bacteroides. Swallowing causes severe pain; the patient is febrile and toxic, tilts the head toward the side of the abscess, and has marked trismus. The tonsil is displaced medially by the peritonsillar cellulitis and abscess, the soft palate is erythematous and swollen, and the uvula is edematous and displaced to the opposite side.

### Treatment

Cellulitis without pus formation responds to penicillin in 24 to 48 h. Initially, penicillin G 1 million U IV q 4 h is given. If pus is present and does not drain spontaneously, aspiration or incision and drainage are required. Antibiotic therapy with penicillin V 250 mg q 6 h should be continued orally for 12 days unless cultures and sensitivity studies indicate that another antibiotic is preferable. Peritonsillar abscesses may recur, and tonsillectomy may be considered, especially for patients with recurrent abscesses. It is usually performed 6 wk after the acute infection has subsided, but if antibiotic therapy is given, it can be performed during the acute infection.

## Lemierre's Syndrome

Lemierre's syndrome is a rare suppurative infection of the lateral pharyngeal space accompanied by septic jugular thrombophlebitis. Occasionally, infection spreads, primarily to

the lungs and joints, sometimes with abscess formation. The usual cause is Fusobacterium necrophorum, but Streptococcus, Bacteroides, Peptostreptococcus, and Eikenella are also causes. Symptoms develop about 1 wk after acute pharyngitis. Diagnosis requires cultures and either ultrasound or MRI of the neck. Treatment is with IV antibiotics such as ticarcillin/clavulanate or ampicillin/sulbactam, and surgical drainage of any abscesses. Excision of the internal jugular vein may be necessary. Use of anticoagulants is controversial.

### Parapharyngeal Abscess

Suppuration of a parapharyngeal lymph node with consequent abscess formation is usually secondary to pharyngitis or tonsillitis and may occur at any age. The abscess is lateral to the superior pharyngeal constrictor muscle and close to the carotid sheath. Pharyngeal inflammation may not be apparent. The anterior cervical triangle is markedly swollen.

Initially, children should be given penicillin G 150,000 U/kg/day IV in four equal doses, and adults penicillin V 500 mg po q 6 h; the abscess should be drained through a cervical, not pharyngeal, incision. Subsequently, penicillin V (250 mg for children; 500 mg for adults) is given orally q 6 h to complete 12 days of therapy.

### Velopharyngeal Insufficiency

Incomplete closure of the velopharyngeal sphincter between the oropharynx and the nasopharynx, resulting in impaired speech and deglutition.

Speech is characterized by nasal emission of air and weak oral plosive and fricative articulation. Closure, normally achieved by the sphincteric action of the soft palate and the superior constrictor muscle, is impaired in patients with cleft palates, repaired cleft palates, congenitally short palates, submucous cleft palates, and palatal paralysis.

### Diagnosis and Treatment

Regurgitation of solid foods and fluids through the nose denotes gross velopharyngeal insufficiency, but normal speech is a more exacting criterion of competency. Inspection of the palate during phonation may reveal palatal paralysis. Palpation of the midline of the soft palate and transillumination with a flexible nasopharyngolaryngoscope may demonstrate a submucous cleft. A lateral x-ray may show a congenitally short palate or an unusually large nasopharynx and, if taken during phonation, indicates the degree of insufficiency; cinefluoroscopy during connected speech verifies the inability to maintain velopharyngeal competence.

Treatment consists of speech therapy and surgical correction by a palatal push-back procedure, pharyngeal flap, or pharyngoplasty.

### Nasopharyngeal Squamous Cell Carcinoma

Squamous cell carcinoma of the nasopharynx occurs in children and young adults. Rare in North America, it is one of the most common cancers among persons of Chinese origin, especially in southern China and Southeast Asia. Cancer of the nasopharynx is

prevalent in Chinese immigrants to North America and slightly less prevalent in first-generation Chinese Americans. The first symptom is often nasal or eustachian tube obstruction; the latter may result in middle ear effusion. Purulent bloody rhinorrhea, frank epistaxis, cranial nerve paralysis due to invasion of the parapharyngeal space and cranial cavity by the tumor, and cervical lymphadenopathy resulting from metastasis are common presenting complaints.

