5 - Ear, Nose, Throat, and Dental Disorders

Chapter 46. Approach to the Patient With Ear Problems

Introduction

Earache, hearing loss, otorrhea, tinnitus, and vertigo are the principal symptoms of ear problems. Hearing loss is discussed in Ch. 47.

In addition to the ears, nose, nasopharynx, and paranasal sinuses, the teeth, tongue, tonsils, hypopharynx, larynx, salivary glands, and temporomandibular joint are examined; pain and discomfort may be referred from them to the ears. It is important to examine cranial nerve function (see pp. 1587 and 1745) and to perform tests of hearing (see p. 431) and of the vestibular apparatus. The patient is also examined for nystagmus (a rhythmic movement of the eyes—see Sidebar 46-1).

Testing

Patients with abnormal hearing on history or physical examination or with tinnitus or vertigo undergo an audiogram (see p. 433). Patients with nystagmus or altered vestibular function may benefit from computerized electronystagmography (ENG), which quantifies spontaneous, gaze, or positional nystagmus that might not be visually detectable. Computerized ENG caloric testing quantifies the strength of response of the vestibular system to cool and warm irrigations in each ear, enabling the physician to discriminate unilateral weakness. Different components of the vestibular system can be tested by varying head and body position or by presenting visual stimuli.

Sidebar 46-1 Nystagmus

Nystagmus is a rhythmic movement of the eyes that can have various causes. Vestibular disorders can result in nystagmus because the vestibular system and the oculomotor nuclei are interconnected. The presence of vestibular nystagmus helps identify vestibular disorders and sometimes distinguishes central from peripheral vertigo. Vestibular nystagmus has a slow component caused by the vestibular input and a quick, corrective component that causes movement in the opposite direction. The direction of the nystagmus is defined by the direction of the quick component because it is easier to see. Nystagmus may be rotary, vertical, or horizontal and may occur spontaneously, with gaze, or with head motion.

Initial inspection for nystagmus is done with the patient lying supine with unfocused gaze (+30 diopter or Frenzel lenses can be used to prevent gaze fixation). The patient is then slowly rotated to a left and then to a right lateral position. The direction and duration of nystagmus are noted. If nystagmus is not detected, the Dix-Hallpike (or Barany) maneuver is done. In this maneuver, the patient sits erect on a stretcher so that when lying back, the head extends beyond the end. With support, the patient is rapidly lowered to horizontal, and the head is extended back 45° below horizontal and rotated 45° to the left. Direction and duration of nystagmus and development of vertigo are noted. The patient is returned to an upright position, and the maneuver is repeated with rotation to the right. Any position or maneuver that causes nystagmus should be repeated to see whether it fatigues.

Nystagmus secondary to peripheral nervous system disorders has a latency period of 3 to 10 sec and fatigues rapidly, whereas nystagmus secondary to CNS has no latency period and does not fatigue. During induced nystagmus, the patient is instructed to focus on an object. Nystagmus caused by peripheral disorders is inhibited by visual fixation. Because Frenzel lenses prevent visual fixation, they must be removed to assess visual fixation.

Caloric stimulation of the ear canal induces nystagmus in a person with an intact vestibular system. Failure to induce nystagmus or > 20% difference in duration between sides suggests a lesion on the side of the decreased response. Quantification of caloric response is best done with formal (computerized) electronystagmography.

Ability of the vestibular system to respond to peripheral stimulation can be assessed at the bedside. Care

should be taken not to irrigate an ear with a known tympanic membrane perforation or chronic infection. With the patient supine and the head elevated 30°, each ear is irrigated sequentially with 3 mL of ice water. Alternatively, 240 mL of warm water (40 to 44°C) may be used, taking care not to burn the patient with overly hot water. Cold water causes nystagmus to the opposite side; warm water causes nystagmus to the same side. A mnemonic device is COWS (*C*old to the *O*pposite and *W*arm to the *S*ame).

Primary imaging tests include CT of the temporal bone with or without radiopaque dye and gadolinium-enhanced MRI of the brain, with attention paid to the internal auditory canals to rule out an acoustic neuroma. These tests may be indicated in cases of trauma to the ear, head, or both; chronic infection; hearing loss; vertigo; facial paralysis; and otalgia of obscure origin.

Earache

(Otalgia)

Earache may occur in isolation or along with discharge or, rarely, hearing loss.

Pathophysiology

Pain may come from a process within the ear itself or may be referred to the ear from a nearby nonotologic disorder.

Pain from the ear itself may result from a pressure gradient between the middle ear and outside air, from local inflammation, or both. A middle ear pressure gradient usually involves eustachian tube obstruction, which inhibits equilibration between middle ear pressure and atmospheric pressure and also allows fluid to accumulate in the middle ear. Otitis media causes painful inflammation of the tympanic membrane (TM) as well as pain from increased middle ear pressure (causing bulging of the TM).

Referred pain can result from disorders in areas innervated by cranial nerves responsible for sensation in the external and middle ear (5th, 9th, and 10th). Specific areas include the nose, paranasal sinuses, nasopharynx, teeth, gingiva, temporomandibular joint (TMJ), mandible, parotid glands, tongue, palatine tonsils, pharynx, larynx, trachea, and esophagus. Disorders in these areas sometimes also obstruct the eustachian tube, causing pain from a middle ear pressure gradient.

Etiology

Earache results from otologic causes (involving the middle ear or external ear) or from nonotologic causes referred to the ear from nearby disease processes (see Table 46-1).

With **acute pain**, the most common causes are

• Middle ear infection

• External ear infection

With **chronic pain** (> 2 to 3 wk), the most common causes are

• TMJ dysfunction

• Chronic eustachian tube dysfunction

• Chronic otitis externa

Also with chronic pain, a tumor must be considered, particularly in elderly patients and if the pain is associated with ear drainage. People with diabetes or in other immunocompromised states may develop a particularly severe form of external otitis termed malignant or necrotizing external otitis. In this situation, if

abnormal soft tissue is found on examination of the ear canal, the tissue must be biopsied to rule out cancer.

TMJ dysfunction is a common cause of earache in patients with a normal ear examination.

Evaluation

History: History of present illness should assess the location, duration, and severity of pain and whether it is constant or intermittent. If intermittent, it is important to determine whether it is random or occurs mainly with swallowing or jaw movement. Important associated symptoms include ear drainage, hearing loss, and sore throat. The patient should be asked about any attempts at cleaning the ear canal (eg, with cotton swab) or other recent instrumentation, foreign bodies, recent air travel or scuba diving, and swimming or other recurrent water exposure to ears.

Review of systems should ask about symptoms of chronic illness, such as weight loss and fevers.

Past medical history should ask about known diabetes or other immunocompromised state, previous ear disorders (particularly infections), and amount and duration of tobacco and alcohol use.

Physical examination: Vital signs should be checked for fever.

Examination focuses on the ears, nose, and throat.

The pinna and area over the mastoid process should be inspected for redness and swelling. The ear canal should be examined for redness, discharge, cerumen or foreign body, and any other lesions. The TM should be examined for redness, perforation, and signs of middle ear fluid collection (eg, bulging, distortion, change in normal light reflex). A brief bedside test of hearing (see p. 431) should be conducted.

The throat should be examined for erythema, tonsillar exudate, peritonsillar swelling, and any mucosal lesions suggesting cancer.

TMJ function should be assessed by palpation of the joints on opening and closing of the mouth, and notation should be made of trismus or evidence of bruxism.

The neck should be palpated for lymphadenopathy. In-office fiberoptic examination of the pharynx and larynx should be considered, particularly if no cause for the pain is identified on routine examination and if nonotologic symptoms such as hoarseness, difficulty swallowing, or nasal obstruction are reported.

Red flags: The following findings are of particular concern:

• Diabetes or immunocompromised state

• Redness and fluctuance over mastoid and protrusion of auricle

• Severe swelling at external auditory canal meatus

• Chronic pain, especially if associated with other head/neck symptoms

Interpretation of findings: An important differentiator is whether the ear examination

[**Table 46-1.** Some Causes of Earache]

is normal; middle and external ear disorders cause abnormal physical findings, which, when combined with history, usually suggest an etiology (see Table 46-1). For example, those with chronic eustachian tube dysfunction have abnormalities of the TM, typically a retraction pocket.

Those with a normal ear examination may have a visible oropharyngeal cause, such as tonsillitis or peritonsillar abscess. Ear pain due to neuralgia has a classic manifestation as brief (usually seconds,

always < 2 min) episodes of extremely severe, sharp pain. Chronic ear pain without abnormality on ear examination might be due to a TMJ disorder, but patients should have a thorough head and neck examination (including fiberoptic examination) to rule out cancer.

Testing: Most cases are clear after history and physical examination. Depending on clinical findings, nonotologic causes may require testing (see Table 46-1). Those with a normal ear examination, particularly with chronic or recurrent pain, may warrant evaluation with an MRI to rule out cancer.

Treatment

Underlying disorders are treated.

Pain is treated with oral analgesics; usually an NSAID or acetaminophen is adequate, but sometimes a brief course of an oral opioid is necessary, particularly for cases of severe otitis externa. In cases of severe otitis externa, effective treatment requires suction of debris from the ear canal and insertion of a wick to allow for delivery of antibiotic ear drops to the infected tissue. Topical analgesics (eg, antipyrine-benzocaine combinations) are generally not very effective but can be used on a limited basis.

Patients should be instructed to avoid digging in their ears with any objects (no matter how soft the objects are or how careful the patient claims to be). Also, patients should not irrigate their ears unless instructed by a physician to do so, and then only gently. An oral irrigator should never be used to irrigate the ear.

Key Points

• Most cases are due to infection of the middle or external ear.

• History and physical examination are usually adequate for diagnosis.

• Nonotologic causes should be considered when ear examination is normal.

Otorrhea

Ear discharge (otorrhea) is drainage exiting the ear. It may be serous, serosanguineous, or purulent. Associated symptoms may include ear pain, fever, pruritus, vertigo, tinnitus, and hearing loss.

Etiology

Causes may originate from the ear canal, the middle ear, or the cranial vault. Certain causes tend to manifest acutely because of the severity of their symptoms or associated conditions. Others usually have a more indolent, chronic course but sometimes manifest acutely (see Table 46-2).

Overall, the most common causes are

• Acute otitis media with perforation

• Chronic otitis media (with a perforation of the eardrum, cholesteatoma, or both)

• Otitis externa

The most serious causes are necrotizing external otitis and cancer of the ear.

Evaluation

History: History of present illness should cover duration of symptoms and whether symptoms have been recurrent. Important associated symptoms include pain, itching, decreased hearing, vertigo, and tinnitus. Patients are questioned about activities that can affect the canal or tympanic membrane (TM

—eg, swimming; insertion of objects, including cotton swabs; use of ear drops). Head trauma sufficient to cause a CSF leak is readily apparent.

Review of systems should seek symptoms of cranial nerve deficit and systemic symptoms suggesting Wegener's granulomatosis (eg, nasal discharge, cough, joint pains).

Past medical history should note any previous known ear disorders, ear surgery (particularly tympanostomy tube placement), and diabetes or immunodeficiency.

Physical examination: Examination begins with a review of vital signs for fever.

Ear and surrounding tissues (particularly the area over the mastoid) are inspected for erythema and edema. The pinna is pulled and the tragus is pushed gently to see whether pain is worsened. The ear canal is inspected with an otoscope; the character of discharge and presence of canal lesions, granulation tissue, or foreign body are noted. Edema and discharge may block visualization of all but the distal canal (irrigation should not be used in case there is a TM perforation), but when

[**Table 46-2.** Some Causes of Ear Discharge]

possible, the TM is inspected for inflammation, perforation, distortion, and signs of cholesteatoma (eg, canal debris, polypoid mass from TM).

When the ear canal is severely swollen at the meatus (eg, as with severe otitis externa) or there is copious drainage, careful suctioning can permit an adequate examination and also allow treatment (eg, application of drops, with or without a wick).

The cranial nerves are tested. The nasal mucosa is examined for raised, granular lesions, and the skin is inspected for vasculitic lesions, both of which may suggest Wegener's granulomatosis.

Red flags: The following findings are of particular concern:

• Recent major head trauma

• Any cranial nerve dysfunction (including sensorineural hearing loss)

• Fever

• Erythema of ear or periauricular tissue

• Diabetes or immunodeficiency

Interpretation of findings: Otoscopic examination can usually diagnose perforated TM, external otitis media, foreign body, or other uncomplicated sources of otorrhea. Some findings are highly suggestive (see Table 46-2). Other findings are less specific but indicate a more serious problem that involves more than a localized external ear or middle ear disorder:

• Vertigo and tinnitus (disorder of the inner ear)

• Cranial nerve deficits (disorder involving the skull base)

• Erythema and tenderness of ear, surrounding tissues, or both (significant infection)

Testing: Many cases are clear after clinical evaluation.

If CSF leakage is in question, discharge can be tested for glucose or β_2-transferrin; these substances are present in CSF but not in other types of discharge.

Patients without an obvious etiology on examination require audiogram and CT of the temporal bone or

gadolinium-enhanced MRI. Biopsy should be considered when auditory canal granulation tissue is present.

Treatment

Treatment is directed at the cause. Most physicians do not treat a suspected CSF leak with antibiotics without a definitive diagnosis because drugs might mask the onset of meningitis.

Key Points

- Acute discharge in a patient without chronic ear problems or immunodeficiency is likely the result of otitis externa or perforated otitis media.
- Severe otitis externa may require specialty referral for more extensive cleaning and possible wick placement.
- Those with recurrent ear symptoms (diagnosed or undiagnosed), cranial nerve findings, or systemic symptoms should have specialty referral.

Tinnitus

Tinnitus is a noise in the ears. It is experienced by 10 to 15% of the population.

Subjective tinnitus is perception of sound in the absence of an acoustic stimulus and is heard only by the patient. Most tinnitus is subjective.

Objective tinnitus is uncommon and results from noise generated by structures near the ear. Sometimes the tinnitus is loud enough to be heard by the examiner.

Characteristics: Tinnitus may be described as buzzing, ringing, roaring, whistling, or hissing and is sometimes variable and complex. Objective tinnitus typically is pulsatile (synchronous with the heartbeat) or intermittent. Tinnitus is most noticeable in quiet environments and in the absence of distracting stimuli and, thus, frequently seems worse at bedtime.

Tinnitus may be intermittent or continuous. Continuous tinnitus is at best annoying and is often quite distressing. Some patients adapt to its presence better than others; depression occasionally results. Stress generally exacerbates tinnitus.

Pathophysiology

Subjective tinnitus is thought to be caused by abnormal neuronal activity in the auditory cortex. This activity results when input from the auditory pathway (cochlea, auditory nerve, brain stem nuclei, auditory cortex) is disrupted or altered in some manner. This disruption may cause loss of suppression of intrinsic cortical activity and perhaps creation of new neural connections. Some believe the phenomenon is similar to the development of phantom limb pain after amputation. Conductive hearing loss (eg, caused by cerumen impaction, otitis media, or eustachian tube dysfunction) may also be associated with subjective tinnitus, by altering sound input to the central auditory system.

Objective tinnitus represents actual noise generated by physiologic phenomena occurring near the middle ear. Usually the noise comes from blood vessels, either normal vessels in conditions of increased or turbulent flow (eg, caused by atherosclerosis) or abnormal vessels (eg, in tumors or vascular malformations). Sometimes muscle spasms or myoclonus of palatal muscles or muscles in the middle ear (stapedius, tensor tympani) cause clicking sounds.

Etiology

Causes may be considered by whether they cause subjective or objective tinnitus (see Table 46-3).

Subjective tinnitus: Subjective tinnitus may occur with almost any disorder affecting the auditory pathways.

The most common disorders are those that involve sensorineural hearing loss, particularly

• Acoustic trauma (noise-induced sensorineural hearing loss)

• Aging (presbycusis)

• Ototoxic drugs

• Meniere's disease

Infections and CNS lesions (eg, caused by tumor, stroke, multiple sclerosis) that affect auditory pathways also may be responsible.

Disorders causing conductive hearing loss also may cause tinnitus. These include obstruction of the ear canal by cerumen, a foreign body, or external otitis. Otitis media, barotrauma, eustachian tube dysfunction, and otosclerosis may also be associated with tinnitus.

Temporomandibular joint dysfunction may be associated with tinnitus in some patients.

Objective tinnitus: Objective tinnitus usually involves noise from vascular flow, which causes an audible pulsating sound synchronous with the pulse. Causes include

• Turbulent flow through the carotid artery or jugular vein

• Highly vascular middle ear tumors

• Dural arteriovenous malformations (AVMs)

Muscle spasms or myoclonus of palatal muscles or those of the middle ear (stapedius, tensor tympani) may cause perceptible noise, typically a rhythmic clicking. Such spasms may be idiopathic or caused by tumors, head trauma, and infectious or demyelinating diseases (eg, multiple sclerosis). Palatal myoclonus causes visible movement of the palate, tympanic membrane, or both that coincides with tinnitus.

Evaluation

History: History of present illness should note duration of tinnitus, whether it is in one or both ears, and whether it is a constant tone or intermittent. If intermittent, the clinician should determine whether it is regular and whether it is about the rate of the pulse or sporadic. Any exacerbating or relieving factors (eg, swallowing, head position) should be noted. Important associated symptoms include hearing loss, vertigo, ear pain, and ear discharge.

Review of systems should seek symptoms of possible causes, including diplopia and difficulty swallowing or speaking (lesions of the brain stem) and focal weakness and sensory changes (peripheral nervous system disorders). The impact of the tinnitus on the patient also should be assessed. Whether the tinnitus is sufficiently distressing to cause significant anxiety, depression, or sleeplessness should be noted.

Past medical history should ask about risk factors for tinnitus, including exposure to loud noise, sudden pressure change (from diving or air travel), history of ear or CNS infections or trauma, radiation therapy to the head, and recent major weight loss (risk of eustachian dysfunction). Drug use should be ascertained, particularly any salicylates, aminoglycosides, or loop diuretics.

Physical examination: Physical examination focuses on the ear and the nervous system.

The ear canal should be inspected for discharge, foreign body, and cerumen. The tympanic membrane should be inspected for signs of acute infection (eg, redness, bulging), chronic infection (eg, perforation, cholesteatoma), and tumor (red or bluish mass). A bedside hearing test should be done.

Cranial nerves, particularly vestibular function (see p. 423), are tested along with peripheral strength, sensation, and reflexes. A stethoscope is used to listen for vascular noise over the course of the carotid arteries and jugular veins and over and adjacent to the ear.

Red flags: The following findings are of particular concern:

• Bruit, particularly over the ear or skull

• Accompanying neurologic symptoms or signs (other than hearing loss)

• Unilateral tinnitus

Interpretation of findings: In some cases, tinnitus may indicate retrocochlear pathology, such as an acoustic neuroma (benign but invasive tumor originating from the vestibular portion of the 8th cranial nerve in the internal auditory canal).

It is important to note whether the tinnitus is unilateral because acoustic neuromas may manifest only with unilateral tinnitus. This diagnosis is more likely if there is also unilateral sensorineural hearing loss or asymmetric hearing loss with worse hearing in the ear with tinnitus.

It also is important to distinguish the uncommon cases of objective tinnitus from the more common cases of subjective tinnitus. Tinnitus that is pulsatile or intermittent is almost always objective (although not always detectable by the examiner), as is that associated with a bruit. Pulsatile tinnitus is nearly always benign. Continuous tinnitus is usually subjective (except perhaps for that caused by a venous hum, which may be identified by presence of a bruit and often by a change in tinnitus with head rotation or jugular vein compression).

Specific causes can often be suspected by findings on examination (see Table 46-3). In particular, exposure to loud noise, barotrauma, or certain drugs before onset suggests those factors as the cause.

Testing: All patients with significant tinnitus should be referred for comprehensive audiologic evaluation to determine the presence, degree, and type of hearing loss.

In patients with unilateral tinnitus and hearing loss, acoustic neuroma should be ruled out by gadolinium-enhanced MRI. In those with unilateral tinnitus and normal hearing and physical examination, MRI is not necessary unless tinnitus persists > 6 mo.

Other testing depends on patient presentation (see Table 46-3).

Those with visible evidence of a vascular tumor in the middle ear require CT, gadolinium-enhanced MRI, and referral to a subspecialist if the diagnosis is confirmed.

Those with pulsatile, objective tinnitus and no ear abnormalities on examination or audiology require further investigation of the vascular system (carotid, vertebral, and intracranial vessels). The usual test sequence is to begin with magnetic resonance angiography (MRA). However, because MRA is not very sensitive for dural AVMs, many clinicians then consider doing an arteriogram. However, because dural AVMs are rare, the significant risks of arteriography must be weighed against the potential benefit of diagnosis and treatment (with embolization) of this vascular anomaly.

Treatment

Treatment of the underlying disorder may lessen tinnitus. Correcting hearing loss (eg, with a hearing aid) relieves tinnitus in about 50% of patients.

Because stress and other mental factors (eg, depression) can exacerbate symptoms, efforts to recognize and treat these factors may help. Many patients are reassured by learning that their tinnitus does not represent a serious medical problem. Tinnitus also can be worsened by caffeine and other stimulants, so patients should try eliminating use of these substances.

Although no specific medical or surgical therapy is available, many patients find that background sound masks the tinnitus and may help them fall asleep. Some patients benefit from a tinnitus masker, a device worn like a hearing aid that provides a low-level sound that can cover up the tinnitus. Tinnitus retraining therapy, offered by programs that specialize in tinnitus treatment, are helpful for many patients. Electrical stimulation of the inner ear, as with a cochlear implant, occasionally

[Table 46-3. Some Causes of Tinnitus]

reduces the tinnitus but is appropriate only for patients who are profoundly deaf.

Geriatrics Essentials

One out of 4 people > 65 yr have significant hearing impairment. Because tinnitus is common in people with sensorineural hearing loss, tinnitus is a common complaint among the elderly.

Key Points

• Subjective tinnitus is caused by an abnormality somewhere in the auditory pathway.

• Objective tinnitus is caused by an actual noise produced in a vascular structure near the ear.

• Loud noise, aging, Meniere's disease, and drugs are the most common causes of subjective tinnitus.

• Unilateral tinnitus with hearing loss or dizziness/dysequilibrium warrants gadolinium-enhanced MRI to rule out acoustic neuroma.

• Any tinnitus accompanied by a neurologic deficit is of concern.

Dizziness and Vertigo

Dizziness is an imprecise term patients often use to describe various related sensations, including

• Faintness (a feeling of impending syncope)

• Light-headedness

• Feeling of imbalance or unsteadiness

• A vague spaced-out or swimmy-headed feeling

Vertigo is a false sensation of movement of the self or the environment. Usually the perceived movement is rotary—a spinning or wheeling sensation—but some patients simply feel pulled to one side. Vertigo is not a diagnosis—it is a description of a sensation.

Both sensations may be accompanied by nausea and vomiting or difficulty with balance, gait, or both.

Perhaps because these sensations are hard to describe in words, patients often use "dizziness," "vertigo," and other terms interchangeably and inconsistently. Different patients with the same underlying disorder may describe their symptoms very differently. A patient may even give different descriptions of the same "dizzy" event during a given visit depending on how the question is asked. Because of this discrepancy, even though vertigo seems to be a clearly delineated subset of dizziness, many clinicians prefer to consider the two symptoms together.

However they are described, dizziness and vertigo may be disturbing and even incapacitating, particularly when accompanied by nausea and vomiting. Symptoms cause particular problems for people doing an exacting or dangerous task, such as driving, flying, or operating heavy machinery.

Dizziness accounts for about 5 to 6% of physician visits. It may occur at any age but becomes more common with increasing age; it affects about 40% of people over 40 yr at some time. Dizziness may be temporary or chronic. Chronic dizziness, defined as lasting > 1 mo, is more common among elderly people.

Pathophysiology

The **vestibular system** is the main neurologic system involved in balance. This system includes

• The vestibular apparatus of the inner ear

• The 8th (vestibulocochlear) cranial nerve, which conducts signals from the vestibular apparatus to the central components of the system

• The vestibular nuclei in the brain stem and cerebellum

Disorders of the inner ear and 8th cranial nerve are considered peripheral disorders. Those of the vestibular nuclei and their pathways in the brain stem and cerebellum are considered central disorders.

The sense of balance also incorporates visual input from the eyes and proprioceptive input from the peripheral nerves (via the spinal cord). The cerebral cortex receives output from the lower centers and integrates the information to produce the perception of motion.

Vestibular apparatus: Perception of stability, motion, and orientation to gravity originates in the vestibular apparatus, which consists of

• The 3 semicircular canals

• The 2 otolith organs—the saccule and utricle

Rotary motion causes flow of endolymph in the semicircular canal oriented in the plane of motion. Depending on the direction of flow, endolymph movement either stimulates or inhibits neuronal output from hair cells lining the canal. Similar hair cells in the saccule and utricle are embedded in a matrix of Ca carbonate crystals (otoliths). Deflection of the otoliths by gravity stimulates or inhibits neuronal output from the attached hair cells.

Etiology

There are numerous structural (trauma, tumors, degenerative), vascular, infectious, toxic (including drug-related), and idiopathic causes (see Table 46-4), but only about 5% of cases are caused by a serious disorder.

[**Table 46-4.** Some Causes of Dizziness and Vertigo]

The **most common causes of dizziness with vertigo** involve some component of the peripheral vestibular system:

• Benign positional vertigo

• Meniere's disease

• Vestibular neuronitis

• Labyrinthitis

Less often, the cause is a central vestibular disorder (most commonly migraine), a disorder with a more global effect on cerebral function, a psychiatric disorder, or a disorder affecting visual or proprioceptive input. Sometimes, no cause can be found.

The **most common causes of dizziness without vertigo** are less clear cut, but they are usually not otologic and probably are

• Drug effects

• Multifactorial or idiopathic

Nonneurologic disorders with a more global effect on cerebral function sometimes manifest as dizziness and rarely as vertigo. These disorders typically involve inadequate substrate (eg, O_2, glucose) delivery caused by hypotension, hypoxemia, anemia, or hypoglycemia; when severe, some of these disorders may manifest as syncope. Additionally, certain hormonal changes (eg, as with thyroid disease, menstruation, pregnancy) can cause dizziness. Numerous CNS-active drugs can cause dizziness independent of any toxic effect on the vestibular system.