### Diagnosis and Treatment

Diagnosis is made by biopsy of the primary nasopharyngeal tumor. Biopsy of the neck metastasis should not be performed until the nasopharynx has been inspected and palpated and biopsy of any suspicious lesion there has been performed.

Treatment of the primary tumor is radiation therapy. Radical neck dissection is required for large (> 2 cm at the greatest dimension) or persistent neck masses. The overall 5-yr survival rate is 35%.

### Squamous Cell Carcinoma Of The Tonsil

Squamous cell carcinoma of the tonsil, second in frequency only to carcinoma of the larynx among malignancies of the upper respiratory tract, occurs predominantly in males and is associated with tobacco smoking and ethanol ingestion. Sore throat is the most common presenting complaint, and pain often radiates to the ear on the same side. A metastatic mass in the neck may be the first symptom.

### Diagnosis and Treatment

Diagnosis is made by biopsy. Direct laryngoscopy, bronchoscopy, and esophagoscopy are performed to exclude a synchronous second primary neoplasm. Treatment combines radiation therapy and surgery, which consists of radical resection of the tonsillar fossa, sometimes with partial mandibulectomy and radical neck dissection. The 5-yr survival rate is about 50%.

## Chapter 88. Larynx

(For acute laryngotracheobronchitis, see Croup under Viral Infections in Ch. 265.)

Examination of the larynx includes inspection of the epiglottis, false cords, true cords, arytenoids, pyriform sinuses (lateral to the larynx), and subglottic region below the cords. The voice should be assessed: If any surgical procedures are to be performed, the voice should be recorded. The mobility of the vocal cords should be noted.

### Vocal Cord Polyps

Chronic edema in the lamina propria of the true vocal cords.

These lesions are caused by voice abuse, chronic laryngeal allergic reactions, and chronic inhalation of irritants, such as industrial fumes and cigarette smoke. They result in

hoarseness and a breathy voice. Biopsy of discrete lesions should be performed by microlaryngoscopy to exclude carcinoma.

Treatment involves surgical removal of the polyp at direct microlaryngoscopy to restore the voice and correction of the underlying cause to prevent recurrence, including voice therapy for voice abuse.

In microlaryngoscopy, an operating microscope is used to examine, biopsy, and perform surgical procedures on the larynx. Patients are anesthetized, and the airway is secured, eg, by high-pressure jet ventilation through the laryngoscope, endotracheal intubation, or, for an inadequate upper airway, tracheotomy. Because the microscope allows observation at different magnifications, tissue can be removed precisely and accurately, minimizing damage to the larynx, which could otherwise result in permanent vocal impairment. Laser can be delivered through the optical system of the microscope to make precise cuts. Three-dimensional images can be recorded with attached cameras. Microlaryngoscopy is preferred for almost all laryngeal biopsies, for procedures involving benign tumors, and for many forms of phonosurgery.

### Vocal Cord Nodules (Singer's Nodules)

Condensations of hyaline connective tissue in the lamina propria at the junction of the anterior 1/3 and posterior 2/3 of the free edges of the true vocal cords.

Vocal cord nodules are caused by chronic voice abuse, such as yelling, shouting, or using an unnaturally low frequency. Hoarseness and a breathy voice result. Carcinoma should be excluded by biopsy.

Treatment for nodules that do not resolve with voice therapy involves surgical removal of the nodules at direct laryngoscopy and correction of the underlying voice abuse. Vocal nodules in children usually regress with voice therapy alone.

### Contact Ulcers

Unilateral or bilateral ulcers of the mucous membrane over the vocal process of the arytenoid cartilage.

Contact ulcers are usually due to voice abuse in the form of a sharp glottal attack (abrupt rise in intensity at the onset of phonation). Reflux of gastric contents may also cause contact ulcers. Mild pain with phonation and swallowing and varying degrees of hoarseness result. Biopsy to exclude carcinoma is important. Prolonged ulceration leads to nonspecific granulomas that also produce varying degrees of hoarseness.

Treatment consists of prolonged voice rest (6 wk minimum) to heal the ulcers. Patients must recognize the limitations of their voices and learn to adjust their vocal activities to avoid recurrence. Granulomas tend to recur after surgical removal but respond to voice therapy. Gastroesophageal reflux must be treated vigorously (see Gastroesophageal Reflux Disease in Ch. 20).