Occasionally, dizziness and vertigo may be psychogenic. Patients with panic disorder, hyperventilation syndrome, anxiety, or depression may present with complaints of dizziness.

In elderly patients, dizziness is often multifactorial secondary to drug adverse effects and age-diminished visual, vestibular, and proprioceptive abilities. Two of the most common specific causes are disorders of the inner ear: benign paroxysmal positional vertigo and Meniere's disease.

Evaluation

History: History of present illness should cover the sensations felt; an open-ended question is best (eg, "Different people use the word 'dizziness' differently. Can you please describe as thoroughly as you can what you feel?"). Brief, specific questioning as to whether the feeling is faintness, light-headedness, loss of balance, or vertiginous may bring some clarity, but persistent efforts to categorize a patient's sensations are unnecessary. Other elements are more valuable and clear-cut:

• Severity of initial episode

• Severity and characteristics of subsequent episodes

• Symptoms continuous or episodic

• If episodic, frequency and duration

• Triggers and relievers (ie, triggered by head/body position change)

• Associated aural symptoms (eg, hearing loss, ear fullness, tinnitus)

• Severity and related disability

Is the patient having a single, sudden, acute event, or has dizziness been chronic and recurrent? Was the first episode the most severe (vestibular crisis)? How long do episodes last, and what seems to trigger and worsen them? The patient should be asked specifically about movement of the head, arising, being in anxious or stressful situations, and menses. Important associated symptoms include headache, hearing loss, tinnitus, nausea and vomiting, impaired vision, focal weakness, and difficulty walking. The severity of impact on the patient's life should be estimated: Has the patient fallen? Is the patient reluctant to drive or leave the house? Has the patient missed work days?

Review of systems should seek symptoms of causative disorders, including URI symptoms (inner ear disorders); chest pain, palpitations, or both (heart disease); dyspnea (lung disease); dark stools (anemia

caused by GI blood loss); and weight change or heat or cold intolerance (thyroid disease).

Past medical history should note presence of recent head trauma (usually obvious), migraine, diabetes, heart or lung disease, and drug and alcohol abuse. In addition to identifying all current drugs, drug history should assess recent changes in drugs, doses, or both.

Physical examination: Examination begins with a review of vital signs, including presence of fever, rapid or irregular pulse, and supine and standing BP, noting any drop in BP on standing up (orthostatic hypotension) and whether standing provokes symptoms. If standing does provoke symptoms, postural symptoms should be distinguished from those triggered by head movement by returning the patient supine until symptoms dissipate and then rotating the head.

The ENT and neurologic examinations are fundamental. Specifically, with the patient supine, the eyes are checked for presence, direction, and duration of spontaneous nystagmus (for full description of examination for nystagmus, see Sidebar 46-1). Direction and duration of nystagmus and development of vertigo are noted.

A gross bedside hearing test is done, the ear canal is inspected for discharge and foreign body, and the tympanic membrane is checked for signs of infection or perforation.

Cerebellar function is tested by assessing gait and doing a finger-nose test and Romberg's test. The remainder of the neurologic examination is done, including testing the rest of the cranial nerves.

Red flags: The following findings are of particular concern:

• Head or neck pain

• Ataxia

• Loss of consciousness

• Focal neurologic deficit

Interpretation of findings: Traditionally, differential diagnosis has been based on the exact nature of the chief complaint (ie, distinguishing dizziness from light-headedness from vertigo). However, the inconsistency of patients' descriptions and the poor specificity of symptoms make this unreliable. A better approach places more weight on the onset and timing of symptoms, the triggers, and associated symptoms and findings, particularly otologic and neurologic ones.

Some constellations of findings are highly suggestive (see Table 46-4), particularly those that help differentiate peripheral from central vestibular disorders.

• Peripheral: Ear symptoms (eg, tinnitus, fullness, hearing loss) usually indicate a peripheral disorder. They are typically associated with vertigo and not generalized dizziness (unless caused by uncompensated peripheral vestibular weakness). Symptoms are usually paroxysmal, severe, and episodic; continuous dizziness is rarely due to peripheral vertigo. Loss of consciousness is not associated with dizziness due to peripheral vestibular pathology.

• Central: Ear symptoms are rarely present, but gait/balance disturbance is common. Nystagmus is not inhibited by visual fixation.

Testing: Patients with a sudden, ongoing attack should have pulse oximetry and finger-stick glucose test. Women should have a pregnancy test. Most clinicians also do an ECG. Other tests are done based on findings (see Table 46-4), but generally gadolinium-enhanced MRI is indicated for patients with acute symptoms who have headache, neurologic abnormalities, or any other findings suggestive of a CNS etiology.

Patients with chronic symptoms should have gadolinium-enhanced MRI to look for evidence of stroke,

multiple sclerosis, or other CNS lesions.

Patients for whom results of bedside tests of hearing and vestibular function are abnormal or equivocal should undergo formal testing with audiometry and electronystagmography.

Laboratory tests are rarely helpful, except for patients with chronic vertigo and bilateral hearing loss, for whom syphilis serology is indicated.

Treatment

Treatment is directed at the cause, including stopping, reducing, or switching any causative drugs.

If a vestibular disorder is present and thought to be secondary to active Meniere's disease or vestibular neuronitis or labyrinthitis, the most effective vestibular nerve suppressants are diazepam (2 to 5 mg po q 6 to 8 h, with higher doses given under supervision for severe vertigo) or oral antihistamine/anticholinergic drugs (eg, meclizine 25 to 50 mg tid). All of these drugs can cause drowsiness, thereby limiting their use for certain patients. Nausea can be treated with prochlorperazine 10 mg IM qid or 25 mg rectally bid. Vertigo associated with benign paroxysmal positional vertigo is treated with the Epley maneuver (otolith repositioning) done by an experienced practitioner (see Fig. 48-1 on p. 443). Meniere's disease is best managed by an otolaryngologist with training in management of this chronic disorder, but initial management consists of a low-salt diet and a K-sparing diuretic.

Patients with persistent or recurrent vertigo secondary to unilateral vestibular weakness (such as secondary to vestibular neuronitis) usually benefit from vestibular rehabilitation therapy done by an experienced physical therapist. Most patients compensate well, although some, especially the elderly, have more difficulty. Physical therapy can also provide important safety information for elderly or particularly disabled patients.

Geriatrics Essentials

As people age, organs involved in balance function less well. For example, seeing in dim light becomes more difficult, inner ear structures deteriorate, proprioception becomes less sensitive, and mechanisms that control BP become less responsive (eg, to postural changes, postprandial demands). Older people also are more likely to have cardiac or cerebrovascular disorders that can contribute to dizziness. They also are more likely to be taking drugs that can cause dizziness, including those for hypertension, angina, heart failure, seizures, and anxiety, as well as certain antibiotics, antihistamines, and sleep aids. Thus, dizziness in elderly patients usually has more than one cause.

Although unpleasant at any age, the consequences of dizziness and vertigo are a particular problem for elderly patients. Those with frailty are at significant risk of falling with consequent fractures; their fear of moving and falling often significantly decreases their ability to do daily activities.

In addition to treatment of specific causes, elderly patients with dizziness or vertigo may benefit from physical therapy and exercises to strengthen muscles and help maintain independent ambulation as long as possible.

Key Points

- Vague or inconsistently described symptoms may still be associated with a serious condition.
- Cerebrovascular disease and drug effects should be sought, particularly in elderly patients.
- Peripheral vestibular system disorders should be differentiated from central vestibular system disorders.
- Immediate neuroimaging should be done when symptoms are accompanied by headache, focal neurologic abnormalities, or both.

Chapter 47. Hearing Loss

Introduction

Nearly 10% of people in the US have some degree of hearing loss. About 1/800 to 1/1000 neonates are born with severe to profound hearing loss. Two to 3 times as many are born with lesser hearing loss. During childhood, another 2 to 3/1000 children acquire moderate to severe hearing loss. Adolescents are at risk from excessive exposure to noise, head trauma, or both. Older adults typically experience a progressive decrease in hearing (presbycusis—see p. 438), which is probably related to aging and noise exposure.

Hearing deficits in early childhood can result in lifelong impairments in receptive and expressive language skills. The severity of the handicap is determined by the age at which the hearing loss occurred; the nature of the loss (its duration, the frequencies affected, and the degree); and the susceptibilities of the individual child (eg, coexisting visual impairment, intellectual disability, primary language deficits, inadequate linguistic environment). Children who have other sensory, linguistic, or cognitive deficiencies are affected most severely.

Pathophysiology

Hearing loss can be classified as conductive, sensorineural, or both (mixed loss).

Conductive hearing loss occurs secondary to lesions in the external auditory canal, tympanic membrane (TM), or middle ear. These lesions prevent sound from being effectively conducted to the inner ear.

Sensorineural hearing loss is caused by lesions of either the inner ear (sensory) or the auditory (8th) nerve (neural—see
Table 47-1). This distinction is important because sensory

[**Table 47-1.** Differences Between Sensory and Neural Hearing Losses]

hearing loss is sometimes reversible and is seldom life threatening. A neural hearing loss is rarely recoverable and may be due to a potentially life-threatening brain tumor—commonly a cerebellopontine angle tumor.

Mixed loss may be caused by severe head injury with or without fracture of the skull or temporal bone, by chronic infection, or by one of many genetic disorders. It may also occur when a transient conductive hearing loss, commonly due to otitis media, is superimposed on a sensorineural hearing loss.

Etiology

Hearing loss can be congenital (see
Table 47-2) or acquired (see
Table 47-3), progressive or sudden (see also p. 438), temporary or permanent, unilateral or bilateral, and mild or profound. Drug-induced ototoxicity is discussed elsewhere (see p. 443).

The **most common causes** overall are the following:

• Cerumen accumulation

• Noise

• Aging

• Infections (particularly among children and young adults)

Cerumen (earwax) accumulation is the most common cause of treatable hearing loss, especially in the

elderly. Foreign bodies obstructing the canal are sometimes a problem in children, both because of their presence and because of any damage inadvertently caused during their removal.

Noise can cause sudden or gradual sensorineural hearing loss. In acoustic trauma, hearing loss results from exposure to a single, extreme noise (eg, a nearby gunshot or explosion); some patients have tinnitus as well. The loss is usually temporary (unless there is also blast damage, which may destroy the TM, ossicles, or both). In noise-induced hearing loss, the loss develops over time because of chronic exposure to noise > 85 decibels (dB—see Sidebar 47-1 on p. 434). Although people vary somewhat in susceptibility to noise-induced hearing loss, nearly everyone loses some hearing if they are exposed to sufficiently intense noise for an adequate time. Repeated exposure to loud noise ultimately results in loss of hair cells in the organ of Corti. Hearing loss typically occurs first at 4 kHz and gradually spreads to the lower and higher frequencies as exposure continues. In contrast to most other causes of sensorineural hearing losses, noise-induced hearing loss may be less severe at 8 kHz than at 4 kHz.

[**Table 47-2.** Congenital Causes of Hearing Loss*]

Acute otitis media (AOM—see p. 448) is a common cause of transient mild to moderate hearing loss (mainly in children). However, without treatment, AOM sequelae and chronic otitis media (and the rarer purulent labyrinthitis) can cause permanent loss, particularly if a cholesteatoma forms.

Secretory otitis media (SOM—see p. 450) occurs in several ways. Almost all episodes of AOM are followed by a period of 2 to 4 wk of SOM. SOM can also be caused by eustachian tube dysfunction (eg, resulting from cleft palate, benign or malignant tumors of the nasopharynx, or rapid changes in external air pressure as occur during descent from high altitudes or rapid ascent while scuba diving).

Autoimmune disorders can cause sensorineural hearing loss at all ages and can cause other symptoms and signs as well.

Evaluation

Evaluation consists of detecting and quantifying hearing loss and determining etiology (particularly reversible causes).

Screening: Most adults and older children notice a sudden hearing loss, and caregivers may suspect that a neonate has a severe hearing loss within the first week of life when the neonate does not respond to voices or other sounds. However, progressive losses and nearly all losses in infants and young children must be detected by screening. Screening should begin at birth (see p. 2717) so that linguistic input can allow optimal language development. Suspected hearing loss at any time should prompt referral to a specialist. If screening is not done, severe bilateral losses may not be recognized until age 2 yr, and mild to moderate or severe unilateral losses are often not recognized until children reach school age.

History: History of present illness should note how long hearing loss has been perceived, how it began (eg, gradual, acute), whether it is unilateral or bilateral, and whether sound is distorted (eg, music is off—dull or lifeless) or there is difficulty with speech discrimination. The patient should be asked whether the loss followed any acute event (eg, head injury, barotrauma [particularly a diving injury], starting of a drug). Important accompanying symptoms include other otologic symptoms (eg, ear pain, tinnitus, ear discharge), vestibular symptoms (eg, disorientation in the dark, vertigo), and other neurologic symptoms (eg, headache, weakness or asymmetry of the face, an abnormal sense of taste, fullness of the ear). In children, important associated symptoms include presence of delays in speech or language development and delayed motor development.

Review of systems should seek to determine the impact of hearing difficulty on the patient's life.

Past medical history should note previous possibly causative disorders, including CNS infection, repeated ear infections, chronic exposure to loud noise, head trauma, rheumatic disorders (eg, RA, lupus), and a family history of hearing loss. Drug history should specifically query current or previous use of ototoxic drugs (see Table 47-3).

Physical examination: The focus is examination of the ears and hearing and the neurologic examination. The external ear is inspected for obstruction, infection, congenital malformations, and other lesions. The TM is examined for perforation, drainage, otitis media, and cholesteatoma. During the neurologic examination, particular attention needs to be paid to the 2nd through 7th cranial nerves as well as to vestibular and cerebellar function because abnormalities in these areas often occur with tumors of the brain stem and cerebellopontine angle. Weber's test and the Rinne test require a tuning fork to differentiate conductive from sensorineural hearing loss.

In **Weber's test**, the stem of a vibrating 512-Hz or 1024-Hz tuning fork is placed on the midline of the head, and the patient indicates in which ear the tone is louder. In unilateral conductive hearing loss, the tone is louder in the ear with hearing loss. In unilateral sensorineural hearing loss, the tone is louder in the normal ear because the tuning fork stimulates both inner ears equally and the patient perceives the stimulus with the unaffected ear.

In the **Rinne test**, hearing by bone and by air conduction is compared. Bone conduction bypasses the external and middle ear and tests the integrity of the inner ear, 8th cranial nerve, and central auditory pathways. The stem of a vibrating tuning fork is held against the mastoid (for bone conduction); as soon as the sound is no longer perceived, the fork is removed from the mastoid, and the still-vibrating tines are held close to the pinna (for air conduction). Normally, the fork can once more be heard, indicating that air conduction is better than bone conduction. With conductive hearing loss, the relationship is reversed; bone conduction is louder than air conduction. With sensorineural hearing loss, both air and bone conduction are reduced, but air conduction remains louder.

Red flags: Findings of particular concern are

• Unilateral sensorineural hearing loss

• Abnormalities of cranial nerves (other than hearing loss)

Interpretation of findings: Many causes of hearing loss (eg, cerumen, injury, significant noise exposure, infectious sequelae, drugs) are readily apparent based on results of the examination (see Table 47-3).

Associated findings are helpful in diagnosing the remaining small number of patients in

[**Table 47-3.** Some Causes of Acquired Hearing Loss]

whom no clear cause can be found. Those who have focal neurologic abnormalities are of particular concern. The 5th or 7th cranial nerve or both are often affected by tumors that involve the 8th nerve, so loss of facial sensation and weak jaw clench (5th) and hemifacial weakness and taste abnormalities (7th) point to a lesion in that area. Signs of autoimmune disorders, maxillofacial malformations, and renal dysfunction may suggest these disorders as a cause.

Any child with delays in speech or language development or difficulty in school should be evaluated for hearing loss. Intellectual disability, aphasia, and autism must also be considered. Delayed motor development may signal vestibular deficit, which is often associated with a sensorineural hearing loss.

Testing: Testing includes

• Audiologic tests

• Sometimes MRI or CT

Audiologic tests are required for all people who have hearing loss; these tests usually include

• Measurement of pure-tone thresholds with air and bone conduction

• Speech reception threshold

• Speech discrimination

• Tympanometry

• Acoustic reflex testing

Information gained from these tests helps determine whether more definitive differentiation of sensory from neural hearing loss is needed.

Pure-tone audiometry quantifies hearing loss. An audiometer delivers sounds of specific frequencies (pure tones) at different intensities to determine the patient's hearing threshold (how loud a sound must be to be perceived) for each frequency. Hearing in each ear is tested from 125 or 250 to 8000 Hz by air conduction (using earphones) and up to 4 kHz by bone conduction (using an oscillator in contact with the mastoid process or forehead). Test results are plotted on graphs called audiograms (see Fig. 47-1), which show the difference between the patient's hearing threshold and normal hearing at each frequency. The difference is measured in dB (see Sidebar 47-1). The normal threshold is considered 0 dB hearing level (Hl); hearing loss is considered present if the patient's threshold is > 25 dB Hl. When hearing loss is such as to require loud test tones, intense tones presented to one ear may be heard in the other ear. In such cases, a masking sound, usually narrow band noise, is presented to the ear not being tested to isolate it.

Speech audiometry includes the speech reception threshold (SRT) and the word recognition score. The SRT is a measure of the intensity at which speech is recognized. To determine the SRT, the examiner presents the patient with a list of words at specific sound intensities. These words usually have 2 equally accented syllables (spondees), such as railroad, staircase, and baseball. The examiner notes the intensity at which the patient repeats 50% of the words correctly. The SRT approximates the average hearing level at speech frequencies (eg, 500 Hz, 1000 Hz, 2000 Hz).

Sidebar 47-1 Sound Levels

Sound intensity and pressure (the physical correlates of loudness) are measured in decibels (dB). A dB is a unitless figure that compares 2 values and is defined as the logarithm of the ratio of a measured value to a reference value, multiplied by a constant:

$$dB = k \log (V_{measured}/V_{ref})$$

By convention, the reference value for sound pressure level (SPL) is taken as the quietest 1000-Hz sound detectable by young, healthy human ears.* The sound may be measured in terms of pressure (N/m^2) or intensity ($watts/m^2$).

Because sound intensity equals the square of sound pressure, the constant (k) for SPL is 20; for sound intensity, 10. Thus, each 20-dB increase represents a 10-fold increase in SPL but a 100-fold increase in sound intensity.

The dB values in the table below give only a rough idea of the risk of hearing loss. Some of them are dB SPL values (referenced to N/m^2), whereas others represent peak dB or dB on the A-scale (a scale that emphasizes the frequencies that are most hazardous to human hearing).

Db	Example
0	Faintest sound heard by human ear
30	Whisper, quiet library
60	Normal conversation, sewing machine, typewriter
90	Lawnmower, shop tools, truck traffic (8 h/day is the maximum exposure without protection†)
100	Chain saw, pneumatic drill, snowmobile (2 h/day is the maximum exposure without protection)

115 Sandblasting, loud rock concert, automobile horn (15 min/day is the maximum exposure without protection)

140 Gun muzzle blast, jet engine (noise causes pain and even brief exposure injures unprotected ears; injury may occur even with hearing protectors)

180 Rocket launching pad

*In audiometric testing, because human ears respond differently at different frequencies, the reference value changes for each frequency tested. Threshold values reported on audiograms take this into account; the normal threshold is always 0 dB, regardless of the actual SPL.

†Mandatory federal standard, but protection is recommended for more than brief exposure to sound levels > 85 db.

The word recognition score tests the ability to discriminate among the various speech sounds or phonemes. It is determined by presenting 50 phonetically balanced one-syllable words at an intensity of 35 to 40 dB above the patient's SRT. The word list contains phonemes in the same relative frequency found in conversational English. The score is the percentage of words correctly repeated by the patient and reflects the ability to understand speech

[Fig. 47-1. Audiogram of right ear in a patient with normal hearing.]

under optimal listening conditions. A normal score ranges from 90 to 100%. The word recognition score is normal with conductive hearing loss, albeit at a higher intensity level, but can be reduced at all intensity levels with sensorineural hearing loss. Discrimination is even poorer in neural than in sensory hearing loss.

Tympanometry measures the impedance of the middle ear to acoustic energy and does not require patient participation. It is commonly used to screen children for middle ear effusions. A probe containing a sound source, microphone, and air pressure regulator is placed snugly with an airtight seal into the ear canal. The probe microphone records the reflected sound from the TM while pressure in the canal is varied. Normally, maximal compliance of the middle ear occurs when the pressure in the ear canal equals atmospheric pressure. Abnormal compliance patterns suggest specific anatomic disruptions. In eustachian tube obstruction and middle ear effusion, maximal compliance occurs with a negative pressure in the ear canal. 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 fixed, as in stapedial ankylosis in otosclerosis, compliance may be normal or reduced.

The acoustic reflex is contraction of the stapedius muscle in response to loud sounds, which changes the compliance of the TM, protecting the middle ear from acoustic trauma. The reflex is tested by presenting a tone and measuring what intensity provokes a change in middle ear impedance as noted by movement of the TM. An absent reflex could indicate middle ear disease or a tumor of the auditory nerve.

Advanced testing is sometimes needed. Gadolinium-enhanced MRI of the head to detect lesions of the cerebellopontine angle may be needed in patients with an abnormal neurologic examination or those whose audiologic testing shows poor word recognition, asymmetric sensorineural hearing loss, or a combination when the etiology is not clear.

CT is done if bony tumors or bony erosion is suspected. Magnetic resonance angiography is done if vascular abnormalities such as glomus tumors are suspected.

The auditory brain stem response uses surface electrodes to monitor brain wave response to acoustic stimulation in people who cannot otherwise respond.

Electrocochleography measures the activity of the cochlea and the auditory nerve with an electrode placed on or through the eardrum. It can be used to assess and monitor patients with dizziness, can be used in patients who are awake, and is useful in intraoperative monitoring.

Otoacoustic emissions testing measures sounds produced by outer hair cells of the cochlea in response to a sound stimulus usually placed in the ear canal. It is used to screen neonates and infants for hearing loss and to monitor the hearing of patients who are using ototoxic drugs (eg, gentamicin, cisplatin).

Certain patients, such as children with a reading or other learning problem and elderly people who seem to hear but do not comprehend, should undergo a central auditory evaluation. It measures discrimination of degraded or distorted speech, discrimination in the presence of a competing message in the opposite ear, the ability to fuse incomplete or partial messages delivered to each ear into a meaningful message, and the capacity to localize sound in space when acoustic stimuli are delivered simultaneously to both ears.

Treatment

The causes of a hearing loss should be determined and treated. Ototoxic drugs should be stopped or the dose should be lowered unless the severity of the disease being treated (usually cancer or a severe infection) requires that the risk of additional ototoxic hearing loss be accepted. Attention to peak and trough drug levels may help minimize risk.

Fluid from middle ear effusion can be drained by myringotomy and prevented with the insertion of a tympanostomy tube. Benign growths (eg, enlarged adenoids, nasal polyps) and malignant tumors (eg, nasopharyngeal cancers, sinus cancers) blocking the eustachian tube or ear canal can be removed. Hearing loss caused by autoimmune disorders may respond to corticosteroids.

Damage to the TM or ossicles or otosclerosis may require reconstructive surgery. Brain tumors causing hearing loss may in some cases be removed and hearing preserved.

Many causes of hearing loss have no cure, and treatment involves compensating for the hearing loss with hearing aids and, for severe to profound loss, a cochlear implant. In addition, various coping mechanisms may help.

Hearing aids: Amplification of sound with a hearing aid helps many people. Although hearing aids do not restore hearing to normal, they can significantly improve communication. Physicians should encourage hearing aid use and help patients overcome a sense of social stigma that continues to obstruct use of these devices, perhaps by making the analogy that a hearing aid is to hearing as eye glasses are to seeing.

All hearing aids have a microphone, amplifier, speaker, earpiece, and volume control, although they differ in the location of these components. An audiologist should be involved in selection and fitting of a hearing aid.

The best models are adjusted to a person's particular pattern of hearing loss. People with mainly high-frequency hearing loss do not benefit from simple amplification, which merely makes the garbled speech they hear sound louder. They usually need a hearing aid that selectively amplifies the high frequencies. Some hearing aids contain vents in the ear mold, which facilitate the passage of high-frequency sound waves. Some use digital sound processing with multiple frequency channels so that amplification more precisely matches hearing loss as measured on the audiogram.

Telephone use can be difficult for people with hearing aids. Typical hearing aids cause squealing when the ear is placed next to the phone handle. Some hearing aids have a phone coil with a switch that turns the microphone off and links the phone coil electromagnetically to the speaker magnet in the phone.

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. Some people with mild hearing loss limited to high frequencies are most comfortably fitted with postauricular aids and completely open ear canals. Canal aids are contained entirely within the ear canal and are cosmetically acceptable to many people who would otherwise refuse

to use a hearing aid, but they are difficult for some people (especially the elderly) to manipulate. The CROS aid (contralateral routing of signals) is occasionally used for 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 radio transmitter. This device enables the wearer to hear sounds from the nonfunctioning side, allowing for some limited capacity to localize sound. If the better ear also has some hearing loss, the sound from both sides can be amplified with the binaural CROS (BiCROS) aid. The body aid type is appropriate for profound hearing loss. 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).

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 held against the head, usually over the mastoid, with a spring band, and sound is conducted through 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 (bone-anchored hearing aids or BAHAs) are surgically implanted in the mastoid process, avoiding the discomfort and prominence of the spring band.

Cochlear implants: Profoundly deaf patients, including those with some hearing but who even with a hearing aid cannot understand speech without the assistance of vision (lip-reading or speech-reading), may benefit from a cochlear implant. This device provides electrical signals directly into the auditory nerve via multiple electrodes implanted in the cochlea. An external microphone and processor convert sound waves to electrical impulses, which are transmitted through the skin electromagnetically from an external induction coil to an internal coil implanted in the skull above and behind the ear. The internal coil connects to electrodes inserted in the scala tympani.

Cochlear implants help with speech-reading by providing information about the intonation of words and the rhythm of speech. Many if not most adults with cochlear implants can discriminate words without visual clues, allowing them to talk on the telephone. Cochlear implants enable deaf people to hear and distinguish environmental sounds and warning signals. They also help deaf people modulate their voice and make their speech more intelligible.

Brain stem implants: Patients who have had both acoustic nerves destroyed (eg, by bilateral temporal bone fractures or neurofibromatosis) can have some hearing restored by means of brain stem implants that have electrodes connected to sound-detecting and sound-processing devices similar to those used for cochlear implants.

Coping mechanisms: Alerting systems that use light let people know when the doorbell is ringing, a smoke detector is sounding, or a baby is crying. Special sound systems transmitting infrared or FM radio signals help people hear in theaters, churches, or other places where competing noise exists. Many television programs carry closed captioning. Telephone communication devices are also available.

Lip-reading or speech-reading is particularly important for people who can hear but have trouble discriminating sounds. Most people get useful speech information from lip-reading even without formal training. Even people with normal hearing can better understand speech in a noisy place if they can see the speaker. To use this information the listener must be able to see the speaker's mouth. Health care personnel should be sensitive to this issue and always position themselves appropriately when speaking to the hearing-impaired. Observing the position of a speaker's lips allows recognition of the consonant being spoken, thereby improving speech comprehension in patients with high-frequency hearing loss. Lip-reading may be learned in aural rehabilitation sessions in which a group of age-matched peers meets regularly for instruction and supervised practice in optimizing communication.

People can gain control over their listening environment by modifying or avoiding difficult situations. For example, people can visit a restaurant during off-peak hours, when it is quieter. They can ask for a booth, which blocks out some extraneous sounds. In direct conversations, people may ask the speaker to face them. At the beginning of a telephone conversation, they can identify themselves as being hearing-impaired. At a conference, the speaker can be asked to use an assistive listening system, which makes use of either inductive loop, infrared, or FM technology that sends sound through the microphone to a patient's hearing aid.

People with profound hearing loss often communicate by using sign language. American Sign Language (ASL) is the most common version in the US. Other forms include Signed English, Signing Exact English, and Cued Speech.

Treatment in Children

In addition to treatment of any cause and the provision of hearing aids, children with hearing loss require support of language development with appropriate therapy. Because children must hear language to learn it spontaneously, most deaf children develop language only with special training, ideally beginning as soon as the hearing loss is identified (an exception would be a deaf child growing up with deaf parents who are fluent sign language users). Deaf infants must be provided with a form of language input. For example, a visually based sign language can provide a foundation for later development of oral language if a cochlear implant is not available.

If infants as young as 1 mo have profound bilateral hearing loss and cannot benefit from hearing aids, they can be a candidate for a cochlear implant. Although cochlear implants allow auditory communication in many children with either congenital or acquired deafness, they are, in the main, more effective in children who already have developed language. Children who have postmeningitic deafness develop an ossified inner ear; they should receive cochlear implants early to maximize effectiveness. Children whose acoustic nerves have been destroyed by tumors may be helped by implantation of brain stem auditory-stimulating electrodes. Children with cochlear implants may have a slightly greater risk of meningitis than children without cochlear implants or adults with cochlear implants.

Children with unilateral deafness should be allowed to use a special system in the classroom, such as an FM auditory trainer. With these systems, the teacher speaks into a microphone that sends signals to a hearing aid in the child's nonaffected ear, improving the child's greatly impaired ability to hear speech against a noisy background.

Geriatrics Essentials

Elderly people typically experience a progressive decrease in hearing (presbycusis). The prevalence of hearing impairment is 30% in people > 65 and is 40 to 50% in those > 75. Nonetheless, hearing loss in the elderly should be evaluated and not ascribed to aging; elderly patients may have a tumor, a neurologic or autoimmune disorder, or an easily correctible conductive hearing loss. Also, hearing loss in the elderly facilitates dementia (which can be mitigated by properly correcting hearing loss).

Presbycusis: Presbycusis is sensorineural hearing loss that probably results from a combination of age-related deterioration and cell death in various components of the hearing system and the effects of chronic noise exposure.

Hearing loss usually affects the highest frequencies (18 to 20 kHz) early on and gradually affects the lower frequencies; it usually becomes clinically significant when it affects the critical 2- to 4-kHz range at about age 55 to 65 (sometimes sooner). The loss of high-frequency hearing significantly affects speech comprehension. Although the loudness of speech seems normal, certain consonant sounds (eg, C, D, K, P, S, T) become hard to hear. Consonant sounds are the most important sounds for speech recognition. For example, when "shoe," "blue," "true," "too," or "new" is spoken, many people with presbycusis can hear the "oo" sound, but most have difficulty recognizing which word has been spoken because they cannot distinguish the consonants. This inability to distinguish consonants causes affected people to often think the speaker is mumbling. A speaker attempting to speak louder usually accentuates vowel sounds (which are low frequency), doing little to improve speech recognition. Speech comprehension is particularly difficult when background noise is present.

Screening: A screening tool is often helpful for elderly people because many do not complain of hearing loss. One tool is the Hearing Handicap Inventory for the Elderly-Screening Version, which asks

• Does a hearing problem cause you to feel embarrassed when you meet people?

• Does a hearing problem cause you to feel frustrated when talking to a family member?

• Do you have difficulty hearing when someone whispers?

• Do you feel handicapped by a hearing problem?

• Does a hearing problem cause you difficulty when visiting friends, relatives, or neighbors?

• Does a hearing problem cause you to attend religious services less often than you would like?

• Does a hearing problem cause you to have arguments with family members?

• Does a hearing problem cause you difficulty when listening to the television or radio?

• Do you feel that any difficulty with your hearing hampers your personal or social life?

• Does a hearing problem cause you difficulty when in a restaurant with relatives or friends?

Scoring is "no" = 0 points, "sometimes" = 2 points, and "yes" = 4 points. Scores > 10 suggest significant hearing impairment and necessitate follow-up.

Prevention

Prevention of hearing loss consists mainly of limiting duration and intensity of noise exposure. People required to expose themselves to loud noise must wear ear protectors (eg, plastic plugs in the ear canals or glycerin-filled muffs over the ears). The Occupational Safety and Health Administration (OSHA) of the US Department of Labor and similar agencies in many other countries have standards regarding the length of time that a person can be exposed to a noise. The louder the noise, the shorter the permissible time of exposure.

Key Points

• Cerumen, genetic disorders, infections, aging, and noise exposure are the most common causes.

• All patients with hearing loss should have audiologic testing.

• Cranial nerve deficits and other neurologic deficits should raise concern and warrant imaging tests.

Sudden Deafness

Sudden deafness is severe sensorineural hearing loss that develops within a few hours or is noticed on awakening. It affects about 1/5000 people each year. Initial hearing loss is typically unilateral (unless drug-induced) and may range in severity from mild to profound. Many also have tinnitus, and some have dizziness, vertigo, or both.

Sudden deafness has some causes that differ from chronic hearing loss and must be addressed urgently.

Etiology

The following are common characteristics of sudden deafness:

• Most cases (see Table 47-4) are idiopathic.

• Some occur in the course of an obvious explanatory event.

A few represent the initial manifestation of an occult but identifiable disorder.

Idiopathic: There are numerous theories for which some evidence (although conflicting and incomplete) exists. The most promising possibilities include viral infections (particularly involving herpes simplex), autoimmune attacks, and acute microvascular occlusion.

Obvious event: Some causes of sudden deafness are readily apparent.

Blunt head trauma with temporal bone fracture or severe concussion involving the cochlea can cause sudden hearing loss.

Large ambient pressure changes (eg, caused by diving) or strenuous activities (eg, weightlifting) can induce a perilymphatic fistula between the middle and inner ear, causing sudden, severe symptoms. Perilymphatic fistula can also be congenital; it can spontaneously cause a sudden loss or loss may occur after trauma or pressure changes.

[**Table 47-4.** Some Causes of Sudden Deafness]

Ototoxic drugs can result in hearing loss occurring sometimes within a day, especially with an overdose (systemically or when applied to a large wound area, such as a burn). There is a rare genetic mitochondrial-transmitted disorder that increases the susceptibility to aminoglycoside ototoxicity.

A number of **infections** cause sudden deafness during or immediately after acute illness. Common causes include bacterial meningitis, Lyme disease, and many viral infections that affect the cochlea (and sometimes the vestibular apparatus). The most common viral causes in the developed world are mumps and herpes. Measles is a very rare cause because most of the population is immunized.

Occult disorders: Sudden deafness rarely can be an isolated first manifestation of some disorders that usually have other initial symptoms. Sudden deafness rarely may be the first manifestation of an acoustic neuroma, multiple sclerosis, Meniere's disease, or a small cerebellar stroke. Syphilis reactivation in HIV-infected patients rarely can cause sudden deafness.

Cogan's syndrome is a rare autoimmune reaction directed against an unknown common autoantigen in the cornea and inner ear; > 50% of patients present with vestibuloauditory symptoms. About 10 to 30% of patients also have a severe systemic vasculitis, which may include life-threatening aortitis.

Some vasculitic disorders can cause hearing loss, some of which is acute. Hematologic disorders, such as Waldenstrom's macroglobulinemia, sickle cell disease, and some forms of leukemia, rarely can cause sudden deafness.

Evaluation

Evaluation consists of detecting and quantifying hearing loss and determining etiology (particularly reversible causes).

History: History of present illness should verify that loss is sudden and not chronic. The history should also note whether loss is unilateral or bilateral and whether there is a current acute event (eg, head injury, barotrauma [particularly a diving injury], infectious illness). Important accompanying symptoms include other otologic symptoms (eg, tinnitus, ear discharge), vestibular symptoms (eg, disorientation in the dark, vertigo), and other neurologic symptoms (eg, headache, weakness or asymmetry of the face, abnormal sense of taste).

Review of systems should seek symptoms of possible causes, including transient, migratory neurologic deficits (multiple sclerosis) and eye irritation and redness (Cogan's syndrome).

Past medical history should ask about known HIV or syphilis infection and risk factors for them (eg, multiple sex partners, unprotected intercourse). Family history should note close relatives with hearing loss (suggesting a congenital fistula). Drug history should specifically query current or previous use of ototoxic drugs (see Table 47-4) and whether the patient has known renal insufficiency or renal failure.

Physical examination: The examination focuses on the ears and hearing and on the neurologic examination.

The tympanic membrane is inspected for perforation, drainage, or other lesions. During the neurologic examination, attention should be paid to the cranial nerves (particularly the 5th, 7th, and 8th) and to vestibular and cerebellar function because abnormalities in these areas often occur with tumors of the brain stem and cerebellopontine angle.

Weber's test and the Rinne test require a tuning fork to differentiate conductive from sensorineural hearing loss (see p. 429).

Additionally, the eyes are examined for redness and photophobia (possible Cogan's syndrome), and the skin is examined for rash (eg, viral infection, syphilis).

Red flags: Findings of particular concern are

• Abnormalities of cranial nerves (other than hearing loss)

Interpretation of findings: Traumatic, ototoxic, and some infectious causes are usually apparent clinically. A patient with perilymphatic fistula may hear an explosive sound in the affected ear when the fistula occurs and may also have sudden vertigo, nystagmus, and tinnitus.

Focal neurologic abnormalities are of particular concern. The 5th cranial nerve, 7th cranial nerve, or both are often affected by tumors that involve the 8th cranial nerve, so loss of facial sensation and weak jaw clench (5th) and hemifacial weakness and taste abnormalities (7th) point to a lesion in that area.

Unilateral hearing loss accompanied by tinnitus and vertigo also suggests Meniere's disease. Systemic symptoms suggesting inflammation (eg, fevers, rash, joint pains, mucosal lesions) should raise suspicion of an occult infection or autoimmune disorder.

Testing: Typically, patients should have an audiogram, and unless the diagnosis is clearly an acute infection or drug toxicity, most clinicians do gadolinium-enhanced MRI to diagnose inapparent causes. Patients with an acute traumatic cause also should have MRI. If a perilymphatic fistula is suspected clinically, it may be confirmed by tympanometry and electronystagmography (ENG); CT is usually done to show the bony characteristics of the inner ear.

Patients who have risk factors for or symptoms that suggest causes should have appropriate tests (eg, serologic tests for possible HIV infection or syphilis, CBC and coagulation profile for hematologic disorders, ESR and antinuclear antibodies for vasculitis).

Treatment

Treatment focuses on the causative disorder when known. Fistulas are explored and repaired surgically.

In viral and idiopathic cases, hearing returns to normal in about 50% of patients and is partially recovered in others.

In patients who recover their hearing, improvement usually occurs within 10 to 14 days.

For patients with idiopathic loss, many clinicians empirically give a short course of glucocorticoids and antiviral drugs effective against herpes simplex (eg, valacyclovir, famciclovir). Glucocorticoids can be given orally or by transtympanic injection; it is unclear which route is more effective.

Key Points

• Most cases are idiopathic.

• A few cases have an obvious cause (eg, major trauma, acute infection, drugs).

- A very few cases represent unusual manifestations of treatable disorders.

Chapter 48. Inner Ear Disorders

Introduction

(See also Hearing Loss on p. 429.)

The inner ear is in the petrous area of the temporal bone. Within the bone is the osseous labyrinth, which encases the membranous labyrinth. The osseous labyrinth includes the vestibular system (made up of the semicircular canals and the vestibule) and the cochlea. The vestibular system is responsible for balance and posture; the cochlea, for hearing.

Acoustic Neuroma

(Acoustic Neurinoma; 8th Nerve Tumor; Vestibular Schwannoma)

An acoustic neuroma is a Schwann cell-derived tumor of the 8th cranial nerve. Symptoms include unilateral hearing loss. Diagnosis is based on audiology and confirmed by MRI. Treatment is surgical removal, stereotactic radiation therapy, or both.

Acoustic neuromas almost always arise from the vestibular division of the 8th cranial nerve and account for about 7% of all intracranial tumors. As the tumor expands, it projects from the internal auditory meatus into the cerebellopontine angle and compresses the cerebellum and brain stem. The 5th cranial nerve and later the 7th cranial nerve are affected.

Bilateral acoustic neuromas are common in neurofibromatosis type 2.

Symptoms and Signs

Slowly progressive unilateral sensorineural hearing loss is the hallmark symptom. However, the onset of hearing loss may be abrupt, and the degree of impairment may fluctuate. Other early symptoms include unilateral tinnitus, dizziness and dysequilibrium, headache, sensation of pressure or fullness in the ear, otalgia, trigeminal neuralgia, and numbness or weakness of the facial nerve.

Diagnosis

• Audiogram

• If positive, gadolinium-enhanced MRI

An audiogram is the first test done (see p. 433). It usually reveals an asymmetric sensorineural hearing loss and a greater impairment of speech discrimination than would be expected for the degree of hearing loss. 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. Although not usually required in the routine evaluation of a patient with asymmetric sensorineural hearing loss, caloric testing shows marked vestibular hypoactivity (canal paresis). Such findings indicate the need for imaging tests, preferably gadolinium-enhanced MRI.

Treatment

• Surgical removal

• Sometimes stereotactic radiation therapy

Small tumors may be removed with microsurgery that preserves the facial nerve. A middle cranial fossa or retrosigmoid approach may preserve remaining hearing; a translabyrinthine route may be used if no useful hearing remains. Large tumors are removed with the translabyrinthine approach regardless of the remaining hearing. Stereotactic radiation therapy as the sole treatment modality is used predominantly in the management of small tumors in older patients; its long-term efficacy and adverse effects are under

study.

Benign Paroxysmal Positional Vertigo

(Benign Postural or Positional Vertigo)

In benign paroxysmal positional vertigo (BPPV), short (< 60 sec) episodes of vertigo occur with certain head positions. Nausea and nystagmus develop. Diagnosis is clinical. Treatment involves canalith repositioning maneuvers. Drugs and surgery are rarely, if ever, indicated.

BPPV is the most common cause of relapsing otogenic vertigo. It affects people increasingly as they age and can severely affect balance in the elderly, leading to potentially injurious falls.

Etiology

The condition is thought to be caused by displacement of otoconial crystals (Ca carbonate crystals normally embedded in the saccule and utricle). This displaced material stimulates hair cells in the posterior semicircular canal, creating the illusion of motion. Etiologic factors include spontaneous degeneration of the utricular otolithic membranes, labyrinthine concussion, otitis media, ear surgery, recent viral infection (eg, viral neuronitis), head trauma, prolonged anesthesia or bed rest, previous vestibular disorders (eg, Meniere's disease), and occlusion of the anterior vestibular artery.

Symptoms and Signs

Vertigo is triggered when the patient's head moves (eg, when rolling over in bed or bending over to pick up something). Acute vertigo lasts only a few seconds to minutes; episodes tend to peak in the morning and abate throughout the day. Nausea and vomiting may occur, but hearing loss and tinnitus do not.

Diagnosis

• Clinical evaluation

• Gadolinium-enhanced MRI if findings suggest CNS lesion

Diagnosis is based on characteristic symptoms, on nystagmus as determined by the Dix-Hallpike maneuver (a provocative test for positional nystagmus—see Sidebar 46-1 on p. 414), and on absence of other abnormalities on neurologic examination. Such patients require no further testing. Patients with nystagmus suggesting a CNS lesion undergo gadolinium-enhanced MRI. Unlike the positional nystagmus of BPPV, 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 caused by a CNS lesion may be vertical or change direction and, if rotary, is likely to be in the unexpected direction.

Treatment

• Provocative maneuvers to fatigue symptoms

• Canalith repositioning maneuvers

• Drug treatment typically not recommended

BPPV usually subsides spontaneously in several weeks or months but may continue for months or years. Because the condition can be long-lasting, drug treatment (like that used in Meniere's disease—see p. 445) is not recommended. Often, the adverse effects of drugs worsen dysequilibrium.

Because BPPV is fatigable, one therapy is to have the patient perform provocative maneuvers early in the day in a safe environment. Symptoms are then minimal for the rest of the day.

Canalith repositioning maneuvers (Epley maneuver—see

Fig. 48-1—and Semont maneuver) involve moving the head through a series of specific positions intended to return the errant canalith to the utricle. After performing these maneuvers, the patient should remain erect or semi-erect for 1 to 2 days. Both maneuvers can be repeated as necessary.

[**Fig. 48-1.** The Epley maneuver.]

For the Semont maneuver, the patient is seated upright in the middle of a stretcher. The patient's head is rotated toward the unaffected ear; this rotation is maintained throughout the maneuver. Next, the torso is lowered laterally onto the stretcher so that the patient is lying on the side of the affected ear with the nose pointed up. After 3 min in this position, the patient is quickly moved through the upright position without straightening the head and is lowered laterally to the other side now with the nose pointed down. After 3 min in this position, the patient is slowly returned to the upright position, and the head is rotated back to normal.

Drug-Induced Ototoxicity

A wide variety of drugs can be ototoxic (see Table 48-1).

Factors affecting ototoxicity include dose, duration of therapy, concurrent renal failure,

[**Table 48-1.** Some Drugs that Cause Ototoxicity]

infusion rate, lifetime dose, co-administration with other drugs having ototoxic potential, and genetic susceptibility. Ototoxic drugs should not be used for otic topical application when the tympanic membrane is perforated because the drugs might diffuse into the inner ear.

Streptomycin tends to cause more damage to the vestibular portion than to the auditory portion of the inner ear. Although vertigo and difficulty maintaining balance tend to be temporary, severe loss of vestibular sensitivity may persist, sometimes permanently. Loss of vestibular sensitivity causes difficulty walking, especially in the dark, and oscillopsia (a sensation of bouncing of the environment with each step). About 4 to 15% of patients who receive 1 g/day for > 1 wk develop measurable hearing loss, which usually occurs 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 may be absorbed to affect hearing, particularly if 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 and are both capable of causing profound, permanent hearing loss while sparing balance. Viomycin has both cochlear and vestibular toxicity. Gentamicin and tobramycin have vestibular and cochlear toxicity, causing impairment in balance and hearing.

Vancomycin can cause hearing loss, especially in the presence of renal insufficiency.

Chemotherapeutic (antineoplastic) drugs, particularly those containing platinum (cisplatin and carboplatin), can cause tinnitus and hearing loss. Hearing loss can be profound and permanent, occurring immediately after the first dose, or can be delayed until several months after completion of treatment. Sensorineural hearing loss strikes bilaterally, progresses decrementally, and is permanent.

Ethacrynic acid and furosemide given IV have caused profound, permanent hearing loss in patients with renal failure who had been receiving aminoglycoside antibiotics.

Salicylates in high doses (> 12 325-mg tablets of aspirin per day) cause temporary hearing loss and tinnitus. Quinine and its synthetic substitutes can also cause temporary hearing loss.

Prevention

Ototoxic antibiotics should be avoided during pregnancy. The elderly and people with preexisting hearing loss should not be treated with ototoxic drugs if other effective drugs are available. The lowest effective dosage of ototoxic drugs should be used and levels should be closely monitored. If possible before treatment with an ototoxic drug, hearing should be measured and then monitored during treatment; symptoms are not reliable warning signs.

Herpes Zoster Oticus

(Geniculate Herpes; Ramsay Hunt Syndrome; Viral Neuronitis)

Herpes zoster oticus is infection of the 8th cranial nerve ganglia and the geniculate ganglion of the facial nerve by the herpes zoster virus.

Risk factors for herpes infection include immunodeficiency secondary to cancer, chemotherapy, radiation therapy, and HIV infection.

Symptoms and Signs

Symptoms include severe ear pain, transient or permanent facial paralysis (resembling Bell's palsy), vertigo lasting days to weeks, and hearing loss (which may be permanent or which may resolve partially or completely). Vesicles occur on the pinna and in the external auditory canal along the distribution of the sensory branch of the facial nerve. Symptoms of meningoencephalitis (eg, headache, confusion, stiff neck) are uncommon. Sometimes other cranial nerves are involved.

Diagnosis

Diagnosis usually is clinical. If there is any question about viral etiology, vesicular scrapings may be collected for direct immunofluorescence or for viral cultures, and MRI is done.

Treatment

• Perhaps corticosteroids, antivirals, and surgical decompression

Although there is no reliable evidence that corticosteroids, antiviral drugs, or surgical decompression makes a difference, they are the only possibly useful treatments. Corticosteroids are started with prednisone 60 mg po once/day for 4 days, followed by gradual tapering of the dose over the next 2 wk. Acyclovir 800 mg po q 4 h 5 times/day or valacyclovir 1 g po bid for 10 days may shorten the clinical course. Vertigo is effectively suppressed with diazepam 2 to 5 mg po q 4 to 6 h. Pain may require oral opioids. Postherpetic neuralgia may be treated with amitriptyline. Surgical decompression of the fallopian canal may be indicated if the facial palsy is complete (no visible facial movement). Before surgery, however, electroneurography is done and should show a > 90% decrement.

Meniere's Disease

(Endolymphatic Hydrops)

Meniere's disease is an inner ear disorder that causes vertigo, fluctuating sensorineural hearing loss, and tinnitus. There is no diagnostic test. Vertigo and nausea are treated with anticholinergics or benzodiazepines. Diuretics and a low-salt diet may decrease frequency and severity of episodes. For severe cases, the vestibular system can be ablated with topical gentamicin or surgery.

In Meniere's disease, pressure and volume changes of the labyrinthine endolymph affect inner ear function. The etiology of endolymphatic fluid buildup is unknown. Risk factors include a family history of Meniere's disease, preexisting autoimmune disorders, allergies, trauma to the head or ear, and, rarely, syphilis (even several decades previously). Peak incidence is between ages 20 and 50.

Symptoms and Signs

Patients have sudden attacks of vertigo lasting up to 24 h, usually with nausea and vomiting. Accompanying symptoms include diaphoresis, diarrhea, and gait unsteadiness. Tinnitus may be constant or intermittent, buzzing or roaring; it is not related to position or motion. Hearing impairment, typically affecting low frequencies, may follow. Before an episode, most patients sense fullness or pressure in the affected ear. In 50% of patients, only one ear is affected.

During the early stages, symptoms remit between episodes; symptom-free interludes may last > 1 yr. As the disease progresses, however, hearing impairment persists and gradually worsens, and tinnitus may be constant.

Diagnosis

• Clinical evaluation

• Audiogram and gadolinium-enhanced MRI to rule out other causes

The diagnosis, made clinically, is primarily one of exclusion. Similar symptoms can result from viral labyrinthitis or neuritis, a cerebellopontine angle tumor (eg, acoustic neuroma), or a brain stem stroke. Patients with suggestive symptoms should have an audiogram and an MRI (with gadolinium enhancement) of the CNS with attention to the internal auditory canals to exclude other causes. Audiogram typically shows a low-frequency sensorineural hearing loss in the affected ear.

On examination during an acute attack, the patient has nystagmus and falls to the affected side. Between attacks, the Fukada stepping test (marching in place with eyes closed) can be used; a patient with Meniere's disease often turns away from the affected ear, consistent with a unilateral labyrinthine lesion. Additionally, the Rinne test and Weber's test may indicate sensorineural hearing loss (see p. 431).

Treatment

• Symptom relief with antiemetics, antihistamines, or benzodiazepines

• Diuretics and low-salt diet

• Rarely vestibular ablation by drugs or surgery

Meniere's disease tends to be self-limited. Treatment of an acute attack is aimed at symptom relief. Anticholinergics (eg, prochlorperazine or promethazine 25 mg rectally or 10 mg po q 6 to 8 h) can minimize vagal-mediated GI symptoms. Antihistamines (eg, diphenhydramine, meclizine, or cyclizine 50 mg po q 6 h) or benzodiazepines (eg, diazepam 5 mg po q 6 to 8 h) are used to sedate the vestibular system. Some physicians also use a corticosteroid burst (eg, prednisone 60 mg once/day for 1 wk, tapered over another wk) for an acute episode.

A low-salt (< 1.5 g/day) diet, avoidance of alcohol and caffeine, and a diuretic (eg, hydrochlorothiazide 25 mg po once/day) may help prevent vertigo and are useful for many patients.

Intratympanic gentamicin (chemical labyrinthectomy) may be used when medical management is unsuccessful. Typical dose is 1 mL (at a 30 mg/mL concentration, made by diluting the commercial 40 mg/mL preparation with bicarbonate) injected through the tympanic membrane. Follow-up with serial audiometry is recommended to distinguish hearing loss from cochleotoxicity. The injection can be repeated in 4 wk if vertigo persists without hearing loss.