## Laryngitis

### Inflammation of the larynx.

The most common cause of acute laryngitis is a viral URI. Laryngitis may also occur in bronchitis, pneumonia, influenza, pertussis, measles, and diphtheria. Excessive use of the voice, allergic reactions, and inhaling irritating substances, such as cigarette smoke, can cause acute or chronic laryngitis.

An unnatural change of voice is usually the most prominent symptom. Hoarseness and even aphonia, with a sensation of tickling, rawness, and a constant urge to clear the throat, may occur. Symptoms vary with the severity of the inflammation. Fever, malaise, dysphagia, and throat pain may occur in more severe infections; laryngeal edema, if present, may cause dyspnea. Indirect laryngoscopy discloses mild to marked erythema of the mucous membrane, which may also be edematous. If a membrane is present, diphtheria must be suspected (see Diphtheria under Bacterial Infections in Ch. 265).

### Treatment

There is no specific treatment for viral laryngitis. Voice rest and steam inhalations relieve symptoms and promote resolution of acute laryngitis. Treatment of acute or chronic bronchitis may relieve laryngitis. Chronic bronchitis may require a broad-spectrum antibiotic, such as ampicillin 250 or 500 mg or tetracycline 250 mg po q 6 h for 10 to 14 days. Because of the increasing prevalence of antibiotic-resistant bacteria, continuing antibiotic therapy may be chosen on the basis of cultures and sensitivity studies.

## Vocal Cord Paralysis

Vocal cord paralysis may result from lesions at the nucleus ambiguus, its supranuclear tracts, the main trunk of the vagus, or the recurrent laryngeal nerves. Intracranial neoplasms, vascular accidents, and demyelinating diseases cause nucleus ambiguus paralysis. Neoplasms at the base of the skull and trauma of the neck cause vagus paralysis. Recurrent laryngeal paralysis is caused by neck or thoracic lesions (eg, aortic aneurysm; mitral stenosis; neoplasms of the thyroid gland, esophagus, lung, or mediastinal structures), trauma, thyroidectomy, neurotoxins (eg, lead), neurotoxic infections (eg, diphtheria), cervical spine injury or surgery, or viral illness. Viral neuronitis probably accounts for most cases of idiopathic vocal cord paralysis.

### Symptoms and Signs

Vocal cord paralysis results in loss of vocal cord abduction or adduction. It may affect phonation, respiration, and deglutition, and food and fluids may be aspirated into the trachea. The paralyzed cord generally lies 2 to 3 mm lateral to the midline. In recurrent laryngeal nerve paralysis, it may move with phonation but not with inspiration. In unilateral vocal cord paralysis, the airway is usually not obstructed because the normal cord abducts sufficiently; the voice is hoarse and breathy. In bilateral vocal cord paralysis, both cords lie generally within 2 to 3 mm of the midline, and the voice is of limited intensity but of good quality. The airway, however, is inadequate, resulting in stridor and dyspnea with moderate exertion.

## Diagnosis and Treatment

The cause must always be sought. Evaluation may include laryngoscopy, bronchoscopy, and esophagoscopy as well as a neurologic examination; enhanced CT of the head, neck, and chest; a thyroid gland scan; and an upper GI series. Cricoarytenoid arthritis, which may cause fixation of the cricoarytenoid joint, must be differentiated.

In unilateral paralysis, augmenting the paralyzed cord by injecting a Teflon suspension may bring the cords closer together to improve the voice and to prevent aspiration. Maintaining an adequate airway is the problem in bilateral paralysis. Tracheostomy may be needed permanently or during a URI. An arytenoidectomy with lateralization of the true vocal cord opens the glottis and improves the airway but may alter voice quality. Laryngoplasty consists of an external incision in the thyroid cartilage and insertion of material to move the vocal cord medially.

## Laryngoceles

Evaginations of the mucous membrane of the laryngeal ventricle.