Surgery is reserved for patients with frequent, severely debilitating episodes who are unresponsive to other modalities. Endolymphatic sac decompression relieves vertigo in some patients and poses minimal risk of hearing loss. Vestibular neurectomy (an intracranial procedure) relieves vertigo in about 95% of patients and usually preserves hearing. A surgical labyrinthectomy is done only if preexisting hearing loss is profound.

Unfortunately, there is no known way to prevent the natural progression of hearing loss. Most patients sustain moderate to severe sensorineural hearing loss in the affected ear within 10 to 15 yr.

Purulent Labyrinthitis

Purulent (suppurative) labyrinthitis is bacterial infection of the inner ear, often causing deafness and loss of vestibular function.

Purulent labyrinthitis usually occurs when bacteria spread to the inner ear during the course of severe acute otitis media, purulent meningitis, or an enlarging cholesteatoma.

Symptoms include severe vertigo and nystagmus, nausea and vomiting, tinnitus, and varying degrees of hearing loss. Pain and fever are common.

Purulent labyrinthitis is suspected if vertigo, nystagmus, sensorineural hearing loss, or a combination occurs during an episode of acute otitis media. CT of the temporal bone is done to identify erosion of the otic capsule bone or other complications of acute otitis media, such as coalescent mastoiditis. MRI may be indicated if symptoms of meningitis or brain abscess, such as altered mental status, meningismus, or high fever, are present; in such cases, a lumbar puncture and blood cultures also are done.

Treatment is with IV antibiotics appropriate for meningitis (eg, ceftriaxone 50 to 100 mg/kg IV once/day to maximum 2 g) adjusted according to results of culture and sensitivity testing. A myringotomy (and sometimes tympanostomy tube placement) is done to drain the middle ear. Mastoidectomy may be required.

Vestibular Neuronitis

Vestibular neuronitis causes a self-limited episode of vertigo, presumably due to inflammation of the vestibular division of the 8th cranial nerve; some vestibular dysfunction may persist.

Although etiology is unclear, a viral cause is suspected.

Symptoms and Signs

Symptoms include a single attack of severe vertigo, with nausea and vomiting and persistent nystagmus toward the affected side, which lasts 7 to 10 days. The nystagmus is unidirectional, horizontal, and spontaneous, with fast-beat oscillations in the direction of the unaffected ear. The absence of concomitant tinnitus or hearing loss is a hallmark of vestibular neuronitis. The condition slowly subsides after this initial episode. Some patients have residual dysequilibrium, especially with rapid head movements, probably due to permanent vestibular injury.

Diagnosis

• Audiology, electronystagmography, and MRI

Patients undergo an audiologic assessment, electronystagmography with caloric testing, and gadolinium-enhanced MRI of the head, with attention to the internal auditory canals to exclude other diagnoses, such as cerebellopontine angle tumor, brain stem hemorrhage, or infarction. MRI may show enhancement of the vestibular nerves, consistent with inflammatory neuritis.

Treatment

• Symptom relief with antiemetics, antihistamines, or benzodiazepines

Symptoms are addressed as in Meniere's disease (see p. 445), ie, with anticholinergics, antiemetics (eg, prochlorperazine or promethazine 25 mg rectally or 10 mg po q 6 to 8 h), antihistamines or benzodiazepines, and a corticosteroid burst with rapid taper. If vomiting is prolonged, IV fluids and

electrolytes may be required. Vestibular rehabilitation (usually given by a physical therapist) helps compensate for any residual vestibular deficit.

Chapter 49. Middle Ear and Tympanic Membrane Disorders

Introduction

(See also Otic Tumors on p. 493)

Middle ear disorders may be secondary to infection, eustachian tube obstruction, or trauma. Information about objects placed in the ear and symptoms such as rhinorrhea, nasal obstruction, sore throat, URI, allergies, headache, systemic symptoms, and fever aid diagnosis. The appearance of the external auditory canal and tympanic membrane (see Fig. 49-1) often yields a diagnosis. The nose, nasopharynx, and oropharynx are examined for signs of infection and allergy and for evidence of tumors. Middle ear function is evaluated with use of pneumatic otoscopy, Weber's tuning fork test and the Rinne tuning fork test, tympanometry, and audiologic tests (see p. 431).

Mastoiditis

Mastoiditis is a bacterial infection of the mastoid air cells, which typically occurs after acute otitis media. Symptoms include redness, tenderness, swelling, and fluctuation over the mastoid process, with displacement of the pinna. Diagnosis is clinical. Treatment is with antibiotics, such as ceftriaxone, and mastoidectomy if drug therapy is not effective.

In acute purulent otitis media, inflammation often extends into the mastoid antrum and air cells, resulting in fluid accumulation. In a few patients, bacterial infection develops in the collected fluid, typically with the same organism causing the otitis media; pneumococcus is most common. Mastoid infection can cause osteitis of the septae, leading to coalescence of the air cells. The infection may decompress through a perforation in the tympanic membrane or extend through the lateral mastoid cortex, forming a postauricular subperiosteal abscess. Rarely, it extends centrally, causing a temporal lobe abscess or a septic thrombosis of the lateral sinus. Occasionally, the infection may erode through the tip of the mastoid and drain into the neck (called a Bezold abscess).

Symptoms and Signs

Symptoms begin days to weeks after onset of acute otitis media and include fever and persistent, throbbing otalgia. Nearly all patients have signs of otitis media (see p. 448) and purulent otorrhea. Redness, swelling,

[**Fig. 49-1.** Tympanic membrane of right ear (A); tympanic cavity with tympanic membrane removed (B).]

tenderness, and fluctuation may develop over the mastoid process; the pinna is typically displaced laterally and inferiorly.

Diagnosis

• Clinical evaluation

• Rarely CT

Diagnosis is clinical. CT is rarely necessary but can confirm the diagnosis and show the extent of the infection. Any middle ear drainage is sent for culture and sensitivity. Tympanocentesis for culture purposes can be done if no spontaneous drainage occurs. CBC and ESR may be abnormal but are neither sensitive nor specific and add little to the diagnosis.

Treatment

• IV ceftriaxone

IV antibiotic treatment is initiated immediately with a drug that provides CNS penetration, such as

ceftriaxone 1 to 2 g (children, 50 to 75 mg/kg) once/day continued for ≥ 2 wk. Oral treatment with a quinolone may be acceptable. Subsequent antibiotic choice is guided by culture and sensitivity test results.

A subperiosteal abscess usually requires a simple mastoidectomy, in which the abscess is drained, the infected mastoid cells are removed, and drainage is established from the antrum of the mastoid to the middle ear cavity.

Myringitis

(Bullous Myringitis)

Myringitis is a form of acute otitis media in which vesicles develop on the tympanic membrane.

Myringitis can develop with viral, bacterial (particularly *Streptococcus pneumoniae*), or mycoplasmal otitis media. Pain occurs suddenly and persists for 24 to 48 h. Hearing loss and fever suggest a bacterial origin. Diagnosis is based on otoscopic visualization of vesicles on the tympanic membrane.

Because differentiation among a viral, bacterial, and mycoplasmal cause is difficult, antibiotics effective against organisms causing otitis media are prescribed (see Table 49-1). Severe, continued pain may be relieved by rupturing the vesicles with a myringotomy knife or by oral analgesics (eg, oxycodone with acetaminophen). Topical analgesics (eg, benzocaine, antipyrine) may also be beneficial.

Acute Otitis Media

Acute otitis media (AOM) is a bacterial or viral infection of the middle ear, usually accompanying a URI. Symptoms include otalgia, often with systemic symptoms (eg, fever, nausea, vomiting, diarrhea), especially in the very young. Diagnosis is based on otoscopy. Treatment is with analgesics and sometimes antibiotics.

Although AOM can occur at any age, it is most common between ages 3 mo and 3 yr. At this age, the eustachian tube is structurally and functionally immature; the angle of the eustachian tube is more horizontal; and the angle of the tensor veli palatini muscle and the cartilaginous eustachian tube renders the opening mechanism less efficient.

The etiology may be viral or bacterial. Viral infections are often complicated by secondary bacterial infection. In neonates, gram-negative enteric bacilli, particularly *Escherichia coli*, and *Staphylococcus aureus* cause AOM. In older infants and children < 14 yr, the most common organisms are *Streptococcus pneumoniae*, *Moraxella (Branhamella) catarrhalis*, and nontypeable *Haemophilus influenzae*; less common causes are group A β-hemolytic streptococci and *S. aureus*. In patients > 14 yr, *S. pneumoniae*, group A β-hemolytic streptococci, and *S. aureus* are most common, followed by *H. influenzae*.

In rare cases, bacterial middle ear infection spreads locally, resulting in acute mastoiditis, petrositis, or labyrinthitis. Intracranial spread is extremely rare and usually causes meningitis, but brain abscess, subdural empyema, epidural abscess, lateral sinus thrombosis, or otitic hydrocephalus may occur. Even with antibiotic treatment, intracranial complications are slow to resolve, especially in immunocompromised patients.

Symptoms and Signs

The usual initial symptom is earache, often with hearing loss. Infants may simply be cranky or have difficulty sleeping. Fever, nausea, vomiting, and diarrhea often occur in young children. Otoscopic examination can show a bulging, erythematous tympanic membrane (TM) with indistinct landmarks and displacement of the light reflex. Air insufflation (pneumatic otoscopy) shows poor mobility of the TM. Spontaneous perforation of the TM causes serosanguineous or purulent otorrhea.

Severe headache, confusion, or focal neurologic signs may occur with intracranial spread of infection.

Facial paralysis or vertigo suggests local extension to the fallopian canal or labyrinth.

[**Table 49-1.** Antibiotics for Otitis Media]

Diagnosis

Diagnosis usually is clinical. Except for fluid obtained during myringotomy, cultures are not generally done.

Treatment

• Analgesics

• Sometimes antibiotics

• Rarely myringotomy

Although 80% of cases resolve spontaneously, in the US, antibiotics are often given (see Table 49-1). Antibiotics relieve symptoms quicker (although results after 1 to 2 wk are similar) and may reduce the chance of residual hearing loss and labyrinthine or intracranial sequelae. However, with the recent emergence of resistant organisms, pediatric organizations have strongly recommended initial antibiotics only for those at highest risk (eg, those who are younger or more severely ill—see Table 49-2) or for those with recurrent AOM (eg, ≥ 4 episodes in 6 mo). Others, provided there is good follow-up, can safely

[**Table 49-2.** Guidelines for Using Antibiotics in Acute Otitis Media]

be observed for up to 72 h and given antibiotics only if no improvement is seen; if follow-up by phone is planned, a prescription can be given at the initial visit to save time and expense.

All patients receive analgesics (eg, acetaminophen, ibuprofen). In adults, topical intranasal vasoconstrictors, such as phenylephrine 0.25% 3 drops q 3 h, improve eustachian tube function. To avoid rebound congestion, these preparations should not be used > 4 days. Systemic decongestants (eg, pseudoephedrine 30 to 60 mg po q 6 h prn) may be helpful. Antihistamines (eg, chlorpheniramine 4 mg po q 4 to 6 h for 7 to 10 days) may improve eustachian tube function in people with allergies but should be reserved for the truly allergic. For children, neither vasoconstrictors nor antihistamines are of benefit.

Myringotomy may be done for a bulging TM, particularly if severe or persistent pain, fever, vomiting, or diarrhea is present. The patient's hearing, tympanometry, and TM appearance and movement are monitored until normal.

Prevention

Routine childhood vaccination against pneumococci (with pneumococcal conjugate vaccine), *H. influenzae* type B, and influenza decreases the incidence of AOM. Infants should not sleep with a bottle, and elimination of household smoking may decrease incidence.

Secretory Otitis Media

(Serous Otitis Media)

Secretory otitis media is an effusion in the middle ear resulting from incomplete resolution of acute otitis media or obstruction of the eustachian tube without infection. Symptoms include hearing loss and a sense of fullness or pressure in the ear. Diagnosis is based on appearance of the tympanic membrane and sometimes on tympanometry. Most cases resolve in 2 to 3 wk. If there is no improvement in 1 to 3 mo, some form of myringotomy is indicated, usually with insertion of a tympanostomy tube. Antibiotics and decongestants are not effective.

Normally, the middle ear is ventilated 3 to 4 times/min as the eustachian tube opens during swallowing, and O_2 is absorbed by blood in the vessels of the middle ear mucous membrane. If patency of the eustachian tube is impaired, a relative negative pressure develops within the middle ear, which can lead to fluid accumulation. This fluid may cause hearing loss.

Secretory otitis media is a common sequela to acute otitis media in children (often identified on routine ear recheck) and may persist for weeks to months. In other cases, eustachian tube obstruction may be secondary to inflammatory processes in the nasopharynx, allergies, hypertrophic adenoids or other obstructive lymphoid aggregations on the torus of the eustachian tube and in Rosenmuller's fossa, or benign or malignant tumors. The effusion may be sterile or (more commonly) contain pathogenic bacteria sometimes as a biofilm, although inflammation is not observed.

Symptoms and Signs

Patients may report no symptoms, but some (or their family members) note hearing loss. Patients may experience a feeling of fullness, pressure, or popping in the ear with swallowing. Otalgia is rare.

Various possible changes to the tympanic membrane (TM) include an amber or gray color, displacement of the light reflex, mild to severe retraction, and accentuated landmarks. On air insufflation, the TM may be immobile. An air-fluid level or bubbles of air may be visible through the TM.

Diagnosis

Diagnosis is clinical. Tympanometry may be done to confirm middle ear effusion. Adults and adolescents must undergo nasopharyngeal examination to exclude malignant or benign tumors.

Treatment

• Observation

• If unresolved, myringotomy with tympanostomy tube insertion

• If recurrent in childhood, sometimes adenoidectomy

For most patients, watchful waiting is all that is required. Antibiotics and decongestants are not helpful. For patients in whom allergies are clearly involved, antihistamines and topical corticosteroids may be helpful.

If no improvement occurs in 1 to 3 mo, myringotomy may be done for aspiration of fluid and insertion of a tympanostomy tube, which allows ventilation of the middle ear and temporarily ameliorates eustachian tube obstruction, regardless of cause. Tympanostomy tubes may be inserted for persistent conductive hearing loss or to help prevent recurrence of acute otitis media.

Occasionally, the middle ear is temporarily ventilated with the Valsalva maneuver or politzerization. To do the Valsalva maneuver, patients keep their mouth closed and try to forcibly blow air out through their pinched nostrils (ie, popping the ear). To do politzerization, the physician blows air with a special syringe (middle ear inflator) into one of the patient's nostrils and blocks the other while the patient swallows. This forces the air into the eustachian tube and middle ear. Neither procedure should be done if the patient has a cold and rhinorrhea.

Persistent, recurrent secretory otitis media may require correction of underlying nasopharyngeal conditions. Children may benefit from adenoidectomy, including the removal of the central adenoid mass as well as lymphoid aggregations on the torus of the eustachian tube and in Rosenmuller's fossa. Antibiotics should be given for bacterial rhinitis, sinusitis, and nasopharyngitis. Demonstrated allergens should be eliminated from the patient's environment and immunotherapy should be considered.

Chronic Otitis Media

Chronic otitis media is a persistent, chronically draining (> 6 wk), suppurative perforation of the tympanic membrane. Symptoms include painless otorrhea with conductive hearing loss. Complications include development of aural polyps, cholesteatoma, and other infections. Treatment requires complete cleaning of the ear canal several times daily, careful removal of granulation tissue, and application of topical corticosteroids and antibiotics. Systemic antibiotics and surgery are reserved for severe cases.

Chronic otitis media can result from acute otitis media, eustachian tube obstruction, mechanical trauma, thermal or chemical burns, blast injuries, or iatrogenic causes (eg, after tympanostomy tube placement). Further, patients with craniofacial abnormalities (eg, Down syndrome, cri du chat syndrome, cleft lip and/or cleft palate, velocardiofacial syndrome [Shprintzen's syndrome]) have an increased risk.

Chronic otitis media may become exacerbated after a URI or when water enters the middle ear through a tympanic membrane (TM) perforation during bathing or swimming. Infections often are caused by gram-negative bacilli or *Staphylococcus aureus*, resulting in painless, purulent, sometimes foul-smelling otorrhea. Persistent chronic otitis media may result in destructive changes in the middle ear (such as necrosis of the long process of the incus) or aural polyps (granulation tissue prolapsing into the ear canal through the TM perforation). Aural polyps are a serious sign, almost invariably suggesting cholesteatoma.

A cholesteatoma is an epithelial cell growth that forms in the middle ear, mastoid, or epitympanum after chronic otitis media (see
Plate 1). Lytic enzymes, such as collagenases, produced by the cholesteatoma can destroy adjacent bone and soft tissue. The cholesteatoma is also a nidus for infection; purulent labyrinthitis, facial paralysis, or intracranial abscess may develop.

Symptoms and Signs

Chronic otitis media usually manifests with conductive hearing loss and otorrhea. Pain is uncommon unless an associated osteitis of the temporal bone occurs. The TM is perforated and draining, and the auditory canal is macerated and littered with granulation tissue.

A patient with cholesteatoma has white debris in the middle ear, a draining polypoid mass protruding through the TM perforation, and an ear canal that appears clogged with mucopurulent granulation tissue.

Diagnosis

Diagnosis is usually clinical. Drainage is cultured. When cholesteatoma or other complications are suspected (as in a febrile patient or one with vertigo or otalgia), CT or MRI is done. These tests may reveal intratemporal or intracranial processes (eg, labyrinthitis, ossicular or temporal erosion, abscesses).

Treatment

• Irrigation and topical antibiotic drops

• Removal of granulation tissue

The ear canal is irrigated with a bulb syringe 3 times/day with a slightly warmed solution of half vinegar and half sterile water. After the ear drains, 10 drops topical ofloxacin solution are instilled in the affected ear 2 times/day for 14 days.

When granulation tissue is present, it is removed with microinstruments or cauterization with silver nitrate sticks. Ciprofloxacin 0.3% and dexamethasone 0.1% is then instilled into the ear canal for 7 to 10 days.

Severe exacerbations require systemic antibiotic therapy with amoxicillin 250 to 500 mg po q 8 h for 10 days or a 3rd-generation cephalosporin, subsequently modified by culture results and response to therapy.

Tympanoplasty is indicated for patients with marginal or attic perforations and chronic central TM

perforations. A disrupted ossicular chain may be repaired during tympanoplasty as well.

Cholesteatomas must be removed surgically. Because recurrence is common, reconstruction of the middle ear is usually deferred until a 2nd-look operation is done 6 to 8 mo later.

Otic Barotrauma

(Barotitis Media or Aerotitis Media)

Otic barotrauma is ear pain or damage to the tympanic membrane caused by rapid changes in pressure.

To maintain equal pressure on both sides of the tympanic membrane (TM), gas must move freely between the nasopharynx and middle ear. When a URI, allergy, or other mechanism interferes with eustachian tube functioning during changes in environmental pressure, the pressure in the middle ear either falls below ambient pressure, causing retraction of the TM, or rises above it, causing bulging. With negative middle ear pressure, a transudate of fluid may form in the middle ear. As the pressure differential increases, ecchymosis and subepithelial hematoma may develop in the mucous membrane of the middle ear and the TM. A very large pressure differential may cause bleeding into the middle ear, TM rupture, and the development of a perilymph fistula through the oval or round window.

Symptoms are severe pain, conductive hearing loss, and, if there is a perilymph fistula, sensory neural loss. Symptoms usually worsen during rapid increase in external air pressures, such as a rapid ascent (eg, during scuba diving) or descent (eg, during air travel). Sensorineural hearing loss or vertigo during descent suggests the development of a perilymph fistula; the same symptoms during ascent from a deep-sea dive can additionally suggest an air bubble formation in the inner ear.

Treatment

• Methods to equalize pressure (eg, yawning, swallowing, chewing gum)

Routine self-treatment of pain associated with changing pressure in an aircraft includes chewing gum, attempting to yawn and swallow, blowing against closed nostrils, and using decongestant nasal sprays.

If hearing loss is sensorineural and vertigo is present, a perilymph fistula is suspected and middle ear exploration to close a fistula is considered. If pain is severe and hearing loss is conductive, myringotomy is helpful.

Prevention

A person with nasal congestion due to URI or allergies should avoid flying and diving. When these activities are unavoidable, a topical nasal vasoconstrictor (eg, phenylephrine 0.25 to 1.0%) is applied 30 to 60 min before descent or ascent.

Otosclerosis

Otosclerosis is a disease of the bone of the otic capsule that causes an abnormal accumulation of new bone within the oval window.

In otosclerosis, the new bone traps and restricts the movement of the stapes, causing conductive hearing loss (see p. 429). Otosclerosis also may cause a sensorineural hearing loss, particularly when the foci of otosclerotic bone are adjacent to the scala media. Half of all cases are inherited. The measles virus plays an inciting role in patients with a genetic predisposition for otosclerosis.

Although about 10% of white adults have some otosclerosis (compared with 1% of blacks), only about 10% of affected people develop conductive hearing loss. Hearing loss caused by otosclerosis may manifest as early as age 7 or 8, but most cases do not become evident until the late teen or early adult years, when slowly progressive, asymmetric hearing loss is diagnosed. Fixation of the stapes may

progress rapidly during pregnancy.

A hearing aid may restore hearing. Alternatively, microsurgery to remove some or all of the stapes and to replace it with a prosthesis may be beneficial.

Traumatic Perforation of the Tympanic Membrane

Traumatic perforation of the tympanic membrane (TM) can cause pain, bleeding, hearing loss, tinnitus, and vertigo. Diagnosis is based on otoscopy. Treatment often is unnecessary. Antibiotics may be needed for infection. Surgery may be needed for perforations persisting > 2 mo, disruption of the ossicular chain, or injuries affecting the inner ear.

Traumatic causes of TM perforation include

• Insertion of objects into the ear canal purposely (eg, cotton swabs) or accidentally

• Concussion caused by an explosion or open-handed slap across the ear

• Head trauma (with or without basilar fracture)

• Sudden negative pressure (eg, strong suction applied to the ear canal)

• Barotrauma (eg, during air travel or scuba diving)

• Iatrogenic perforation during irrigation or foreign body removal

Penetrating injuries of the TM may result in dislocations of the ossicular chain, fracture of the stapedial footplate, displacement of fragments of the ossicles, bleeding, a perilymph fistula from the oval or round window resulting in leakage of perilymph into the middle ear space, or facial nerve injury.

Symptoms and Signs

Traumatic perforation of the TM causes sudden severe pain sometimes followed by bleeding from the ear, hearing loss, and tinnitus. Hearing loss is more severe if the ossicular chain is disrupted or the inner ear is injured. Vertigo suggests injury to the inner ear. Purulent otorrhea may begin in 24 to 48 h, particularly if water enters the middle ear.

Diagnosis

• Otoscopy

• Audiometry

Perforation is generally evident on otoscopy. Any blood obscuring the ear canal is carefully suctioned. Irrigation and pneumatic otoscopy are avoided. Extremely small perforations may require otomicroscopy or middle ear impedance studies for definitive diagnosis. If possible, audiometric studies are done before and after treatment to avoid confusion between trauma-induced and treatment-induced hearing loss.

Patients with marked hearing loss or severe vertigo are evaluated by an otolaryngologist as soon as possible. Exploratory tympanotomy may be needed to assess and repair damage. Patients with a large TM defect should also be evaluated, because the displaced flaps may need to be repositioned.

Treatment

• Ear kept dry

• Oral or topical antibiotics if dirty injury

Often, no specific treatment is needed. The ear should be kept dry; routine antibiotic eardrops are unnecessary. However, prophylaxis with oral broad-spectrum antibiotics or antibiotic eardrops is necessary if contaminants may have entered through the perforation as occurs in dirty injuries.

If the ear becomes infected, amoxicillin 500 mg po q 8 h is given for 7 days.

Although most perforations close spontaneously, surgery is indicated for a perforation persisting > 2 mo. Persistent conductive hearing loss suggests disruption of the ossicular chain, necessitating surgical exploration and repair.

Chapter 50. External Ear Disorders

Introduction

The external ear (pinna and external auditory canal) can be affected by congenital, dermatologic, infectious, neoplastic, obstructive, and traumatic disorders. Congenital defects are discussed on p. 2971. Ear trauma is discussed on p. 3231.

Dermatitis

Dermatitis is inflammation of the ear canal involving itching and skin changes that are caused by exposure to allergens (contact dermatitis) or are spontaneous occurrences (aural eczematoid dermatitis).

Common contact allergens include nickel-containing earrings and numerous beauty products (eg, hairsprays, lotions, hair dye). Aural eczematoid dermatitis is more common among people with a predisposition toward atopy and with other similar dermatitides (eg, seborrhea, psoriasis).

Both contact dermatitis and aural eczematoid dermatitis cause itching, redness, discharge, desquamation, hyperpigmentation, and, sometimes, fissuring. A secondary infection can occur.

Contact dermatitis requires avoidance or withdrawal of allergic triggers. Trial and error may be needed to identify the offending agent. Topical corticosteroids (eg, 1% hydrocortisone cream) can decrease inflammation and itching.

Aural eczematoid dermatitis can be treated with dilute aluminum acetate solution (Burow's solution), which can be applied as often as required for comfort. Itching and inflammation can be reduced with topical corticosteroids. If diffuse external otitis ensues, antibiotic therapy may be required (see p. 455).

External Otitis

External otitis is infection of the ear canal, typically by bacteria. Symptoms include itching, pain, and discharge. Diagnosis is based on inspection. Treatment is with topical drugs, including antibiotics, corticosteroids, and acetic acid or a combination.

External otitis may manifest as a localized furuncle or as a diffuse infection of the entire canal (generalized or diffuse external otitis). This condition is often called swimmer's ear because it sometimes afflicts people who swim. Malignant external otitis (see p. 455) is a severe *Pseudomonas* infection of the temporal bone and is especially dangerous in diabetics.

Etiology

Diffuse external otitis is usually caused by bacteria, such as *Pseudomonas aeruginosa*, *Proteus vulgaris*, *Staphylococcus aureus*, or *Escherichia coli*. Fungal external otitis (otomycosis), typically caused by *Aspergillus niger* or *Candida albicans*, is less common. Furuncles usually are due to *S. aureus*.