Internal laryngoceles displace and enlarge the false vocal cords, resulting in hoarseness and airway obstruction. External laryngoceles extend through the thyrohyoid membrane, producing a mass in the neck. Laryngoceles, which are filled with air, can be expanded by the Valsalva maneuver and tend to occur in musicians who play wind instruments. They appear on CT as smooth, ovoid, low-density masses. Laryngoceles may become infected (laryngopyocele) or filled with mucoid fluid. Treatment is excision.

## Benign Neoplasms

Benign laryngeal neoplasms include juvenile papillomas (see Ch. 272), hemangiomas, fibromas, chondromas, myxomas, and neurofibromas. They may affect any part of the larynx. Removal restores the voice, the functional integrity of the laryngeal sphincter, and the airway.

## Malignant Neoplasms

Squamous cell carcinoma is the most common malignancy of the larynx and of the head and neck. The incidence is higher in males. It is associated with cigarette smoking and ethanol consumption. The true vocal cords (particularly the anterior portion), epiglottis, pyriform sinus, and postcricoid area are common sites of origin. Cordal or glottic carcinoma produces hoarseness early, and all patients with hoarseness lasting > 2 wk should be examined by indirect laryngoscopy. Biopsy of a discrete lesion of the laryngeal mucous membrane should be performed during direct laryngoscopy. Carcinoma of the supraglottic larynx (epiglottis), hypopharyngeal carcinoma (pyriform sinus), and postcricoid carcinoma cause pain and difficulty when swallowing. In the first two forms, a metastatic mass in the neck may be the first symptom. Angiogenesis is correlated with regional recurrence.

Verrucous carcinoma, a rare variant of squamous cell carcinoma, usually arises in the glottic area. The diagnosis may require multiple biopsies.

## Treatment

For early glottic carcinoma, radiation therapy or cordectomy results in a 5-yr survival rate of 85 to 95%. For early cordal carcinoma, radiation therapy is often preferred, because it usually results in a normal voice. For advanced carcinoma with anterior commissure involvement, impaired vocal cord mobility, thyroid cartilage invasion, or subglottic extension, surgery is necessary. A hemilaryngectomy, which preserves laryngeal phonation and sphincteric functions, is often possible with lesions limited to one vocal cord. More advanced glottic carcinoma requires total laryngectomy.

Early supraglottic carcinoma can be effectively treated with radiation therapy. If the carcinoma is more advanced but does not affect the true vocal cords, a supraglottic partial laryngectomy can be performed to preserve the voice and glottic sphincter. If the true vocal cords are affected, a total laryngectomy is required. Early hypopharyngeal carcinoma may be managed by an extended partial laryngectomy; more advanced lesions require a total laryngectomy. In advanced supraglottic and hypopharyngeal carcinoma, a combination of radiation therapy and surgery is more successful than surgery alone. Postcricoid carcinoma requires a total laryngopharyngectomy and replacement of the hypopharynx and cervical esophagus with a free jejunal graft with microvascular anastomoses. For metastasis to the cervical lymph nodes, laryngeal surgery is combined with radical or modified radical neck dissection. Verrucous carcinoma is treated surgically.

Rehabilitation after total laryngectomy requires developing a new voice using esophageal speech, a tracheoesophageal fistula, or an electrolarynx. Esophageal speech involves taking air into the esophagus during inspiration and gradually eructating the air through the pharyngoesophageal junction to produce a sound. A tracheoesophageal fistula, created by inserting a one-way valve between the trachea and the esophagus, forces air into the esophagus during expiration to produce a sound. If the valve misfunctions, fluids and food may be aspirated into the tracheobronchial tree. An electrolarynx is a sound source that must be held against the neck while it produces sound. In all three techniques, sound is articulated into speech by the pharynx, palate, tongue, teeth, and lips.

# Chapter 89. Neoplasms Of The Head And Neck

Principles of head and neck neoplasms are presented in general terms, and many specific exceptions to these statements must be acknowledged. Neoplasms of specific organs are discussed elsewhere in The Manual.

## Epidemiology

Excluding the skin and thyroid gland, > 90% of head and neck cancers are squamous cell (epidermoid) carcinomas; 5% are melanomas, lymphomas, and sarcomas. The average age of patients with head and neck cancers is 59 yr; those with sarcomas or carcinomas of the salivary glands, thyroid gland, or paranasal sinuses are usually < 59 yr; those with squamous cell carcinoma of the oral cavity, pharynx, or larynx are generally > 59 yr.