Predisposing conditions include allergies, psoriasis, eczema, seborrheic dermatitis, decreased canal acidity (possibly due to the repeated presence of water), irritants (eg, hair spray, hair dye), and inadvertent injury to the canal caused by excessive cleaning with cotton swabs or other objects. Attempts to clean the ear canal may push debris and cerumen deeper into the canal; these accumulated substances tend to trap water, resulting in skin maceration that sets the stage for bacterial infection.

Symptoms and Signs

Patients have itching and pain. Sometimes, a foul-smelling discharge and hearing loss occur if the canal becomes swollen or filled with purulent debris. Exquisite tenderness accompanies traction of the pinna or pressure over the tragus. Otoscopic examination is painful and difficult to conduct. It shows the ear canal

to be red, swollen, and littered with moist, purulent debris. Otomycosis caused by *A. niger* usually manifests with grayish black or yellow dots (fungal conidiophores) surrounded by a cottonlike material (fungal hyphae). Infection caused by *C. albicans* does not show any visible fungi but usually contains a thickened, creamy white exudate.

Furuncles cause severe pain and may drain sanguineous, purulent material. They appear as a focal, erythematous swelling.

Diagnosis

• Clinical evaluation

Diagnosis is based on inspection. When discharge is copious, external otitis can be difficult to differentiate from perforated otitis media; pain with pulling on the pinna may indicate an external otitis. Fungal infection is diagnosed by appearance or culture.

Treatment

• Topical acetic acid and corticosteroids

• Sometimes topical antibiotics

In diffuse external otitis, topical antibiotics and corticosteroids are effective. First, the infected debris should be gently and thoroughly removed from the canal with suction or dry cotton wipes. Mild external otitis can be treated by altering the ear canal's pH with 2% acetic acid and by relieving inflammation with topical hydrocortisone; these are given as 5 drops tid for 7 days. Moderate external otitis requires the addition of an antibacterial solution or suspension, such as neomycin, polymyxin, ciprofloxacin, or ofloxacin. When inflammation of the ear canal is relatively severe, an ear wick should be placed into the ear canal and wetted with the necessary drugs 4 times/day. The wick is left in place for 24 to 72 h, after which time the swelling may have receded enough to allow the instillation of drops directly into the canal.

Severe external otitis or the presence of cellulitis extending beyond the ear canal may require systemic antibiotics, such as cephalexin 500 mg po tid for 10 days or ciprofloxacin 500 mg po bid for 10 days. An analgesic, such as an NSAID or even an oral opioid, may be necessary for the first 24 to 48 h. Fungal external otitis requires thorough cleaning of the ear canal and application of an antimycotic solution (eg, gentian violet, cresylate acetate, nystatin, clotrimazole). Repeated cleanings and treatments may be needed.

A furuncle, if obviously pointing, should be incised and drained. Incision is of little value, however, if the patient is seen at an early stage. Topical antibiotics are ineffective; oral antistaphylococcal antibiotics should be given. Analgesics, such as oxycodone with acetaminophen, may be necessary for pain relief. Dry heat can also lessen pain and hasten resolution.

Prevention

External otitis often can 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.

Malignant External Otitis

Malignant external otitis is typically a *Pseudomonas* osteomyelitis of the temporal bone.

Soft tissue, cartilage, and bone are all affected. The osteomyelitis spreads along the base of the skull and may cross the midline.

Malignant external otitis occurs mainly in elderly patients with diabetes or in immunocompromised patients and is often initiated by *Pseudomonas* external otitis. It is characterized by persistent and severe earache,

foul-smelling purulent otorrhea, and granulation tissue in the ear canal (usually at the junction of the bony and cartilaginous portions of the canal). Varying degrees of conductive hearing loss may occur. In severe cases, facial nerve paralysis may ensue.

Diagnosis is based on a CT scan of the temporal bone, which may show increased radiodensity in the air-cell system and middle ear radiolucency (demineralization) in some areas. Cultures are done, and the ear canal is biopsied to differentiate the granulation tissue of this disorder from a malignant tumor.

Treatment is with a 6-wk IV course of a fluoroquinolone or an aminoglycoside-semisynthetic penicillin combination. Extensive bone disease may require more prolonged antibiotic therapy. Careful control of diabetes is essential. Surgery usually is not necessary.

Obstructions

The ear canal may be obstructed by cerumen (earwax), insertion of a foreign object, or an insect. Itching, pain, and temporary conductive hearing loss may result. Most causes of obstruction are readily apparent during otoscopic examination. Treatment is manual removal.

Cerumen: Cerumen may get pushed further into the ear canal and accumulate during ill-advised attempts to clean the ear canal with cotton swabs, resulting in obstruction. Cerumen solvents (hydrogen peroxide, carbamide peroxide, glycerin, triethanolamine) may be used to soften very hard wax before irrigation or direct removal. However, the prolonged use of these agents may lead to canal skin irritation or allergic reactions. Although cerumen may be removed by irrigation, rolling the cerumen out of the ear canal with a blunt curet or loop or removing it with a suction tip (eg, Baron, size 7 French) is quicker, neater, 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.

Foreign bodies: Foreign bodies are common, particularly among children, who often insert objects, particularly beads, erasers, and beans, into the ear canal. Foreign bodies may remain unnoticed until they provoke an inflammatory response, causing pain, itching, infection, and foul-smelling, purulent drainage. A foreign body in the ear canal is best removed by reaching behind it and rolling it out with a blunt hook. Forceps tend to push smooth objects deeper into the canal. Unfortunately, a foreign body lying medial to the isthmus (the bony cartilaginous junction of the external auditory canal) is difficult to remove without injuring the tympanic membrane and ossicular chain. Metal and glass beads can sometimes be removed by irrigation, but hygroscopic foreign bodies (eg, beans or other vegetable matter) swell when water is added, complicating removal. A general anesthetic may be needed when a child cannot remain still or when removal is difficult, threatening injury to the tympanic membrane or ossicles. Further, if manipulating a presumed foreign object results in bleeding, immediate otolaryngologic consultation should be sought. Bleeding may indicate a mucosal polyp originating in the middle ear, which may be attached to the ossicles or facial nerve.

Insects in the canal are most annoying while alive. Filling the canal with viscous lidocaine kills the insect, which provides immediate relief and allows the immobilized insect to be removed with forceps.

Perichondritis

Perichondritis is infection of the perichondrium of the pinna in which pus accumulates between the cartilage and the perichondrium.

Causes of perichondritis include trauma, insect bites, body piercings, and incision of superficial infections of the pinna. Because the cartilage's blood supply is provided by the perichondrium, separation of the perichondrium from both sides of the cartilage may lead to avascular necrosis and a deformed pinna. Septic necrosis may also ensue, often with infection by gram-negative bacilli. Symptoms include redness, pain, and swelling. The course of perichondritis tends to be indolent, long-term, and destructive.

The affected area is incised, and a drain is left in place for 24 to 72 h. Systemic antibiotics are initiated with an aminoglycoside and semisynthetic penicillin. Subsequent antibiotic choice is guided by culture

and sensitivity tests. Warm compresses may help.

Chapter 51. Approach to the Patient With Nasal and Pharyngeal Symptoms

Introduction

The nose and pharynx (consisting of the nasopharynx, oropharynx, and hypopharynx) may be affected by inflammation, infection, trauma, tumors, and several miscellaneous conditions.

Anatomy

Throat: The uvula hangs in the midline at the far end of the soft palate. It varies greatly in length. A long uvula and loose or excess velopharyngeal tissue may cause snoring and occasionally contribute to obstructive sleep apnea.

Tonsils and adenoids are patches of lymphoid tissue surrounding the posterior pharynx in an area termed Waldeyer's ring. Their role is to combat infection.

The larynx is discussed in Ch. 54.

Nose: The nasal cavity is covered with a highly vascular mucosa that warms and humidifies incoming air. Each lateral wall of the cavity has 3 turbinates, which are bony shelves that increase the surface area, thereby allowing more effective heat and moisture exchange. Nasal mucus traps incoming particulate matter. The space between the middle and inferior turbinate is the middle meatus, into which the maxillary and most of the ethmoid sinuses drain. Polyps may develop between the turbinates, often in association with asthma, allergy, aspirin use, and cystic fibrosis.

Sinuses: The paranasal sinuses are mucus-lined bony cavities that connect to the nasopharynx. The 4 types are maxillary, frontal, ethmoid, and sphenoid sinuses. They are located in the facial and cranial bones (see
Fig. 51-1). The physiologic role of the sinuses is unclear.

[**Fig. 51-1.** Paranasal sinuses.]

Evaluation

Examination of the nose and pharynx is part of every general physical examination.

History: General information includes use of alcohol or tobacco (both major risk factors for head and neck cancer) and systemic symptoms, such as fever and weight loss. Oropharyngeal symptoms include pain, ulcers, and difficulty swallowing or speaking. Nasal and sinus symptoms include presence and duration of congestion, discharge, or bleeding.

Physical examination: Most physicians use a head-mounted light. However, because the light cannot be precisely aligned on the axis of vision, it is difficult to avoid shadowing in narrow areas (eg, nasal cavity). Better illumination results with a head-mounted convex mirror; the physician looks through a hole in the center of the mirror, so the illumination is always on-axis. The head mirror reflects light from a source (any incandescent light) placed behind the patient and slightly to one side and requires practice to use effectively.

The nose is examined using a nasal speculum, which is held so that the 2 blades open in an anteroposterior (or slightly oblique) direction and do not press against the septum. The physician notes crusting, discharge, septal deviation, or perforation; whether mucosa is erythematous, boggy, or swollen; and presence of polyps. The skin over the frontal and maxillary sinuses is examined for erythema and tenderness, suggesting sinus inflammation.

If necessary, the nasopharynx and hypopharynx can be examined with mirrors, which should be warmed before use to avoid fogging. A small mirror is used for the nasopharynx. It is held just below the uvula, angling upward; the tongue is pushed down with a tongue blade. A larger mirror is used for the hypopharynx and larynx. The tongue is retracted by grasping it with a gauze pad, and the mirror is placed

against the soft palate, angling downward. If patients do not tolerate mirror examination, a flexible fiberoptic nasopharyngoscope is helpful. A topical anesthetic (eg, lidocaine 4%) is sprayed in the nose and throat, and the nose is also sprayed with a decongestant (eg, phenylephrine 0.5%). After several minutes, the scope is gently passed through the nares, and the nasal cavity, hypopharynx, and larynx are inspected.

Neck examination consists of inspection and palpation for masses. If masses are found, the physician notes whether they are tender; fluctuant, firm, or stony hard; and movable or fixed. Masses caused by infection are tender and mobile; cancers tend to be nontender, hard, and fixed. Particular attention is paid to the cervical lymph nodes and thyroid and parotid glands.

Epistaxis

Epistaxis is nose bleeding. Bleeding can range from a trickle to a strong flow, and the consequences can range from a minor annoyance to life-threatening hemorrhage. Swallowed blood is a gastric irritant, so patients also may describe vomiting blood.

Pathophysiology

Most nasal bleeding is anterior, originating from a plexus of vessels in the anteroinferior septum (Kiesselbach's area).

Less common but more serious are posterior nosebleeds, which originate in the posterior septum overlying the vomer bone, or laterally on the inferior or middle turbinate. Posterior nosebleeds tend to occur in patients who have preexisting atherosclerotic vessels or bleeding disorders and have undergone nasal or sinus surgery.

Etiology

The most common causes of epistaxis are

• Local trauma (eg, nose blowing and picking)

• Drying of the nasal mucosa

There are a number of less common causes (see Table 51-1). Hypertension may contribute to the persistence of a nosebleed that has already begun but is unlikely to be the sole etiology.

Evaluation

History: History of present illness should try to determine which side began bleeding first; although major epistaxis quickly involves both nares, most patients can localize the initial flow to one side, which focuses the physical examination. Also, the duration of bleeding should be established, as well as any triggers (eg, sneezing, nose blowing, picking) and attempts by the patient to stop the bleeding. Important associated symptoms prior to onset include symptoms of a URI, sensation of nasal obstruction, and nasal or facial pain. The time and number of previous nose-bleeding episodes and their resolution should be identified.

[**Table 51-1.** Some Causes of Epistaxis]

Review of systems should ask about symptoms of excessive bleeding, including easy bruising; bloody or tarry stools; hemoptysis; blood in urine; and excess bleeding with toothbrushing, phlebotomy, or minor trauma.

Past medical history should note presence of known bleeding disorders (including a family history) and conditions associated with defects in platelets or coagulation, particularly cancer, cirrhosis, HIV, and pregnancy. Drug history should specifically query about use of drugs that may promote bleeding,

including aspirin and other NSAIDs, other antiplatelet drugs (eg, clopidogrel), heparin, and warfarin.

Physical examination: Vital signs should be reviewed for indications of intravascular volume depletion (tachycardia, hypotension) and marked hypertension. With active bleeding, treatment takes place simultaneously with evaluation.

During active bleeding, inspection is difficult, so attempts are first made to stop the bleeding as described below. The nose is then examined using a nasal speculum and a bright head lamp or head mirror, which leaves one hand free to manipulate suction or an instrument.

Anterior bleeding sites are usually apparent on direct examination. If no site is apparent and there have been only 1 or 2 minor nosebleeds, further examination is not needed. If bleeding is severe or recurrent and no site is seen, fiberoptic endoscopy may be necessary.

The general examination should look for signs of bleeding disorders, including petechiae, purpura, and perioral and oral mucosal telangiectasias as well as any intranasal masses.

Red flags: The following findings are of particular concern:

• Signs of hypovolemia or hemorrhagic shock

• Anticoagulant drug use

• Cutaneous signs of a bleeding disorder

• Bleeding not stopped by direct pressure or vasoconstrictor-soaked pledgets

• Multiple recurrences, particularly with no clear cause

Interpretation of findings: Many cases have a clear-cut trigger (particularly nose blowing or picking) as suggested by findings (see Table 51-1).

Testing: Routine laboratory testing is not required. Patients with symptoms or signs of a bleeding disorder and those with severe or recurrent epistaxis should have CBC, PT, and PTT.

CT may be done if a foreign body, a tumor, or sinusitis is suspected.

Treatment

Presumptive treatment for actively bleeding patients is that for anterior bleeding. The need for blood replacement is determined by the Hb level, symptoms of anemia, and vital signs. Any identified bleeding disorders are treated.

Anterior epistaxis: Bleeding can usually be controlled by pinching the nasal alae together for 10 min while the patient sits upright (if possible). If this maneuver fails, a cotton pledget impregnated with a vasoconstrictor (eg, phenylephrine 0.25%) and a topical anesthetic (eg, lidocaine 2%) is inserted and the nose pinched for another 10 min. The bleeding point may then be cauterized with electrocautery or silver nitrate on an applicator stick. Cauterizing 4 quadrants immediately adjacent to the bleeding vessel is most effective. Care must be taken to avoid burning the mucous membrane too deeply; therefore, silver nitrate is the preferred method. Alternatively, a nasal tampon of expandable foam may be inserted. Coating the tampon with a topical ointment, such as bacitracin or mupirocin, may help. If these methods are ineffective, various commercial nasal balloons can be used to compress bleeding sites. Alternatively, an anterior nasal pack consisting of 1/2-in petrolatum gauze may be inserted; up to 72 in of gauze may be required. This procedure is painful, and analgesics usually are needed; it should be used only when other methods fail or are not available.

Posterior epistaxis: Posterior bleeding may be difficult to control. Commercial nasal balloons are quick and convenient; a gauze posterior pack is effective but more difficult to position. Both are very

uncomfortable; IV sedation and analgesia may be needed, and hospitalization is required.

Commercial balloons are inserted according to the instructions accompanying the product.

The posterior gauze pack consists of 4-in gauze squares folded, rolled, tied into a tight bundle with 2 strands of heavy silk suture, and coated with antibiotic ointment. 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 pulled into place above the soft palate in the nasopharynx. The 2nd suture hangs down the back of the throat and is trimmed below the level of the soft palate so that it can be used to remove the pack. The nasal cavity anterior to this pack is firmly packed with 1/2-in petrolatum gauze, and the 1st suture is tied over a roll of gauze at the anterior nares to secure the postnasal pack. The packing remains in place for 4 to 5 days. An antibiotic (eg, amoxicillin/clavulanate 875 mg po bid for 7 to 10 days) is given to prevent sinusitis and otitis media. Posterior nasal packing lowers the arterial PO_2, and supplementary O_2 is given while the packing is in place.

Rarely, the internal maxillary artery and its branches must be ligated to control the bleeding. The arteries may be ligated with clips using endoscopic or microscopic guidance and a surgical approach through the maxillary sinus. Alternatively, angiographic embolization may be done by a skilled radiologist.

Bleeding disorders: In Rendu-Osler-Weber syndrome, a split-thickness skin graft (septal dermatoplasty) reduces the number of nosebleeds and allows the anemia to be corrected. Laser (Nd:YAG) photocoagulation can be done in the operating room. Selective embolization also is very effective, particularly in patients who cannot tolerate general anesthesia or for whom surgical intervention has not been successful. New endoscopic sinus devices have made transnasal surgery more effective.

Blood may be swallowed in large amounts and, in patients with liver disease, should be eliminated promptly with enemas and cathartics to prevent hepatic encephalopathy. 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.

Key Points

• Most nosebleeds are anterior and stop with direct pressure.

• Screening (by history and physical examination) for bleeding disorders is important.

• Patients should always be asked about aspirin or ibuprofen use.

Nasal Congestion and Rhinorrhea

Nasal congestion and rhinorrhea (runny nose) are extremely common problems that commonly occur together but occasionally occur alone.

Etiology

The most common causes (see Table 51-2) are the following:

• Viral infections

• Allergic reactions

Dry air may provoke congestion. Acute sinusitis is slightly less common, and a nasal foreign body is unusual (and occurs predominantly in children).

Patients who use topical decongestants for > 1 day often experience significant rebound congestion when the effects of the drug wear off, causing them to continue using the decongestant in a vicious circle

of persistent, worsening congestion. This situation (rhinitis medicamentosa) may persist for some time and may be misinterpreted as a continuation of the original problem rather than a consequence of treatment.

Evaluation

History: History of present illness should determine the nature of the discharge (eg, watery, mucoid, purulent, bloody) and whether discharge is chronic or recurrent. If recurrent, any relation to patient location, season, or exposure to potential triggering allergens (numerous) should be determined.

Review of systems should seek symptoms of possible causes, including fever and facial pain (sinusitis); watery, itchy eyes (allergies); and sore throat, malaise, fever, and cough (viral URI).

Past medical history should seek known allergies and existence of diabetes or immunocompromise. Drug history should ask specifically about topical decongestant use.

Physical examination: Vital signs are reviewed for fever.

Examination focuses on the nose and area over the sinuses. The face is inspected for focal erythema over the frontal and maxillary sinuses; these areas are also palpated for tenderness. Nasal mucosa is inspected for color (eg, red or pale), swelling, color and nature of discharge, and (particularly in children) presence of any foreign body.

Red flags: The following findings are of particular concern:

• Unilateral discharge, particularly if purulent or bloody

• Facial pain, tenderness, or both

Interpretation of findings: Symptoms and examination are often enough to suggest a diagnosis (see Table 51-2).

In children, unilateral foul-smelling discharge suggests a nasal foreign body. If no foreign body is seen, sinusitis is suspected when purulent rhinorrhea persists for > 10 days along with fatigue and cough.

Testing: Testing is generally not indicated for acute nasal symptoms unless invasive sinusitis is suspected in a diabetic or immunocompromised patient; these patients usually should undergo CT.

[**Table 51-2.** Some Causes of Nasal Congestion and Rhinorrhea]

Treatment

Specific conditions are treated. Symptomatic relief of congestion can be achieved with topical or oral decongestants. Topical decongestants include oxymetazoline, 2 sprays each nostril once/day or bid for 3 days. Oral decongestants include pseudoephedrine 60 mg bid. Prolonged use should be avoided.

Viral rhinorrhea can be treated with oral antihistamines (eg, diphenhydramine 25 to 50 mg po bid), which are recommended because of their anticholinergic properties unrelated to their H_2-blocking properties.

Allergic congestion and rhinorrhea can be treated with antihistamines; in such cases, nonanticholinergic antihistamines (eg, fexofenadine 60 mg po bid) as needed provoke fewer adverse effects. Nasal corticosteroids (eg, mometasone 2 sprays each nostril daily) also help allergic conditions.

Antihistamines and decongestants are not recommended for children < 6 yr.

Geriatrics Essentials

Antihistamines and can have sedating and anticholinergic effects and should be given in decreased

dosage in the elderly. Similarly, sympathomimetics should be used with the lowest dosage that is clinically effective.

Key Points

• Most nasal congestion and rhinorrhea are caused by URI or allergies.

• A foreign body should be considered in children.

• Rebound from topical decongestant overuse should also be considered.

Neck Mass

Patients or their family members may notice a mass on the neck, or one may be discovered during routine examination. A neck mass may be painless or painful depending on the cause. When a neck mass is painless, much time may pass before patients seek medical care.

Etiology

There are many causes of neck mass, including infectious, cancerous, and congenital causes (see Table 51-3).

The most common causes in younger patients include the following:

• Reactive adenitis

• Primary bacterial lymph node infection

• Systemic infections

[**Table 51-3.** Some Causes of Neck Mass]

Reactive adenitis occurs in response to viral or bacterial infection somewhere in the oropharynx. Some systemic infections (eg, mononucleosis, HIV, TB) cause cervical lymph node enlargement—usually generalized rather than isolated.

Congenital disorders may cause a neck mass, typically longstanding. The most common are thyroglossal duct cysts, branchial cleft cysts, and dermoid or sebaceous cysts.

Cancerous masses are more common among older patients but may occur in younger ones. These masses may represent a local primary tumor or lymph node involvement from a local, regional, or distant primary cancer. About 60% of supraclavicular triangle masses are metastases from distant primary sites. Elsewhere in the neck, 80% of cancerous cervical adenopathy originates in the upper respiratory or alimentary tract. Likely sites of origin are the posterior-lateral border of the tongue and the floor of the mouth followed by the nasopharynx, palatine tonsil, laryngeal surface of the epiglottis, and hypopharynx, including the pyriform sinuses.

The thyroid gland may enlarge in various disorders, including simple nontoxic goiter, subacute thyroiditis, and, less often, thyroid cancer.

Evaluation

History: History of present illness should note how long the mass has been present and whether it is painful. Important associated acute symptoms include sore throat, URI symptoms, and toothache.

Review of systems should ask about difficulty swallowing or speaking and symptoms of chronic disease (eg, fever, weight loss, malaise). Regional and distant cancers causing metastases to the neck occasionally cause symptoms in their system of origin (eg, cough in lung cancer, swallowing difficulty in

esophageal cancer). Because numerous cancers can metastasize to the neck, a complete review of systems is important to help identify a source.

Past medical history should inquire about known HIV or TB and risk factors for them. Risk factors for cancer are assessed, including consumption of alcohol or use of tobacco (particularly snuff or chewing tobacco), ill-fitting dental appliances, and chronic oral candidiasis. Poor oral hygiene also may be a risk.

Physical examination: The neck mass is palpated to determine consistency (ie, whether soft and fluctuant, rubbery, or hard) and presence and degree of tenderness. Whether the mass is freely mobile or appears fixed to the skin or underlying tissue also needs to be determined.

The scalp, ears, nasal cavities, oral cavity, nasopharynx, oropharynx, hypopharynx, and larynx are closely inspected for signs of infection and any other visible lesions. Teeth are percussed to detect the exquisite tenderness of root infection. The base of the tongue, floor of the mouth, and the thyroid and salivary glands are palpated for masses.

The breasts and prostate gland are palpated for masses, and the spleen is palpated for enlargement. Stool is checked for occult blood, suggestive of a GI cancer.

Other lymph nodes are palpated (eg, axillary, inguinal).

Red flags: The following findings are of particular concern:

• Hard, fixed mass

• Older patient

• Presence of oropharyngeal lesions (other than simple pharyngitis or dental infection)

• A history of persistent hoarseness or dysphagia

Interpretation of findings: Important differentiating factors for a neck mass (see also Table 51-3) include acuity, pain and tenderness, and consistency and mobility.

A new mass (ie, developing over only a few days), particularly after symptoms of a URI or pharyngitis, suggests benign reactive lymphadenopathy. An acute tender mass suggests lymphadenitis or an infected dermoid cyst.

A chronic mass in younger patients suggests a cyst. A non-midline mass in older patients, particularly those with risk factors, should be considered cancer until proven otherwise; a midline mass is likely of thyroid origin (benign or malignant).

Pain, tenderness, or both in the mass suggest inflammation (particularly infectious), whereas a painless mass suggests a cyst or tumor. A hard, fixed, nontender mass suggests cancer, whereas rubbery consistency and mobility suggest otherwise.

Generalized adenopathy and splenomegaly suggest infectious mononucleosis or a lymphoreticular cancer. Generalized adenopathy alone may suggest HIV infection, particularly in those with risk factors.

Red and white mucosal patches (erythroplakia and leukoplakia) in the oropharynx may be malignant lesions responsible for the neck mass.

Difficulty swallowing may be noted with thyroid enlargement or cancer originating in various sites in the neck. Difficulty speaking suggests a cancer involving the larynx or recurrent laryngeal nerve.

Testing: If the nature of the mass is readily apparent (eg, lymphadenopathy caused by recent pharyngitis) or is in a healthy young patient with a recent, tender swelling and no other findings, then no immediate testing is required. However, the patient is reexamined regularly; if the mass fails to resolve,

further evaluation is needed.

Most other patients should have a CBC and chest x-ray. Those with findings suggesting specific causes should also have testing for those disorders (see Table 51-3).

If examination reveals an oral or nasopharyngeal lesion that fails to begin resolving within 2 wk, testing may include CT or MRI and fine-needle biopsy of that lesion.

In young patients with no risk factors for head and neck cancer and no other apparent lesions, the neck mass may be biopsied.

Older patients, particularly those with risk factors for cancer, should first undergo further testing to identify the primary site; biopsy of the neck mass may simply reveal undifferentiated squamous cell carcinoma without illuminating the source. Such patients should have direct laryngoscopy, bronchoscopy, and esophagoscopy with biopsy of all suspicious areas. CT of the head, neck, and chest and possibly a thyroid scan are done. If a primary tumor is not found, fine-needle aspiration biopsy of the neck mass should be done, which is preferable to an incisional biopsy because it does not leave a transected mass in the neck. If the neck mass is cancerous and a primary tumor has not been identified, random biopsy of the nasopharynx, palatine tonsils, and base of the tongue should be considered.