## Etiology and Pathogenesis

The most common cancer of the upper respiratory and alimentary tracts is squamous cell carcinoma of the larynx, followed by squamous cell carcinoma of the palatine tonsil and hypopharynx. About 85% of patients with cancer of the head and neck have a history of ethanol or tobacco consumption. Oral cavity cancer may also result from poor oral hygiene, ill-fitting dental appliances, and use of snuff or chewing tobacco; in India, chewing betel nut is a major cause.

The Epstein-Barr virus plays a role in the pathogenesis of nasopharyngeal cancer. Patients who were treated with small doses of radiation >= 25 yr ago (for acne, excess facial hair, an enlarged thymus, or hypertrophic tonsils and adenoids) are predisposed to develop thyroid and salivary gland cancer.

Head and neck cancers usually remain localized to the head and neck for months to years. Local tissue invasion is followed by metastasis to regional lymph nodes. Distant lymphatic metastases tend to occur late. Hematogenous metastases are usually associated with large or persistent tumors and occur more commonly in immunocompromised patients.

## Clinical Staging and Prognosis

Head and neck cancers are traditionally classified clinically according to size and site of the primary neoplasm (T), number and size of metastases to the cervical lymph nodes (N), and evidence of distant metastases (M); several stages are described. Stage I: The primary neoplasm is <= 2 cm at greatest dimension or localized to one anatomic site without regional or distant metastasis (T1N0M0). Stage II: The primary neoplasm measures 2 to 4 cm at greatest dimension or affects two areas within a specific site (eg, larynx) without regional or distant metastasis (T2N0M0). Stage III: The primary neoplasm is > 4 cm at greatest dimension or affects three adjacent areas in a specific head and neck site and/or has an isolated neck metastasis of <= 3 cm at greatest dimension (T3N0M0 or T1-3N1M0). Stage IV: The cancer is massive, invades bone and cartilage, and/or extends outside of its site of origin into another site (eg, oral cavity into oropharynx). The neck metastasis measures > 3 cm; it affects multiple ipsilateral, contralateral, or bilateral lymph nodes or is fixed to surrounding tissue; and/or there is evidence of distant metastases (T1-4N1-3M0-1). Clinical staging is usually supplemented by radiologic staging using CT and/or MRI.

Exophytic or verrucous tumors generally respond to treatment better than do infiltrative, ulcerative, or indurated lesions. Cervical or distant metastasis is associated with limited survival. The more poorly differentiated the cancer, generally the greater the chance of regional and distant metastases. With invasion of muscle, bone, or cartilage, cure rates are lower. Perineural spread as evidenced by pain, paralysis, or numbness indicates a highly aggressive neoplasm likely to persist.

With appropriate treatment, the survival rate generally approaches 90% for stage I, 75% for stage II, 45 to 75% for stage III, and < 35% for stage IV. The overall 5-yr survival rate is 65% for all patients with local stage II or III squamous cell carcinoma of the head and neck. The rate drops to <= 30% for patients with metastasis to lymph nodes. Patients > 70 yr often have longer disease-free intervals and better survival rates than do younger patients.

## Treatment

Many stage I neoplasms, regardless of location in the upper respiratory or alimentary tract, respond similarly to surgery and to radiation therapy; other factors may determine the choice of therapy. If radiation therapy is chosen for primary therapy, it is delivered to the primary site and, if the probability of regional nonpalpable metastasis is > 20%, also bilaterally to the cervical lymph nodes. The expected 5-yr cure rate is 90%. In some cases, surgical procedures are needed to achieve the 90% cure rate. Lesions > 2 cm or with bone or cartilage invasion (with or without regional neck metastasis) require surgical resection of the primary site and possibly resection of regional lymph nodes. If lymph node metastases are found or deemed very likely to occur, postoperative radiation to the primary site and bilaterally to any remaining cervical lymph nodes is generally recommended. As an alternative to surgery, radiation therapy--with or without chemotherapy--may be chosen. If the cancer recurs, the patient may have recourse to surgery.

In advanced (most stage II and all stages III and IV) squamous cell carcinoma, a combination of surgery and radiation therapy offers a better chance of cure than does treatment with either alone. Surgery is more effective than radiation therapy and/or chemotherapy in controlling large primary cancers, whereas radiation is more effective in controlling the periphery of the primary lesion and microscopic or nonpalpable metastases. Radiation therapy may be given preoperatively or postoperatively, but the latter is usually preferred.

The aim of chemotherapy is to kill tumor cells at the local site, in regional lymph nodes, and in distant metastases. Whether adjuvant chemotherapy (combined with surgery or radiation therapy) increases the cure rate is not known; however, combined therapy prolongs the cancer-free interval. Several drugs--cisplatin, fluorouracil, bleomycin, and methotrexate--provide palliation for pain and shrink the neoplasm in patients who cannot be treated with surgery or radiation therapy.

If the cancer is excised after chemotherapy or radiation therapy, the surgeon must remove the tissues that were affected by the cancer before nonsurgical therapy was started.

Adverse effects of treatment: Surgery requires rehabilitation for swallowing and speaking. Reconstruction procedures, including grafts, regional pedicle flaps, and complex free flaps, are used to facilitate the restoration of function. Radiation produces skin changes, fibrosis, ageusia, xerostomia, and, rarely, osteoradionecrosis. Toxic effects of chemotherapy include severe nausea and vomiting, transient hair loss, gastroenteritis, and hematopoietic and immune depression.

Persisting cancer: A palpable mass or ulcerated lesion with edema or pain at the primary site after therapy strongly suggests a persistent tumor. Such a tumor is more difficult to detect after radiation therapy or chemotherapy than after surgery alone, but it is usually more difficult to eradicate after surgery alone than after radiation therapy and/or chemotherapy. Gallium scanning, CT with enhancement, and MRI can sometimes detect tumors that are persistent or >= 2 cm.

For adequate local control after surgical failure, all scar planes and reconstructive flaps must be excised in addition to the cancer. Radiation and/or chemotherapy given after surgical failure is much less effective than that given before or immediately after surgery.

Terminal care of persons with incurable head and neck cancers is not easy. Pain, difficulty in eating, choking on secretions, and other problems make adequate symptomatic treatment essential. Patient directives regarding terminal care should be clarified early (see Ch. 293).

## Cervical Metastases

A palpable mass in the neck may result from infectious, inflammatory, congenital, traumatic, or neoplastic processes. Neoplasms include metastasis of carcinoma from the upper respiratory or upper alimentary tract to a lymph node; lymphoma; metastasis of thyroid or salivary gland carcinoma; and metastasis from a distant primary site, such as the lung, prostate, breast, stomach, colon, or kidney. About 60% of supraclavicular triangle masses are metastases from distant primary sites. Elsewhere in the neck, for 80% of patients with cancerous cervical adenopathy, the primary carcinoma is found in the upper respiratory or alimentary tract. Likely sites are the nasopharynx, palatine tonsil, base of tongue, laryngeal surface of the epiglottis, and hypopharynx, including the pyriform sinuses.

## Diagnosis and Treatment

Evaluation of a patient with a neck mass should include inspection of the scalp, ears, nasal cavities, nasopharynx, oropharynx, hypopharynx, and larynx as well as palpation of the palatine tonsils, base of the tongue, and thyroid and salivary glands. An upper GI series, a thyroid scan, and CT of the head, neck, and chest may be required. Direct laryngoscopy, bronchoscopy, and esophagoscopy with biopsy of suspicious areas are indicated. When a primary neoplasm is not identified, random biopsy of the nasopharynx, palatine tonsils, and base of the tongue should be considered. If a primary site is not found, the mass may be aspirated with a fine needle for cytologic evaluation, and if necessary, a biopsy should be performed. An excisional biopsy, if possible, is preferable to an incisional biopsy because it does not leave a transected mass in the neck. The excisional biopsy should be performed so that the biopsy site can be excised if a malignancy is found and further regional surgery is required.

Squamous cell carcinoma with cervical metastases from an unknown primary site is treated with radiation therapy to the nasopharynx, palatine tonsils, base of tongue, and both sides of the neck, followed by radical neck dissection if the cervical mass was >= 2 cm at greatest dimension when radiation therapy began or if the mass persists after radiation therapy.