Treatment

Treatment is directed at the cause.

Key Points

• An acute neck mass in younger patients is usually benign.

• Neck mass in an elderly patient raises concern of cancer.

• Thorough oropharyngeal examination is important.

Pharyngitis

Pharyngitis (sore throat) is pain in the posterior pharynx that occurs with or without swallowing. Pain can be severe; many patients refuse oral intake.

Etiology

Sore throat results from infection; the most common cause is

• Tonsillopharyngitis

Rarely, an abscess or epiglottitis is involved; although uncommon, these are of particular concern because they may compromise the airway.

Tonsillopharyngitis: Tonsillopharyngitis is predominantly a viral infection; a lesser number of cases are caused by bacteria.

The respiratory viruses (rhinovirus, adenovirus, influenza, coronavirus, respiratory syncytial virus) are the most common viral causes, but occasionally Epstein-Barr virus (the cause of mononucleosis), herpes simplex, cytomegalovirus, or primary HIV infection is involved.

The main bacterial cause is group A β-hemolytic streptococcus (GABHS), which, although estimates vary, causes perhaps 10% of cases in adults and slightly more in children. GABHS is a concern because of the possibility of the poststreptococcal sequelae of rheumatic fever, glomerulonephritis, and abscess. Uncommon bacterial causes include gonorrhea, diphtheria, mycoplasma, and chlamydia.

Abscess: An abscess in the pharyngeal area (peritonsillar, parapharyngeal, and, in children, retropharyngeal) is uncommon but causes significant throat pain. The usual causative organism is GABHS.

Epiglottitis: Epiglottitis, perhaps better termed supraglottitis, used to occur primarily in children and usually was caused by *Haemophilus influenzae* type B (HiB). Now, because of widespread childhood vaccination against HiB, supraglottitis/epiglottitis has been almost eradicated in children (more cases occur in adults). Causal organisms in children and adults include *Streptococcus pneumoniae*, *Staphylococcus aureus*, nontypeable *H. influenzae*, *Haemophilus parainfluenzae*, β-hemolytic streptococci, *Branhamella catarrhalis*, and *Klebsiella pneumoniae*. HiB is still a cause in adults and unvaccinated children.

Evaluation

History: History of present illness should note the duration and severity of sore throat.

Review of systems should seek important associated symptoms, such as runny nose, cough, and difficulty swallowing, speaking, or breathing. The presence and duration of any preceding weakness and malaise (suggesting mononucleosis) are noted.

Past medical history should seek history of previous documented mononucleosis (recurrence is highly unlikely). Social history should inquire about close contact with people with documented GABHS infection, risk factors for gonorrhea transmission (eg, recent oral-genital sexual contact), and risk factors for HIV acquisition (eg, unprotected intercourse, multiple sex partners, IV drug abuse).

Physical examination: General examination should note fever and signs of respiratory distress, such as tachypnea, dyspnea, stridor, and, in children, the tripod position (sitting upright, leaning forward with neck hyperextended and jaw thrust forward).

Pharyngeal examination should not be done in children if supraglottitis/epiglottitis is suspected, because it may trigger complete airway obstruction. Adults with no respiratory distress may be examined but with care. Erythema, exudates, and any signs of swelling around the tonsils or retropharyngeal area should be noted. Whether the uvula is in the midline or appears pushed to one side should also be noted.

The neck is examined for presence of enlarged, tender lymph nodes. The abdomen is palpated for presence of splenomegaly.

Red flags: The following findings are of particular concern:

• Stridor or other sign of respiratory distress

• Drooling

• Muffled, "hot potato" voice

• Visible bulge in pharynx

Interpretation of findings: Supraglottitis/epiglottitis and pharyngeal abscess pose a threat to the airway and must be differentiated from simple tonsillopharyngitis, which is uncomfortable but not acutely dangerous. Clinical findings help make this distinction.

With supraglottitis/epiglottitis, there is abrupt onset of severe throat pain and dysphagia, usually with no preceding URI symptoms. Children often have drooling and signs of toxicity. Sometimes (more often in children), there are respiratory manifestations, with tachypnea, dyspnea, stridor, and sitting in the tripod position. If examined, the pharynx almost always appears unremarkable.

Pharyngeal abscess and tonsillopharyngitis both may cause pharyngeal erythema, exudate, or both. However, some findings are more likely in one condition or another:

• Pharyngeal abscess: Muffled, "hot potato" voice (speaking as if a hot object is being held in the mouth); visible focal swelling in the posterior pharyngeal area (often with deviation of the uvula)

• Tonsillopharyngitis: Accompanied by URI symptoms (eg, runny nose, cough)

Although tonsillopharyngitis is easily recognized clinically, its cause is not. Manifestations of viral and GABHS infection overlap significantly, although URI symptoms are more common with a viral cause. In adults, clinical criteria that increase suspicion of GABHS as a cause include

• Tonsillar exudate

• Tender lymphadenopathy

• Fever (including history)

• Absence of cough

Those with ≤ 1 criterion reasonably may be presumed to have viral illness. If ≥ 2 criteria are present, the likelihood of GABHS is high enough to warrant testing but probably not high enough to warrant antibiotics, but this decision needs to be patient-specific (ie, threshold for testing and treatment may be lower in those at risk because of diabetes or immunocompromise). In children, testing usually is done.

Regarding rarer causes of tonsillopharyngitis, infectious mononucleosis should be considered when there is posterior cervical or generalized adenopathy, hepatosplenomegaly, and fatigue and malaise for > 1 wk. Those with no URI symptoms but recent oral-genital contact may have pharyngeal gonorrhea. A dirty-gray, thick, tough membrane on the posterior pharynx that bleeds if peeled away indicates diphtheria (rare in the US). HIV infection should be considered in patients with risk factors.

Testing: If supraglottitis/epiglottitis is considered possible after evaluation, testing is required. Patients who do not appear seriously ill and have no respiratory symptoms may have plain lateral neck x-rays to look for an edematous epiglottis. However, a child who appears seriously ill or has stridor or any other respiratory symptoms should not be transported to the x-ray suite. Such patients (and those with positive or equivocal x-ray findings) usually should have flexible fiberoptic laryngoscopy. (CAUTION: *Examination of the pharynx and larynx may precipitate complete respiratory obstruction in children, and the pharynx and larynx should not be directly examined except in the operating room, where the most advanced airway intervention is available*.)

Many abscesses are managed clinically, but if location and extent are unclear, immediate CT of the neck should be done.

In tonsillopharyngitis, throat culture is the only reliable way to differentiate viral infection from GABHS. To balance timeliness of diagnosis, cost, and accuracy, one strategy in children is to do a rapid strep screen in the office, treat if positive, and send a formal culture if negative. In adults, because other bacterial pathogens may be involved, throat culture for all bacterial pathogens is appropriate for those meeting clinical criteria described previously.

Testing for mononucleosis, gonorrhea, or HIV is done only when clinically suspected.

Treatment

Specific conditions are treated. Those with severe symptoms of tonsillopharyngitis may be started on a broad-spectrum antibiotic (eg, amoxicillin/clavulanate) pending culture results.

Symptomatic treatments such as warm saltwater gargles and topical anesthetics (eg, benzocaine, lidocaine, dyclonine) may help temporarily relieve pain in tonsillopharyngitis. Patients in severe pain (even from tonsillopharyngitis) may require short-term use of opioids.

Key Points

• Most sore throats are caused by viral tonsillopharyngitis.

• It is difficult to clinically distinguish viral from bacterial causes of tonsillopharyngitis.

• Abscess and epiglottitis are rare but serious causes.

• Severe sore throat in a patient with a normal-appearing pharynx should raise suspicion of epiglottitis.

Smell and Taste Abnormalities

Because distinct flavors depend on aromas to stimulate the olfactory chemoreceptors, smell and taste are physiologically interdependent. 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, although their effect on quality of life can be severe.

Taste: Although abnormal taste sensations may be due to mental disorders, local causes should always be sought. Glossopharyngeal and facial nerve integrity can be determined by testing taste on both sides of the dorsum of the tongue with sugar, salt, vinegar (acid), and quinine (bitter).

Drying of the oral mucosa caused by heavy smoking, Sjogren's syndrome, radiation therapy of the head and neck, or desquamation of the tongue can impair taste, and various drugs (eg, those with anticholinergic properties and 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.

Smell: The inability to detect certain odors, such as gas or smoke, may be dangerous, and several systemic and intracranial disorders should be excluded 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 usually take precedence.

Anosmia (loss of the sense of smell) is probably the most common abnormality. Hyperosmia (increased sensitivity to odors) usually reflects a neurotic or histrionic personality but can occur intermittently with seizure disorders. Dysosmia (disagreeable or distorted sense of smell) may occur with infection of the nasal sinuses, partial damage to the olfactory bulbs, or mental 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.

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

Anosmia

Anosmia is complete loss of smell. Hyposmia is partial loss of smell. Most patients with anosmia have normal perception of salty, sweet, sour, and bitter substances but lack flavor discrimination, which largely depends on olfaction. Therefore, they often complain of losing the sense of taste (ageusia) and of not enjoying food. If unilateral, anosmia is often unrecognized.

Etiology

Anosmia occurs when intranasal swelling or other obstruction prevents odors from gaining access to the olfactory area; when the olfactory neuroepithelium is destroyed; or when the olfactory nerve fila, bulbs, tracts, or central connections are destroyed (see Table 51-4).

Major causes include

• Head trauma (young adults)

• Viral infections and Alzheimer's disease (older adults)

[**Table 51-4.** Some Causes of Anosmia]

Prior URI, especially influenza infection, is implicated in 14 to 26% of all presenting cases of hyposmia or anosmia.

Drugs can contribute to anosmia in susceptible patients. Other causes include prior head and neck radiation, recent nasal or sinus surgery, nasal and brain tumors, and toxins. The role of tobacco is uncertain.

Evaluation

History: History of present illness should assess the time course of symptoms and their relation to any URI or head injury. Important associated symptoms are nasal congestion, rhinorrhea, or both. The nature of rhinorrhea should be assessed (eg, watery, mucoid, purulent, bloody).

Review of systems should assess neurologic symptoms, particularly those involving mental status (eg, difficulty with recent memory) and cranial nerves (eg, diplopia, difficulty speaking or swallowing, tinnitus, vertigo).

Past medical history should include history of sinus disorders, cranial trauma or surgery, allergies, drugs used, and exposure to chemicals or fumes.

Physical examination: The nasal passages should be inspected for swelling, inflammation, discharge, and polyps. Having the patient breathe through each nostril sequentially (while the other is manually occluded) may help identify obstruction.

A complete neurologic examination, particularly of mental status and cranial nerves, is done.

Red flags: The following findings are of particular concern:

• Previous head injury

• Neurologic symptoms or signs

• Sudden onset

Interpretation of findings: Sudden onset after significant head trauma or toxin exposure strongly implicates that event as the cause.

A history of chronic rhinosinusitis is suggestive, particularly when significant congestion, polyps, or both are visible on examination. However, because these findings are common in the population, the physician should be wary of missing another disorder. Progressive confusion and recent memory loss in an elderly patient suggest Alzheimer's disease as a cause. Waxing and waning neurologic symptoms affecting multiple areas suggest a neurodegenerative disease such as multiple sclerosis. Slowly progressive anosmia in an elderly patient with no other symptoms or findings suggests normal aging as the cause.

Testing: An in-office test of olfaction can help confirm olfactory dysfunction. Commonly, one nostril is pressed shut, and a pungent odor such as from a vial containing coffee, cinnamon, or tobacco is placed under the open nostril; if the patient can identify the substance, olfaction is presumed intact. The test is repeated on the other nostril to determine whether the response is bilateral. Unfortunately, the test is crude and unreliable.

If anosmia is present and no cause is readily apparent on clinical evaluation (see Table 51-4), patients

should have CT of the head (including sinuses) with contrast to rule out a tumor or unsuspected fracture of the floor of the anterior cranial fossa. MRI is also used to evaluate intracranial disease and may be needed as well, particularly in those patients with no nasal or sinus pathology on CT.

A psychophysical assessment of odor and taste identification and threshold detection is done as well. This assessment commonly involves use of one or several commercially available testing kits. One kit uses a scratch-and-sniff battery of odors, whereas another kit involves sequential dilutions of an odorous chemical.

Treatment

Specific causes are treated, although smell does not always recover even after successful treatment of sinusitis.

There are no treatments for anosmia. Patients who retain some sense of smell may find adding concentrated flavoring agents to food improves their enjoyment of eating. Smoke alarms, important in all homes, are even more essential for patients with anosmia. Patients should be cautioned about consumption of stored food and use of natural gas for cooking or heating, because they have difficulty detecting food spoilage or gas leaks.

Geriatrics Essentials

There is a significant loss of olfactory receptor neurons with normal aging, leading to a marked diminution of the sense of smell. Changes are usually noticeable by age 60 and can be marked after age 70.

Key Points

• Anosmia may be part of normal aging.

• Common causes include URI, sinusitis, and head trauma.

• Cranial imaging is typically required unless the cause is obvious.

Chapter 52. Oral and Pharyngeal Disorders

Introduction

Oral and pharyngeal disorders include adenoid disorders, epiglottitis, parapharyngeal abscess, peritonsillar abscess and cellulitis, retropharyngeal abscess, salivary stones, sialadenitis, submandibular space infection, tonsillopharyngitis, Tornwaldt's cyst, and velopharyngeal insufficiency. Oral, pharyngeal, and salivary gland tumors are discussed in Ch. 55.

Sialadenitis

Sialadenitis is bacterial infection of a salivary gland, usually due to an obstructing stone or gland hyposecretion. Symptoms are swelling, pain, redness, and tenderness. Diagnosis is clinical. CT, ultrasound, and MRI may help identify the cause. Treatment is with antibiotics.

Etiology

Sialadenitis usually occurs after hyposecretion or duct obstruction but may develop without an obvious cause. The major salivary glands are the parotid, submandibular, and sublingual glands. Sialadenitis is most common in the parotid gland and typically occurs in patients in their 50s and 60s, in chronically ill patients with xerostomia, in those with Sjogren's syndrome, and in those who have had radiation therapy to the oral cavity. Teenagers and young adults with anorexia are also prone to this disorder. The most common causative organism is *Staphylococcus aureus*; others include streptococci, coliforms, and various anaerobic bacteria.

Symptoms and Signs

Fever, chills, and unilateral pain and swelling develop. The gland is firm and diffusely tender, with erythema and edema of the overlying skin. Pus can often be expressed from the duct by compressing the affected gland and should be cultured. Focal enlargement may indicate an abscess.

Diagnosis

CT, ultrasound, and MRI can confirm sialadenitis or abscess that is not obvious clinically, although MRI may miss an obstructing stone.

Treatment

• Antistaphylococcal antibiotics

• Local measures (eg, sialagogues, warm compresses)

Initial treatment is with antibiotics active against *S. aureus* (eg, dicloxacillin, 250 mg po qid, a 1st-generation cephalosporin, or clindamycin), modified according to culture results. With the increasing prevalence of methicillin-resistant *S. aureus*, especially among the elderly living in extended-care nursing facilities, vancomycin is often required. Hydration, sialagogues (eg, lemon juice, hard candy, or some other substance that triggers saliva flow), warm compresses, gland massage, and good oral hygiene are also important. Abscesses require drainage. Occasionally, a superficial parotidectomy or submandibular gland excision is indicated for patients with chronic or relapsing sialadenitis.

Other Salivary Gland Infections

Mumps often cause parotid swelling (see Table 155-1 on p. 1462). Patients with HIV infection often have parotid enlargement secondary to one or more lymphoepithelial cysts. Cat-scratch disease caused by *Bartonella* infection often invades periparotid lymph nodes and may infect the parotid glands by contiguous spread. Although cat-scratch disease is self-limited, antibiotic therapy is often provided, and incision and drainage are necessary if an abscess

develops.

Atypical mycobacterial infections in the tonsils or teeth may spread contiguously to the major salivary glands. The PPD may be negative, and the diagnosis may require biopsy and tissue culture for acid-fast bacteria. Treatment recommendations are controversial. Options include surgical debridement with curettage, complete excision of the infected tissue, and use of anti-TB drug therapy (rarely necessary).

Salivary Stones

(Sialolithiasis)

Stones composed of Ca salts often obstruct salivary glands, causing pain, swelling, and sometimes infection. Diagnosis is made clinically or with CT, ultrasonography, or sialography. Treatment involves stone expression with saliva stimulants, manual manipulation, a probe, or surgery.

The major salivary glands are the paired parotid, submandibular, and sublingual glands. Stones in the salivary glands are most common among adults. Eighty percent of stones originate in the submandibular glands and obstruct Wharton's duct. Most of the rest originate in the parotid glands and block Stensen's duct. Only about 1% originate in the sublingual glands. Multiple stones occur in about 25% of patients.

Etiology

Most salivary stones are composed of Ca phosphate with small amounts of Mg and carbonate. Patients with gout may have uric acid stones. Stone formation requires a nidus on which salts can precipitate during salivary stasis. Stasis occurs in patients who are debilitated, dehydrated, have reduced food intake, or take anticholinergics. Persisting or recurrent stones predispose to infection of the involved gland (sialadenitis—see p. 469).

Symptoms and Signs

Obstructing stones cause glandular swelling and pain, particularly after eating, which stimulates saliva flow. Symptoms may subside after a few hours. Relief may coincide with a gush of saliva. Some stones cause intermittent or no symptoms. If a stone is lodged distally, it may be visible or palpable at the duct's outlet.

Diagnosis

• Clinical evaluation

• Sometimes imaging (eg, CT, ultrasonography, sialography)

If a stone is not apparent on examination, the patient can be given a sialagogue (eg, lemon juice, hard candy, or some other substance that triggers saliva flow). Reproduction of symptoms is almost always diagnostic of a stone. CT, ultrasonography, and sialography are highly sensitive and are used if clinical diagnosis is equivocal. Contrast sialography may be done through a catheter inserted into the duct and can differentiate between stone, stenosis, and tumor. This technique is occasionally therapeutic. Because 90% of submandibular calculi are radiopaque and 90% of parotid calculi are radiolucent, plain x-rays are not always accurate. MRI is not indicated.

Treatment

• Local measures (eg, sialagogues, massage)

• Sometimes manual expression or surgical removal

Analgesics, hydration, and massage can relieve symptoms. Antistaphylococcal antibiotics can be used to prevent acute sialadenitis if started early. Stones may pass spontaneously or when salivary flow is

stimulated by sialagogues; patients are encouraged to suck a lemon wedge or sour candy every 2 to 3 h. Stones right at the duct orifice can sometimes be expressed manually by squeezing with the fingertips. Dilation of the duct with a small probe may facilitate expulsion. Surgical removal of stones succeeds if other methods fail. Stones at or near the orifice of the duct may be removed transorally, whereas those in the hilum of the gland often require complete excision of the salivary gland.

Submandibular Space Infection

(Ludwig's Angina)

Submandibular space infection is acute cellulitis of the soft tissues below the mouth. Symptoms include pain, dysphagia, and potentially fatal airway obstruction. Diagnosis usually is clinical. Treatment includes airway management, surgical drainage, and IV antibiotics.

Submandibular space infection is a rapidly spreading, bilateral, indurated cellulitis occurring in the suprahyoid soft tissues, the floor of the mouth, and both sublingual and submaxillary spaces without abscess formation. Although not a true abscess, it resembles one clinically and is treated similarly.

The condition usually develops from an odontogenic infection, especially of the 2nd and 3rd mandibular molars, or as an extension of peritonsillar cellulitis. Contributing factors may include poor dental hygiene, tooth extractions, and trauma (eg, fractures of the mandible, lacerations of the floor of the mouth).

Symptoms and Signs

Early manifestations are pain in any involved teeth, with severe, tender, localized submental and sublingual induration. Board-like firmness of the floor of the mouth and brawny induration of the suprahyoid soft tissues may develop rapidly. Drooling, trismus, dysphagia, stridor caused by laryngeal edema, and elevation of the posterior tongue against the palate may be present. Fever, chills, and tachycardia are usually present as well. The condition can cause airway obstruction within hours and does so more often than do other neck infections.

Diagnosis

The diagnosis usually is obvious. If not, CT is done.

Treatment

• Maintenance of airway patency

• Surgical incision and drainage

• Antibiotics active against oral flora

Maintaining airway patency is of the highest priority. Because swelling makes oral endotracheal intubation difficult, fiberoptic nasotracheal intubation done with topical anesthesia in the operating room or ICU with the patient awake is preferable. Some patients require a tracheotomy. Patients without immediate need for intubation require intense observation and may benefit temporarily from a nasal trumpet.

Incision and drainage with placement of drains deep into the mylohyoid muscles relieve the pressure. Antibiotics should be chosen to cover both oral anaerobes and aerobes (eg, clindamycin, ampicillin/sulbactam, high-dose penicillin).

Adenoid Disorders

Hypertrophy or inflammation of the adenoids is common among children. Symptoms include nasal obstruction, sleep disturbances, and middle ear effusions with hearing loss. Diagnosis is enhanced by flexible fiberoptic nasopharyngoscopy. Treatment often includes intranasal corticosteroids, antibiotics, and, for significant nasal obstruction or persistent recurrent acute

otitis media or middle ear effusion, adenoidectomy.

The adenoids are a rectangular mass of lymphatic tissue in the posterior nasopharynx. They are largest in children 2 to 6 yr. Enlargement may be physiologic or secondary to viral or bacterial infection, allergy, irritants, and, possibly, gastroesophageal reflux. Other risk factors include ongoing exposure to bacterial or viral infection (eg, to multiple children at a child care center). Severe hypertrophy can obstruct the eustachian tubes (causing otitis media), posterior choanae (causing sinusitis), or both.

Symptoms and Signs

Although patients with adenoid hypertrophy may not complain of symptoms, they usually have chronic mouth breathing, snoring, sleep disturbance, halitosis, recurrent acute otitis media, conductive hearing loss (secondary to recurrent otitis media or persistent middle ear effusions), and a hyponasal voice quality. Chronic adenoiditis can also cause chronic or recurrent nasopharyngitis, rhinosinusitis, epistaxis, halitosis, and cough.

Diagnosis

• Flexible nasopharyngoscopy

Adenoid hypertrophy is suspected in children and adolescents with characteristic symptoms, persistent middle ear effusions, or recurrent acute otitis media or rhinosinusitis. Similar symptoms and signs in a male adolescent may result from an angiofibroma. The gold standard for office assessment of the nasopharynx is flexible nasopharyngoscopy. X-ray imaging and sleep tape recording, although also often used, are not as accurate. A sleep study may help define the severity of any sleep disturbance due to chronic obstruction.

Treatment

• Treatment of cause

• Sometimes adenoidectomy

Underlying allergy is treated with intranasal corticosteroids, and underlying bacterial infection is treated with antibiotics. In children with persistent middle ear effusions or frequent otitis media, adenoidectomy often limits recurrence. Children > 4 yr who require tympanostomy tubes often undergo adenoidectomy when tubes are placed. Surgery is also recommended for younger children with recurrent epistaxis or significant nasal obstruction (eg, sleep disturbance, voice change). Although it requires general anesthesia, adenoidectomy usually can be done on an outpatient basis with recovery in 48 to 72 h.

Retropharyngeal Abscess

Retropharyngeal abscesses, most common among young children, can cause sore throat, fever, neck stiffness, and stridor. Diagnosis requires lateral neck x-ray or CT. Treatment is with endotracheal intubation, drainage, and antibiotics.

Retropharyngeal abscesses develop in the retropharyngeal lymph nodes at the back of the pharynx, adjacent to the vertebrae. They can be seeded by infection of the pharynx, sinuses, adenoids, or nose. They occur mainly in children 1 to 8 yr, as the retropharyngeal lymph nodes begin to recede by 4 to 5 yr. However, adults may develop infection after foreign body ingestion or after instrumentation. Common organisms include aerobic (*Streptococcus* and *Staphylococcus* sp) and anaerobic (*Bacteroides* and *Fusobacterium*) bacteria and, increasingly in adults and children, HIV and TB.

The most serious consequences include airway obstruction, septic shock, rupture of the abscess into the airway resulting in aspiration pneumonia or asphyxia, mediastinitis, carotid rupture, and suppurative thrombophlebitis of the internal jugular veins (Lemierre syndrome).

Symptoms and Signs

Symptoms and signs are usually preceded in children by an acute URI and in adults by foreign body ingestion or instrumentation. Children may have odynophagia, dysphagia, fever, cervical lymphadenopathy, nuchal rigidity, stridor, dyspnea, snoring or noisy breathing, and torticollis. Adults may have severe neck pain but less often have stridor. The posterior pharyngeal wall may bulge to one side.

Diagnosis

• CT

Diagnosis is suspected in patients with severe, unexplained sore throat and neck stiffness; stridor; or noisy breathing. Lateral soft-tissue x-rays of the neck, taken in the maximum possible hyperextension and during inspiration, may show focal widening of the prevertebral soft tissues, reversal of normal cervical lordosis, air in the prevertebral soft tissues, or erosion of the adjacent vertebral body. CT can help diagnose questionable cases, help differentiate cellulitis from an abscess, and assess extent of the abscess.

Treatment

• Antibiotics (eg, ceftriaxone, clindamycin)

• Usually surgical drainage

Antibiotics, such as a broad-spectrum cephalosporin (eg, ceftriaxone 50 to 75 mg/kg IV once/day) or clindamycin, may occasionally be sufficient for children with small abscesses. However, most patients also require drainage through an incision in the posterior pharyngeal wall. Endotracheal intubation is done preoperatively and maintained for 24 to 48 h.

Tornwaldt's Cyst

(Pharyngeal Bursa)

Tornwaldt's cyst is a rare cyst in the midline of the nasopharynx that may become infected.

Tornwaldt's cyst is a remnant of the embryonal notochord superficial to the superior constrictor muscle of the pharynx and is covered by the mucous membrane of the nasopharynx. It may become infected, causing 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. Diagnosis is based on nasopharyngoscopy supplemented by CT or MRI when the diagnosis is in doubt. Treatment consists of marsupialization or excision.

Velopharyngeal Insufficiency

Velopharyngeal insufficiency is incomplete closure of a sphincter between the oropharynx and nasopharynx, often resulting from anatomic abnormalities of the palate and causing hypernasal speech. Treatment is with speech therapy and surgery.

Velopharyngeal insufficiency is incomplete closure of the velopharyngeal sphincter between the oropharynx and the nasopharynx. Closure, normally achieved by the sphincteric action of the soft palate and the superior constrictor muscle, is impaired in patients with cleft palate, repaired cleft palate, congenitally short palate, submucous cleft palate, palatal paralysis, and, sometimes, enlarged tonsils. The condition may also result when adenoidectomy or uvulopalatopharyngoplasty is done in a patient with a congenital underdevelopment (submucous cleft) or paralysis of the palate.

Symptoms and Signs

Speech in a patient with velopharyngeal insufficiency is characterized by hypernasal resonant voice, nasal emission of air, nasal turbulence, and inability to produce sounds requiring oral pressure (plosives). Severe velopharyngeal insufficiency results in regurgitation of solid foods and fluids through the nose. Inspection of the palate during phonation may reveal palatal paralysis.

Diagnosis

• Direct inspection with a fiberoptic nasoendoscope

The diagnosis is suspected in patients with the typical speech abnormalities. Palpation of the midline of the soft palate may reveal an occult submucous cleft. Direct inspection with a fiberoptic nasoendoscope is the primary diagnostic technique. Multiview videofluoroscopy during connected speech and swallowing (modified barium swallow), done in conjunction with a speech pathologist, can also be used.

Treatment

• Surgical repair and speech therapy

Treatment consists of speech therapy and surgical correction by a palatal elongation pushback procedure, posterior pharyngeal wall implant, pharyngeal flap, or pharyngoplasty, depending on the mobility of the lateral pharyngeal walls, the degree of velar elevation, and the size of the defect.

Tonsillopharyngitis

(See also p. 1232.)

Tonsillopharyngitis is acute infection of the pharynx, palatine tonsils, or both. Symptoms may include sore throat, dysphagia, cervical lymphadenopathy, and fever. Diagnosis is clinical, supplemented by culture or rapid antigen test. Treatment depends on symptoms and, in the case of group A β-hemolytic streptococcus, involves antibiotics.

The tonsils participate in systemic immune surveillance. In addition, local tonsillar defenses include a lining of antigen-processing squamous epithelium that involves B- and T-cell responses.

Tonsillopharyngitis of all varieties constitutes about 15% of all office visits to primary care physicians.

Etiology

Tonsillopharyngitis is usually viral, most often caused by the common cold viruses (adenovirus, rhinovirus, influenza, coronavirus, respiratory syncytial virus), but occasionally by Epstein-Barr virus, herpes simplex virus, cytomegalovirus, or HIV.

In about 30% of patients, the cause is bacterial. Group A β-hemolytic streptococcus (GABHS) is most common (see p. 1232), but *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Mycoplasma pneumoniae*, and *Chlamydia pneumoniae* are sometimes involved. Rare causes include pertussis, *Fusobacterium*, diphtheria, syphilis, and gonorrhea.

GABHS occurs most commonly between ages 5 and 15 and is uncommon before age 3.

Symptoms and Signs

Pain with swallowing is the hallmark and is often referred to the ears. Very young children who are not able to complain of sore throat often refuse to eat. High fever, malaise, headache, and GI upset are common, as are halitosis and a muffled voice. A scarlatiniform or nonspecific rash may also be present. The tonsils are swollen and red and often have purulent exudates. Tender cervical lymphadenopathy may be present. Fever, adenopathy, palatal petechiae, and exudates are somewhat more common with GABHS than with viral tonsillopharyngitis, but there is much overlap. GABHS usually resolves within 7

days. Untreated GABHS may lead to local suppurative complications (eg, peritonsillar abscess or cellulitis) and sometimes to rheumatic fever or glomerulonephritis.

Diagnosis

• Clinical evaluation

• GABHS ruled out by rapid antigen test, culture, or both

Pharyngitis itself is easily recognized clinically. However, its cause is not. Rhinorrhea and cough usually indicate a viral cause. Infectious mononucleosis is suggested by posterior cervical or generalized adenopathy, hepatosplenomegaly, fatigue, and malaise for > 1 wk; a full neck with petechiae of the soft palate; and thick tonsillar exudates. A dirty gray, thick, tough membrane that bleeds if peeled away indicates diphtheria (rare in the US).

Because GABHS requires antibiotics, it must be diagnosed early. Criteria for testing are controversial. Many authorities recommend testing with a rapid antigen test or culture for all children. Rapid antigen tests are specific but not sensitive and may need to be followed by a culture, which is about 90% specific and 90% sensitive. In adults, many authorities recommend using the following 4 criteria:

• History of fever

• Tonsillar exudates

• Absence of cough

• Tender anterior cervical lymphadenopathy

Patients who meet 1 or no criteria are unlikely to have GABHS and should not be tested. Patients who meet 2 criteria can be tested. Patients who meet 3 or 4 criteria can be tested or treated empirically for GABHS.

Treatment

• Symptomatic treatment

• Antibiotics for GABHS

• Tonsillectomy considered for recurrent GABHS

Supportive treatments include analgesia, hydration, and rest. Penicillin V is usually considered the drug of choice for GABHS tonsillopharyngitis; dose is 250 mg po bid for 10 days for patients < 27 kg and 500 mg for those > 27 kg (see also p. 1234). Amoxicillin is effective and more palatable if a liquid preparation is required. If adherence is a concern, a single dose of benzathine penicillin 1.2 million units IM (600,000 units for children ≤ 27 kg) is effective. Other oral drugs include macrolides for patients allergic to penicillin, a 1st-generation cephalosporin, and clindamycin.

Treatment may be started immediately or delayed until culture results are known. If treatment is started presumptively, it should be stopped if cultures are negative. Follow-up throat cultures are not done routinely. They are useful in patients with multiple GABHS recurrences or if pharyngitis spreads to close contacts at home or school.

Tonsillectomy: Tonsillectomy should be considered if GABHS tonsillitis recurs repeatedly (> 6 episodes/yr, > 4 episodes/yr for 2 yr, > 3 episodes/yr for 3 yr) or if acute infection is severe and persistent despite antibiotics. Other criteria for tonsillectomy include obstructive sleep disorder, recurrent peritonsillar abscess, and suspicion of cancer.

Numerous effective surgical techniques are used to perform tonsillectomy, including electrocautery,

microdebrider, radiofrequency coblation, and sharp dissection. Significant intraoperative or postoperative bleeding occurs in < 2% of patients, usually within 24 h of surgery or after 7 days, when the eschar detaches. Patients with bleeding should go to the hospital. If bleeding continues on arrival, patients generally are examined in the operating room, and hemostasis is obtained. Any clot present in the tonsillar fossa is removed, and patients are observed for 24 h. Postoperative IV rehydration is necessary in ≤ 3% of patients, possibly in fewer patients with use of optimal preoperative hydration, perioperative antibiotics, analgesics, and corticosteroids. Postoperative airway obstruction occurs most frequently in children < 2 yr who have preexisting severe obstructive sleep disorders and in patients who are morbidly obese or have neurologic disorders, craniofacial anomalies, or significant preoperative obstructive sleep apnea. Complications are generally more common and serious among adults.

Peritonsillar Abscess and Cellulitis

Peritonsillar abscess and cellulitis are acute pharyngeal infections most common among adolescents and young adults. Symptoms are severe sore throat, trismus, "hot potato" voice, and uvular deviation. Diagnosis requires needle aspiration. Treatment includes broad-spectrum antibiotics, drainage of any pus, hydration, analgesics, and, occasionally, acute tonsillectomy.

Etiology

Abscess (quinsy) and cellulitis probably represent a spectrum of the same process in which bacterial infection of the tonsils and pharynx spreads to the soft tissues. Infection is virtually always unilateral and is located between the tonsil and the superior pharyngeal constrictor muscle. It usually involves multiple bacteria. *Streptococcus* and *Staphylococcus* are the most frequent aerobic pathogens, whereas *Bacteroides* sp is the predominant anaerobic pathogen.

Symptoms and Signs

Symptoms include gradual onset of severe unilateral sore throat, dysphagia, fever, otalgia, and asymmetric cervical adenopathy. Trismus, "hot potato" voice (speaking as if a hot object was in the mouth), a toxic appearance (see Epiglottitis on p. 475), drooling, severe halitosis, tonsillar erythema, and exudates are common. Abscess and cellulitis both have swelling above the affected tonsil, but with abscess there is more of a discrete bulge, with deviation of the soft palate and uvula and pronounced trismus.

Diagnosis

• Needle aspiration

• Sometimes CT

Peritonsillar cellulitis is recognized in patients with severe sore throat who have trismus, "hot potato" voice, and uvular deviation. All such patients require needle aspiration of the tonsillar mass and cultures. Aspiration of pus differentiates abscess from cellulitis. CT or ultrasound of the neck can help confirm the diagnosis when the physical examination is difficult or the diagnosis is in doubt, particularly when the condition must be differentiated from a parapharyngeal infection or other deep neck infection.

Treatment

• Antibiotics

• Drainage of abscess

Cellulitis subsides, usually within 48 h, with hydration and high-dose penicillin (eg, 2 million units IV q 4 h or 1 g po qid); alternative drugs include a 1st-generation cephalosporin or clindamycin. Culture-directed antibiotics are then prescribed for 10 days. Abscesses are incised and drained in the emergency department using thorough local anesthesia and sometimes procedural sedation; many clinicians believe needle aspiration alone provides adequate drainage. Although most patients can be treated as

outpatients, some need brief hospitalization for parenteral antibiotics, IV hydration, and airway monitoring. Rarely, an immediate tonsillectomy is done, particularly in a young or uncooperative patient who has other indications for elective tonsillectomy (eg, history of frequently recurrent tonsillitis or obstructive sleep apnea). Otherwise, elective tonsillectomy is done 4 to 6 wk out to prevent abscess recurrence.

Parapharyngeal Abscess

A parapharyngeal abscess is a deep neck abscess treated with antibiotics and surgical drainage.

The parapharyngeal (pharyngomaxillary) space is lateral to the superior pharyngeal constrictor and medial to the masseter muscle. This space connects to every other major fascial neck space and is divided into anterior and posterior compartments by the styloid process. The posterior compartment contains the carotid artery, internal jugular vein, and numerous nerves. Infections in the parapharyngeal space usually originate in the tonsils or pharynx, although local spread from odontogenic sources and lymph nodes may occur.

Abscess swelling can compromise the airway. Posterior space abscess can erode into the carotid artery or cause septic thrombophlebitis of the internal jugular vein (Lemierre syndrome).

Symptoms and Signs

Most patients have fever, sore throat, odynophagia, and swelling in the neck down to the hyoid bone. Anterior space abscesses cause trismus and induration along the angle of the mandible, with medial bulging of the tonsil and lateral pharyngeal wall. Posterior space abscesses cause swelling that is more prominent in the posterior pharyngeal wall. Trismus is minimal. Posterior abscesses may involve structures within the carotid sheath, possibly causing rigors, high fever, bacteremia, neurologic deficits, and massive hemorrhage caused by carotid artery rupture.

Diagnosis

• CT

Diagnosis is suspected in patients with poorly defined deep neck infection or other typical symptoms and is confirmed by using contrast-enhanced CT.

Treatment

• Broad-spectrum antibiotics (eg, ceftriaxone, clindamycin)

• Surgical drainage

Treatment may require airway control. Parenteral broad-spectrum antibiotics (eg, ceftriaxone, clindamycin) and surgical drainage are generally needed. Posterior abscesses are drained externally through the submaxillary fossa. Anterior abscesses can often be drained through an intra-oral incision. Several days of parenteral culture-determined antibiotics are required after drainage, followed by 10 to 14 days of oral antibiotics. Occasionally, small abscesses can be treated with IV antibiotics alone.

Epiglottitis

(Supraglottitis)

Epiglottitis is a rapidly progressive bacterial infection of the epiglottis and surrounding tissues that may lead to sudden respiratory obstruction and death. Symptoms include severe sore throat, dysphagia, high fever, drooling, and inspiratory stridor. Diagnosis requires direct visualization of the supraglottic structures, which is not to be done until full respiratory support is available. Treatment includes airway protection and antibiotics.

Epiglottitis used to be primarily a disease of children and usually was caused by *Haemophilus influenzae* type B. Now, because of widespread vaccination, it has been almost eradicated in children (more cases occur in adults). Causal organisms in children and adults include *Streptococcus pneumoniae*, *Staphylococcus aureus*, nontypeable *H. influenzae*, *Haemophilus parainfluenzae*, β-hemolytic streptococci, *Branhamella catarrhalis*, and *Klebsiella pneumoniae*. *H. influenzae* type B is still a cause in adults and unvaccinated children.

Bacteria that have colonized the nasopharynx spread locally to cause supraglottic cellulitis with marked inflammation of the epiglottis, vallecula, aryepiglottic folds, arytenoids, and laryngeal ventricles. With *H. influenzae* type B, infection may spread hematogenously.

The inflamed supraglottic structures mechanically obstruct the airway, increasing the work of breathing, ultimately causing respiratory failure. Clearance of inflammatory secretions is also impaired.

Symptoms and Signs

In children, sore throat, odynophagia, and dysphagia develop abruptly. Fatal asphyxia may occur within a few hours of onset. Drooling is very common. Additionally, the child has signs of toxicity (poor or absent eye contact, failure to recognize parents, cyanosis, irritability, inability to be consoled or distracted) and is febrile and anxious. Dyspnea, tachypnea, and inspiratory stridor may be present, often causing the child to sit upright, lean forward, and hyperextend the neck with the jaw thrust forward and mouth open in an effort to enhance air exchange (tripod position). Relinquishing this position may herald respiratory failure. Suprasternal, supraclavicular, and subcostal inspiratory retractions may be present.

In adults, symptoms are similar to those of children, including sore throat, fever, dysphagia, and drooling, but peak symptoms usually take > 24 h to develop. Because of the larger diameter of the adult airway, obstruction is less common and less fulminant. Often, there is no visible oropharyngeal inflammation. However, severe throat pain with a normal-appearing pharynx raises suspicion of epiglottitis.

Diagnosis

• Direct inspection (typically in operating room)

• X-ray in milder cases with low suspicion

Epiglottitis is suspected in patients with severe sore throat and no pharyngitis and also in patients with sore throat and inspiratory stridor. Stridor in children may also result from croup (viral laryngotracheal bronchitis—see
Table 52-1 and p. 2879), bacterial tracheitis, and airway foreign body. The tripod position may also occur with peritonsillar or retropharyngeal abscess.

The patient is hospitalized if epiglottitis is suspected. Diagnosis requires direct examination, usually with flexible fiberoptic laryngoscopy. (CAUTION: *Examination of the pharynx and larynx may precipitate complete respiratory obstruction in children, and the pharynx and larynx should not be directly examined except in the operating room, where the most advanced airway intervention is available.*) Although plain x-rays may be helpful, a child with stridor should not be transported to the x-ray suite. Direct laryngoscopy that reveals a beefy-red, stiff, edematous epiglottis is diagnostic. Cultures from the supraglottic tissues and blood can then be taken to search for the causative organism.

[**Table 52-1.** Differentiating Epiglottitis from Croup]

Adults may, in some cases, safely undergo flexible fiberoptic laryngoscopy.

Treatment

• Adequate airway ensured

• Antibiotics (eg, ceftriaxone)

In children, the airway must be secured immediately, preferably by nasotracheal intubation. Securing the airway can be quite difficult and should, if possible, be done by experienced personnel in the operating room. An endotracheal tube is usually required until the patient has been stabilized for 24 to 48 h (usual total intubation time is < 60 h). Alternatively, a tracheotomy is done. If respiratory arrest occurs before an airway is established, bag-mask ventilation may be a life-saving temporary measure. For emergency care of children with epiglottitis, each institution should have a protocol that involves critical care, otolaryngology, anesthesia, and pediatrics.

Adults whose airway is severely obstructed can be endotracheally intubated during flexible fiberoptic laryngoscopy. Other adults may not require immediate intubation but should be observed for airway compromise in an ICU with an intubation set and cricothyrotomy tray at the bedside.

A β-lactamase-resistant antibiotic, such as ceftriaxone 50 to 75 mg/kg IV once/day (maximum 2 g), should be used empirically, pending culture and sensitivity test results.

Epiglottitis caused by *H. influenzae* type B can be effectively prevented with the *H. influenzae* type B (Hib) conjugate vaccine.

Chapter 53. Nose and Paranasal Sinus Disorders

Introduction

(See Ch. 51 for a detailed description of the anatomy of the nose and sinuses.)

Bacterial Infections

Nasal vestibulitis is bacterial infection of the nasal vestibule, typically with *Staphylococcus aureus*. It may result from nose picking or excessive nose blowing and causes annoying crusts and bleeding when the crusts slough off. Bacitracin or mupirocin ointment 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 antistaphylococcal antibiotics (eg, cephalexin 500 mg po qid) are given and warm compresses and topical mupirocin are applied. Furuncles are incised and drained to prevent local thrombophlebitis and subsequent cavernous sinus thrombosis.

Foreign Bodies

Nasal foreign bodies are found occasionally in young children, the intellectually impaired, and psychiatric patients. Common objects pushed into the nose include beads, beans, seeds, nuts, insects, and button batteries (which may cause chemical burns). When mineral salts are deposited on a long-retained foreign body, the object is called a rhinolith.

A nasal foreign body is suspected in any patient with a unilateral, foul-smelling, bloody, purulent rhinorrhea. Diagnosis is often made through another party's observation of the item being pushed into the nose or through visualization with a nasal speculum.

Nasal foreign bodies can sometimes be removed in the office with a nasal speculum and Hartmann's nasal forceps. Pretreatment with topical phenylephrine may aid visualization and removal. To avoid pushing a slippery, round object deeper, it is better to reach behind the object with the bent tip of a blunt probe and pull it forward. Sometimes, general anesthesia is necessary if a rhinolith has formed or if the foreign body may be displaced dorsally and then aspirated, resulting in airway obstruction.

Nasal Polyps

Nasal polyps are fleshy outgrowths of the nasal mucosa that form at the site of dependent edema in the lamina propria of the mucous membrane, usually around the ostia of the maxillary sinuses (see Plate 2).

Allergic rhinitis, acute and chronic infections, and cystic fibrosis all predispose to the formation of nasal polyps. Bleeding polyps occur in rhinosporidiosis. Unilateral polyps occasionally occur in association with or represent benign or malignant tumors of the nose or paranasal sinuses. They can also occur in response to a foreign body. Nasal polyps are strongly associated with aspirin allergy, sinus infections, and asthma.

Symptoms include obstruction and postnasal drainage, congestion, sneezing, rhinorrhea, anosmia, hyposmia, facial pain, and ocular itching.

Diagnosis generally is based on physical examination. A developing polyp is teardrop-shaped; when mature, it resembles a peeled seedless grape.

Treatment

• Topical corticosteroid spray

• Sometimes surgical removal

Corticosteroids (eg, mometasone [30 µg/spray], beclomethasone [42 µg/spray], flunisolide [25 µg/spray] aerosols), given as 1 or 2 sprays bid in each nasal cavity, may shrink or eliminate polyps, as may a 1-wk tapered course of oral corticosteroids. Surgical removal is still required in many cases. Polyps that obstruct the airway or promote sinusitis are removed, as are unilateral polyps that may be obscuring benign or malignant tumors. However, polyps tend to recur unless the underlying allergy or infection is controlled. After removal of nasal polyps, topical beclomethasone or flunisolide therapy tends to retard recurrence. In severe recurrent cases, maxillary sinusotomy or ethmoidectomy may be indicated. These procedures are usually done endoscopically.

Rhinitis

(See also Allergic Rhinitis on p. 1117.)

Rhinitis is inflammation of the nasal mucous membrane, with resultant nasal congestion, rhinorrhea, and variable associated symptoms depending on etiology (eg, itching, sneezing, purulence, anosmia, ozena). The cause is usually viral, although irritants can cause it. Diagnosis is usually clinical. Treatment includes humidification of room air, sympathomimetic amines, and antihistamines. Bacterial superinfection requires appropriate antibiotic treatment.

There are several forms of rhinitis.

Acute rhinitis: This form of rhinitis, manifesting with edema and vasodilation of the nasal mucous membrane, rhinorrhea, and obstruction, is usually the result of a common cold (see p. 1404); other causes include streptococcal, pneumococcal, and staphylococcal infections.

Chronic rhinitis: This form of rhinitis is generally a prolongation of subacute inflammatory or infectious viral 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. Nasal obstruction, purulent rhinorrhea, and frequent bleeding result. Rhinoscleroma causes progressive nasal obstruction from indurated inflammatory tissue in the lamina propria. Rhinosporidiosis is characterized by bleeding polyps. Both low humidity and airborne irritants can result in chronic rhinitis.

Atrophic rhinitis: This form of rhinitis results in atrophy and sclerosis of mucous membrane; the mucous membrane changes from ciliated pseudostratified columnar epithelium to stratified squamous epithelium, and the lamina propria is reduced in amount and vascularity. Atrophic rhinitis is associated with advanced age, Wegener's granulomatosis, and iatrogenically induced excessive nasal tissue extirpation. Although the exact etiology is unknown, bacterial infection frequently plays a role. Nasal mucosal atrophy often occurs in the elderly.

Vasomotor rhinitis: This form of rhinitis is a chronic condition in which intermittent vascular engorgement of the nasal mucous membrane leads to watery rhinorrhea and sneezing. Etiology is uncertain, and no allergy can be identified. A dry atmosphere seems to aggravate the condition.

Symptoms and Signs

Acute rhinitis results in cough, low-grade fever, nasal congestion, rhinorrhea, and sneezing. Symptoms and signs of chronic rhinitis are similar but may include purulent rhinorrhea and bleeding.

Atrophic rhinitis results in abnormal patency of the nasal cavities, crust formation, anosmia, and epistaxis that may be recurrent and severe.

Vasomotor rhinitis results in sneezing and watery rhinorrhea. The turgescent mucous membrane varies from bright red to purple. The condition is marked by periods of remission and exacerbation. 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.

Diagnosis

The different forms of rhinitis are diagnosed clinically. Testing is unnecessary.

Treatment

• For viral rhinitis, decongestants, antihistamines, or both

• For atrophic rhinitis, topical treatment

• For vasomotor rhinitis, humidification and sometimes topical corticosteroids and oral pseudoephedrine

Viral rhinitis may be treated symptomatically with decongestants (either topical vasoconstriction with a sympathomimetic amine, such as oxymetazoline q 8 to 12 h or phenylephrine 0.25% q 3 to 4 h for not more than 7 days, or systemic sympathomimetic amines, such as pseudoephedrine 30 mg po q 4 to 6 h). Antihistamines (see
Table 127-2 on p. 1111) may be helpful. Those with anticholinergic properties dry mucous membranes and therefore may increase irritation. Decongestants also may relieve symptoms of acute bacterial rhinitis and chronic rhinitis, whereas an underlying bacterial infection requires culture or biopsy, pathogen identification, antibiotic sensitivities, and appropriate antimicrobial treatment.

Treatment of atrophic rhinitis is directed at reducing the crusting and eliminating the odor with topical antibiotics (eg, bacitracin), topical or systemic estrogens, and vitamins A and D. Occluding or reducing the patency of the nasal cavities surgically decreases the crusting caused by the drying effect of air flowing over the atrophic mucous membrane.

Treatment of vasomotor rhinitis is by trial and error and is not always satisfactory. Patients benefit from humidified air, which may be provided by a humidified central heating system or a vaporizer in the workroom or bedroom. Systemic sympathomimetic amines (eg, for adults, pseudoephedrine 30 mg po q 4 to 6 h prn) relieve symptoms but are not recommended for long-term use. Topical vasoconstrictors are 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. Topical corticosteroids (eg, mometasone 2 sprays bid) can be of some benefit.

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. Symptomatic septal deviation causes nasal obstruction and predisposes the patient to sinusitis (particularly if the deviation obstructs the ostium of a paranasal sinus) and to epistaxis due to drying air currents. Other symptoms may include facial pain, headaches, and noisy night breathing. Septal deviation is usually evident on examination, although a flashlight and examination of the anterior nasal passage may not be sufficient. Treatment consists of septoplasty (septal reconstruction).

Septal ulcers and perforations may result from nasal surgery; repeated trauma, such as chronic nose picking; cosmetic piercing; toxic exposures (eg, acids, chromium, phosphorus, copper vapor); chronic cocaine use; chronic nasal spray use (including corticosteroids and OTC phenylephrine or oxymetazoline sprays); transnasal O_2 use; or diseases such as TB, syphilis, leprosy, SLE, and Wegener's granulomatosis. Crusting around the margins and repeated epistaxis, which can be severe, may result. Small perforations may whistle. Anterior rhinoscopy or fiberoptic endoscopy can be used to view septal perforations. Topical bacitracin or mupirocin ointment reduces crusting, as may saline nasal spray. Symptomatic septal perforations are occasionally repaired with buccal or septal mucous membrane flaps; closing the perforation with a silicone septal button is a reliable option.

Sinusitis

Sinusitis is inflammation of the paranasal sinuses due to viral, bacterial, or fungal infections or allergic reactions. Symptoms include nasal obstruction and congestion, purulent rhinorrhea,

cough, facial pain, malaise, and sometimes fever. Treatment is with antibiotics, such as amoxicillin, penicillin, erythromycin, or trimethoprim/sulfamethoxazole, given for 12 to 14 days for acute sinusitis and for up to 6 wk for chronic sinusitis. Decongestants and application of heat and humidity may help relieve symptoms and improve sinus drainage. Recurrent sinusitis may require surgery to improve sinus drainage.

Sinusitis may be classified as acute (completely resolved in < 30 days); subacute (completely resolved in 30 to 90 days); recurrent (multiple discrete acute episodes, each completely resolved in < 30 days but recurring in cycles, with at least 10 days between complete resolution of symptoms and initiation of a new episode); and chronic (lasting > 90 days).

Etiology

Acute sinusitis is usually precipitated by viral URI, followed by secondary bacterial colonization with streptococci, pneumococci, *Haemophilus influenzae*, *Moraxella catarrhalis*, or staphylococci. In a URI, the swollen nasal mucous membrane obstructs the ostium of a paranasal sinus, and the O_2 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 through 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.

Chronic sinusitis may be exacerbated by gram-negative bacilli or anaerobic micro-organisms. In a few cases, chronic maxillary sinusitis is secondary to dental infection or exposure to environmental pollution. Fungal infections (*Aspergillus*, *Sporothrix*, *Pseudallescheria*) tend to strike the immunocompromised patient, whereas hospital-acquired infections complicate cystic fibrosis, nasogastric and nasotracheal intubation, and debilitated patients. Typical organisms include *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, and *Enterobacter*.

Allergic fungal sinusitis is characterized by diffuse nasal congestion, markedly viscid nasal secretions, and, often, nasal polyps. It is an allergic response to the presence of topical fungi, often *Aspergillus*, and is not caused by an invasive infection.

Symptoms and Signs

Acute and chronic sinusitis cause similar symptoms and signs, including purulent rhinorrhea, pressure and pain in the face, nasal congestion and obstruction, hyposmia, halitosis, and productive cough (especially at night). Often the pain is more severe in acute sinusitis. The area over the affected sinus may be tender, swollen, and erythematous. Maxillary sinusitis causes pain in the maxillary area, toothache, and frontal headache. Frontal sinusitis causes pain in the frontal area and frontal headache. Ethmoid sinusitis causes pain behind and between the eyes, frontal headache often described as splitting, periorbital cellulitis, and tearing. Pain caused by 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.

Diagnosis

• Clinical evaluation

• Sometimes CT

Sinus infections are usually diagnosed clinically. Absence or dullness of light on transillumination may

suggest fluid-filled maxillary or frontal sinuses. In acute and chronic sinusitis, the swollen mucous membranes and retained exudate cause the affected sinus to appear opaque on 4-view x-rays. Plain x-rays are not as valuable as CT, which 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. When questions persist (eg, regarding intracranial extension, treatment failure, or hospital-acquired causes of sinusitis), culture and sensitivity tests can be done on sinus secretions obtained through endoscopy or sinus puncture and aspiration.

Sinusitis in children is suspected when purulent rhinorrhea persists for > 10 days along with fatigue and cough. Fever is uncommon. Local facial pain or discomfort may be present. Nasal examination discloses purulent drainage; CT is confirmatory. CT is of limited cuts in the coronal projection to limit radiation exposure.

Treatment

• Local measures to enhance drainage (eg, steam, topical vasoconstrictors)

• Antibiotics (eg, amoxicillin, erythromycin, trimethoprim/sulfamethoxazole)

In acute sinusitis, improved drainage and control of infection are the aims of therapy. Steam inhalation; hot, wet towels over the affected sinuses; and hot beverages alleviate nasal vasoconstriction and promote drainage. Topical vasoconstrictors, such as phenylephrine 0.25% spray q 3 h, are effective but should be used for a maximum of 5 days or for a repeating cycle of 3 days on and 3 days off until the sinusitis is resolved. Systemic vasoconstrictors, such as pseudoephedrine 30 mg po (for adults) q 4 to 6 h, are less effective.

In acute and chronic sinusitis, antibiotics are given for at least 10 days and often for 14 days. In acute sinusitis, amoxicillin 500 mg po q 8 h with or without clavulanate is primary therapy. Erythromycin 250 mg po q 6 h or trimethoprim/sulfamethoxazole 80/400 mg q 6 h can be given to patients allergic to penicillin. Second-line therapy includes cefuroxime 500 mg q 12 h or moxifloxacin 400 mg once/day. For children, similar antibiotics are used, adjusted for the child's weight. Fluoroquinolones, however, are not used in children because of concerns of premature epiphyseal growth plate closure.

In exacerbations of chronic sinusitis in children or adults, a broad-spectrum antibiotic, such as amoxicillin/clavulanate 875 mg po q 12 h (12.5 to 25 mg/kg q 12 h in children), cefuroxime, or, in adults, moxifloxacin, is used. In chronic sinusitis, prolonged antibiotic therapy for 4 to 6 wk often brings complete resolution. The sensitivities of pathogens isolated from the sinus exudate and the patient's response guide subsequent therapy.

Sinusitis unresponsive to antibiotic therapy may require surgery (maxillary sinusotomy, ethmoidectomy, or sphenoid sinusotomy) to improve ventilation and drainage and to remove inspissated mucopurulent material, epithelial debris, and hypertrophic mucous membrane. These procedures usually are done intranasally with the aid of an endoscope. Chronic frontal sinusitis may be managed either with osteoplastic obliteration of the frontal sinuses or endoscopically in selected patients. The use of intraoperative computer-aided surgery to localize disease and prevent injury to surrounding contiguous structures (such as the eye and brain) has become common.

Sinusitis in Immunocompromised Patients

Aggressive and even fatal fungal or bacterial sinusitis can occur in patients who are immunocompromised because of poorly controlled diabetes, neutropenia, or HIV infection.

Mucormycosis: 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 based on histopathologic demonstration of mycelia in the avascularized tissue. Treatment requires control of the underlying condition (such as reversal of ketoacidosis in diabetes) and IV amphotericin B therapy. Prompt

biopsy of intranasal tissue for histology and culture is warranted.

Aspergillosis and candidiasis: *Aspergillus* and *Candida* spp may infect the paranasal sinuses of patients who are immunocompromised secondary to therapy with cytotoxic drugs or to immunosuppressive diseases, such as leukemia, lymphoma, multiple myeloma, and AIDS. These infections can appear as polypoid tissue in the nose as well as thickened mucosa; tissue is required for diagnosis. Aggressive paranasal sinus surgery and IV amphotericin B therapy are used to control these often-fatal infections.

Chapter 54. Laryngeal Disorders

Introduction

The larynx contains the vocal cords and serves as the opening to the tracheobronchial tree. Laryngeal disorders include various benign and malignant tumors, contact ulcers, granulomas, laryngitis, laryngoceles, spasmodic dysphonia, vocal cord paralysis, and vocal cord polyps and nodules. For acute laryngotracheobronchitis, see Croup on p. 2879.

Laryngeal cancer is discussed on p. 489.

Most laryngeal disorders cause dysphonia, which is impairment of the voice (see Sidebar 54-1). A persistent change in the voice (eg, > 3 wk) requires visualization of the vocal cords, including their mobility. Although the voice changes with advancing age, becoming breathy and aperiodic, acute or prominent changes in the elderly should not be presumed to result from aging, and evaluation is required.

The voice should be assessed and recorded, particularly if surgical procedures are planned. Examination of the larynx includes external inspection and palpation of the neck and internal visualization of the epiglottis, false cords, true cords, arytenoids, pyriform sinuses, and subglottic region below the cords. Internal visualization is accomplished by either indirect mirror examination (see Fig. 54-1) or direct flexible fiberoptic laryngoscopy in an outpatient setting with a topical anesthetic. Rigid laryngoscopy with the patient under general anesthesia allows for biopsy when necessary or assessment of passive mobility of the vocal cords when immobilized by either paralysis or fixation.

Sidebar 54-1 The Professional Voice

People who use their voice professionally for public speaking and singing often experience voice disorders manifesting as hoarseness or breathiness, lowered vocal pitch, vocal fatigue, nonproductive cough, persistent throat clearing, and/or throat ache. These symptoms often have benign causes, such as vocal nodules, vocal fold edema, polyps, or granulomas. Such disorders are usually caused by vocal fold hyperfunction (excessive laryngeal muscular tension when speaking) and possibly laryngopharyngeal reflux.

Treatment in most cases includes the following:

- Voice evaluation by a speech pathologist or experienced physician, including, when available, use of a computer-assisted program to assess pitch and intensity and to determine parameters of vocal acoustics
- Behavioral treatment (decreasing musculoskeletal laryngeal tension when speaking) using the same computer program for visual and auditory biofeedback
- A vocal hygiene program to eliminate vocally abusive behaviors, such as excessive loudness, long duration, vocal tension, and habitual throat clearing
- An antireflux regimen, when appropriate
- Adequate hydration to promote an adequate glottal mucosal wave
- Diet modification before vocal performances, which may include avoidance of dairy products, caffeine, and ambient tobacco smoke and other inhaled irritants

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Benign Tumors

Benign laryngeal tumors include juvenile papillomas hemangiomas, fibromas, chondromas, myxomas, and neurofibromas. They may appear in any part of the larynx. Symptoms include hoarseness, breathy voice,

dyspnea, aspiration, dysphagia, pain, otalgia (pain referred to the ear), and hemoptysis. Diagnosis is based on direct or indirect visualization of the larynx, supplemented by CT. Removal restores the voice, the functional integrity of the laryngeal sphincter, and the airway. Smaller lesions may be excised endoscopically by using a CO_2 laser and general anesthesia. Larger lesions extending beyond the laryngeal framework often require pharyngotomy or laryngofissure.

Cancerous tumors are discussed in Ch. 55.

Contact Ulcers

Contact ulcers are unilateral or bilateral erosions of the mucous membrane over the vocal process of the arytenoid cartilage.

Contact ulcers are usually caused by voice abuse in the form of repeated sharp glottal attacks (abrupt loudness at the onset of phonation), often experienced by singers. They may also occur after endotracheal intubation if an oversized tube erodes the mucosa overlying the cartilaginous vocal processes. Gastroesophageal reflux may also cause or aggravate contact ulcers. Symptoms include varying degrees of hoarseness and mild pain with phonation and swallowing. Biopsy to exclude carcinoma or TB is important. Prolonged ulceration leads to nonspecific granulomas that also cause varying degrees of hoarseness.

Treatment consists of ≥ 6 wk of voice rest. Patients must recognize the limitations of their voice and learn to adjust their postrecovery vocal activities to avoid recurrence. Granulomas tend to recur after surgical removal. Risk of recurrence is reduced through vigorous treatment of gastroesophageal reflux (see p. 125). Suppression of bacterial flora by antibiotics during postoperative healing is also recommended.

Laryngitis

Laryngitis is inflammation of the larynx, usually the result of a virus or overuse. The result is acute change in the voice, with decreased volume and hoarseness. Diagnosis is based on clinical findings. Laryngoscopy is required for symptoms persisting > 3 wk. Viral laryngitis is self-limited. Other infectious or irritating causes may require specific treatment.

The most common cause of acute laryngitis is a viral URI. Coughing-induced laryngitis may also occur in bronchitis, pneumonia, influenza, pertussis, measles, and diphtheria.

[**Fig. 54-1.** Laryngeal disorders.]

Excessive use of the voice (especially with loud speaking or singing), allergic reactions, gastroesophageal reflux, bulimia, or inhalation of irritating substances (eg, cigarette smoke or certain aerosolized drugs) can cause acute or chronic laryngitis. Bacterial laryngitis is extremely rare. Smoking can cause Reinke's edema, which is a watery swelling of both vocal cords.

Symptoms and Signs

An unnatural change of voice is usually the most prominent symptom. Volume is typically greatly decreased; some patients have aphonia. Hoarseness, 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, although rare, may cause dyspnea.

Diagnosis

• Clinical evaluation

• Sometimes direct or indirect laryngoscopy

Diagnosis is based on symptoms. Indirect or direct flexible laryngoscopy is recommended for symptoms

persisting > 3 wk; findings in laryngitis include mild to marked erythema of the mucous membrane, which may also be edematous. With reflux, there is swelling of the inner lining of the larynx and redness of the vocal cords that extends above and below the edges of the back part of the cords. If a pseudomembrane is present, diphtheria is suspected.

Treatment

• Symptomatic treatment (eg, cough suppressants, voice rest, steam inhalations)

No specific treatment is available for viral laryngitis. Cough suppressants, voice rest, and steam inhalations relieve symptoms and promote resolution of acute laryngitis. Smoking cessation and treatment of acute or chronic bronchitis may relieve laryngitis. Depending on the presumed cause, specific treatments to control gastroesophageal reflux, bulimia, or drug-induced laryngitis may be beneficial.

Laryngoceles

Laryngoceles are 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, causing a mass in the neck. Laryngoceles tend to occur in musicians who play wind instruments. Laryngoceles are filled with air and can be expanded by the Valsalva maneuver. They appear on CT as smooth, ovoid, low-density masses. Laryngoceles may become infected (laryngopyocele) or filled with mucoid fluid. Treatment is excision.

Spasmodic Dysphonia

Spasmodic dysphonia (vocal cord spasms) is intermittent spasm of laryngeal muscles that causes an abnormal voice.

Cause is unknown. Patients often describe the onset of symptoms following a URI, a period of excessive voice use, or occupational or emotional stress. As a localized form of movement disorder, spasmodic dysphonia has an onset between ages 30 and 50 yr, and about 60% of patients are women.

In the adductor type of spasmodic dysphonia, patients attempt to speak through the spasmodic closure with a voice that sounds squeezed, effortful, or strained. These spasmodic episodes usually occur when vowel sounds are being formed, particularly at the beginning of words. The less common abductor form results in sudden interruptions of sound caused by momentary abduction of the vocal cords accompanied by audible escape of air during connected speech.

Surgery has been more successful than other approaches for adductor spasmodic dysphonia. The use of botulinum toxin injection has restored a normal voice in 70% of patients for up to 3 mo. Because the effect is temporary, injections may be repeated. There is no known temporary alleviation of the abductor form of this disorder.

Vocal Cord Paralysis

Vocal cord paralysis has numerous causes and can affect speaking, breathing, and swallowing. The left vocal cord is affected twice as often as the right, and females are affected more often than males (3:2). Diagnosis is based on direct visualization. An extensive assessment may be necessary to determine the cause. Several direct surgical approaches are available if treating the cause is not curative.

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.

Paralysis is usually unilateral. About one third of unilateral vocal cord paralyses are neoplastic in origin, one third are traumatic, and one third are idiopathic. Intracranial tumors, vascular accidents, and demyelinating diseases cause nucleus ambiguus paralysis. Tumors at the base of the skull and trauma to

the neck cause vagus paralysis. Recurrent laryngeal nerve paralysis is caused by neck or thoracic lesions (eg, aortic aneurysm; mitral stenosis; mediastinal tuberculous adenitis; tumors of the thyroid gland, esophagus, lung, or mediastinal structures), trauma, thyroidectomy, neurotoxins (eg, lead, arsenic, mercury), neurotoxic infections (eg, diphtheria), cervical spine injury or surgery, Lyme disease, and viral illness. Viral neuronitis probably accounts for most idiopathic cases.

Bilateral vocal cord paralysis is a life-threatening disorder caused by thyroid and cervical surgery, tracheal intubation, trauma, and neurodegenerative and neuromuscular diseases.

Symptoms and Signs

Vocal cord paralysis results in loss of vocal cord abduction and adduction. Paralysis 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, the cord may move with phonation but not with inspiration. In unilateral paralysis, the voice may be hoarse and breathy, but the airway is usually not obstructed because the normal cord abducts sufficiently. In bilateral paralysis, both cords generally lie within 2 to 3 mm of the midline, and the voice is of good quality but of limited intensity. The airway, however, is inadequate, resulting in stridor and dyspnea with moderate exertion as each cord is drawn to the midline glottis by an inspiratory Bernoulli effect. Aspiration is also a danger.

Diagnosis

• Laryngoscopy

• Various tests for possible causes

Diagnosis is based on laryngoscopy. The cause must always be sought. Evaluation is guided by abnormalities identified on history and physical examination. During the history, the physician asks about all possible causes of peripheral neuropathy, including chronic heavy metal exposure (arsenic, lead, mercury), drug effects from phenytoin and vincristine, and history of connective tissue disorders, Lyme disease, sarcoidosis, diabetes, and alcoholism. Further evaluation may include enhanced CT or MRI of the head, neck, and chest; thyroid scan; barium swallow or bronchoscopy; and esophagoscopy. Cricoarytenoid arthritis, which may cause fixation of the cricoarytenoid joint, must be differentiated from a neuromuscular etiology. Fixation is best documented by absence of passive mobility during rigid laryngoscopy under general anesthesia. Cricoarytenoid arthritis may complicate such conditions as RA, external blunt trauma, and prolonged endotracheal intubation.

Treatment

• For unilateral paralysis, surgical procedures to move cords closer together

• For bilateral paralysis, surgical procedures and measures to maintain airway

In unilateral paralysis, treatment is directed at improving voice quality through augmentation, medialization, or reinnervation.

Augmentation involves injecting a paste of plasticized particles, collagen, micronized dermis, or autologous fat into the paralyzed cord, bringing the cords closer together to improve the voice and prevent aspiration.

Medialization is shifting the vocal cord toward the midline by inserting an adjustable spacer laterally to the affected cord. This can be done with a local anesthetic, allowing the position of the spacer to be "tuned" to the patient's voice. Unlike augmentation with plasticized particles, which permanently fixes the cord, the spacer is both adjustable and removable.

Reinnervation has only rarely been successful.

In bilateral paralysis, an adequate airway must be reestablished. Tracheotomy may be needed permanently or temporarily during a URI. An arytenoidectomy with lateralization of the true vocal cord opens the glottis and improves the airway but may adversely affect voice quality. Posterior laser cordectomy opens the posterior glottis and may be preferred to endoscopic or open arytenoidectomy. Successful laser establishment of a posterior glottic airway usually obviates the need for long-term tracheotomy while preserving a serviceable voice quality.

Vocal Cord Polyps and Nodules

Acute trauma or chronic irritation causes changes in the vocal cords that can lead to polyps or nodules. Both cause hoarseness and a breathy voice. Persistence of these symptoms for > 3 wk dictates visualization of the vocal cords. Diagnosis is based on laryngoscopy and on biopsy to rule out cancer. Surgical removal restores the voice, and removal of the irritating source prevents recurrence.

Etiology

Polyps and nodules result from injury to the lamina propria of the true vocal cords. Polyps may occur at the mid third of the membranous cords and are more often unilateral. They frequently result from an initiating acute phonatory injury. Nodules usually occur bilaterally at the junction of the anterior and middle third of the cords. Their main cause is chronic voice abuse—yelling, shouting, singing loudly, or using an unnaturally low frequency. Polyps may have several other causes, including gastric reflux, untreated hypothyroid states, chronic laryngeal allergic reactions, or chronic inhalation of irritants, such as industrial fumes or cigarette smoke. Polyps tend to be larger and more protuberant than nodules and often have a dominant surface blood vessel.

Symptoms and Signs

Both result in slowly developing hoarseness and a breathy voice.

Diagnosis

• Laryngoscopy

• Sometimes biopsy

Diagnosis is based on direct or indirect visualization of the larynx with a mirror or laryngoscope. Biopsy of discrete lesions to exclude carcinoma is done by microlaryngoscopy.

Treatment

• Avoidance of cause

• For polyps, usually surgical removal

Correction of the underlying voice abuse cures most nodules and prevents recurrence. Removal of the offending irritants allows healing, and voice therapy with a speech therapist reduces the trauma to the vocal cords caused by improper singing or protracted loud speaking. Nodules usually regress with voice therapy alone.

Most polyps must be surgically removed to restore a normal voice. Cold-knife microsurgical excision during direct microlaryngoscopy is preferable to laser excision, which is more likely to cause collateral thermal injury if improperly applied.

In microlaryngoscopy, an operating microscope is used to examine, biopsy, and operate on the larynx. Images can be recorded on video as well. The patient is anesthetized, and the airway is secured by high-pressure jet ventilation through the laryngoscope, endotracheal intubation, or, for an inadequate upper airway, tracheotomy. Because the microscope allows observation with magnification, tissue can be

removed precisely and accurately, minimizing damage (possibly permanent) to the vocal mechanism. Laser surgery can be done through the optical system of the microscope to allow for precise cuts. Microlaryngoscopy is preferred for almost all laryngeal biopsies, for procedures involving benign tumors, and for many forms of phonosurgery.

Chapter 55. Tumors of the Head and Neck

Introduction

The most common noncutaneous tumor of the head and neck is squamous cell carcinoma of the larynx, followed by squamous cell carcinomas of the tongue, palatine tonsil, and floor of the mouth. Less common are tumors of the salivary glands, jaw, nose and paranasal sinuses, and ear. Tumors of the thyroid gland, eye, and skin are discussed elsewhere in THE MANUAL.

Excluding the skin and thyroid gland, > 90% of head and neck cancers are squamous cell (epidermoid) carcinomas, and 5% are melanomas, lymphomas, and sarcomas. Patients with sarcomas or carcinomas of the salivary glands or paranasal sinuses are often younger than patients with squamous cell carcinoma, who are more commonly in their mid-50s and older.

Etiology

The vast majority of patients, 85% or more, with cancer of the head and neck have a history of alcohol use, smoking, or both. Other suspected causes include use of snuff or chewing tobacco, sunlight exposure, previous x-rays of the head and neck, certain viral infections, ill-fitting dental appliances, chronic candidiasis, and poor oral hygiene. In India, oral cancer is extremely common, probably because of chewing betel quid (a mixture of substances, also called paan). Long-term exposure to sunlight and the use of tobacco products are the primary causes of squamous cell carcinoma of the lower lip.

Patients who in the past were treated with radiation for acne, excess facial hair, enlarged thymus, or hypertrophic tonsils and adenoids are predisposed to thyroid and salivary gland cancers and benign salivary tumors.

Epstein-Barr virus plays a role in the pathogenesis of nasopharyngeal cancer, and serum measures of certain Epstein-Barr virus proteins may be biomarkers of recurrence. Human papillomavirus seems to be associated with head and neck squamous cell carcinoma, particularly oropharyngeal cancer. The mechanism for viral-mediated tumor genesis may be distinct from tobacco-related pathways and seem to have a different, better, prognosis.

Symptoms and Signs

Most head and neck cancers first manifest as an asymptomatic neck mass, painful mucosal ulceration, or visible mucosal lesion (eg, leukoplakia, erythroplakia). Subsequent symptoms depend on location and extent of the tumor and include pain, paresthesia, nerve palsies, trismus, and halitosis. Otalgia is an often overlooked symptom usually representing referred pain from the primary tumor. Weight loss caused by perturbed eating and odynophagia is also common.

Diagnosis

• Clinical evaluation

• Biopsy

• Imaging tests and endoscopy to evaluate extent of disease

Routine physical examination (including a thorough oral examination) is the best way to detect cancers early before they become symptomatic. Commercially available brush biopsy kits help screen for oral cancers. Any head and neck symptom (eg, sore throat, hoarseness, otalgia) lasting > 2 to 3 wk should prompt referral to a head and neck specialist.

Definitive diagnosis usually requires a biopsy. Additional important information is obtained from a combination of imaging tests (eg, CT, MRI, PET/CT), endoscopy, and fine-needle aspiration of any neck mass.

Staging

Head and neck cancers may remain localized for months to years. Local tissue invasion eventually is followed by metastasis to regional lymph nodes, related in large part to tumor size and extent, and reduces overall survival by nearly half. Distant metastases tend to occur late, usually in patients with advanced tumor and nodal stages. Metastases occur more commonly among immunocompromised patients. Common sites of distant metastases are the lungs, liver, bone, and brain.

Head and neck cancers are staged (see Table 55-1) according to size and site of the primary tumor (T), number and size of metastases to the cervical lymph nodes (N), and evidence of distant metastases (M). Staging usually requires imaging with CT, MRI, or both, and often PET.

Prognosis

Prognosis is favorable if diagnosis is early and treatment is timely and appropriate. In general, the more poorly differentiated the cancer, the greater the chance of regional and distant metastases. The presence of regional nodal spread reduces overall survival by nearly half. Distant metastasis greatly reduces survival, having only rare cures. Local invasion, a criterion for advanced T stage, with invasion of muscle, bone, or cartilage, also significantly decreases cure rate. Perineural spread, as evidenced by pain, paralysis, or numbness, indicates a highly aggressive tumor, is associated with nodal metastasis, and has a less favorable prognosis than a similar lesion without perineural invasion.

With appropriate treatment, 5-yr survival can be as high as 90% for stage I, 75 to 80% for stage II, 45 to 75% for stage III, and up to 40% for stage IV. The survival rates vary greatly depending on the primary site. Stage I laryngeal cancers have an excellent survival rate when compared to other sites.

Treatment

• Surgery, radiation therapy, or both

• Sometimes chemotherapy

Many stage I tumors, regardless of location, respond similarly to surgery and to radiation therapy, allowing other factors (eg, patient preference) to determine choice of therapy. Thus, the treating physician should carefully review risks and benefits with the patient. However, at certain locations, there is clear superiority of one modality over another. For

[**Table 55-1.** Staging of Head and Neck Cancer]

example, surgery is the better treatment for early-stage disease involving the oral cavity. In select head and neck cancers, endoscopic surgery has cure rates similar to those of open surgery or radiation, and morbidity is significantly less. However, many physicians still recommend radiation for early-stage laryngeal cancer.

If radiation therapy is chosen for primary therapy, it is delivered to the primary site and sometimes bilaterally to the cervical lymph nodes. The treatment of lymphatics, whether by radiation or surgery, is determined by the primary site, histologic criteria, and risk of nodal disease.

Advanced-stage disease (stages III and IV) often requires multimodality treatment, incorporating some combination of chemotherapy, radiation therapy, and surgery. Bone or cartilage invasion requires surgical resection of the primary site and usually regional lymph nodes because of the high risk of nodal spread. If the primary site is treated surgically, then postoperative radiation to the cervical lymph nodes is delivered if there are high-risk features, such as multiple lymph nodes with cancer or extracapsular extension. Postoperative radiation usually is preferred over preoperative radiation, because radiated tissues heal poorly. Recent studies have shown that adding chemotherapy to adjuvant radiation therapy to the neck improves regional control of the cancer and improves survival. There are significant risks to this approach, so the decision to add chemotherapy should be carefully considered.

Advanced squamous cell carcinoma without bony invasion often is treated with concomitant chemotherapy and radiation therapy. Although advocated as organ-sparing, combining chemotherapy with radiation therapy doubles the rate of acute toxicities, particularly severe dysphagia. Radiation may be used alone for debilitated patients with advanced disease who cannot tolerate the sequelae of chemotherapy and are too high a risk for general anesthesia.

Primary chemotherapy is reserved for chemosensitive tumors, such as Burkitt's lymphoma, or for patients who have widespread metastases (eg, hepatic or pulmonary involvement). Several drugs—cisplatin, fluorouracil, bleomycin, and methotrexate—provide palliation for pain and shrink the tumor in patients who cannot be treated with other methods. Response may be good initially but is not durable, and the cancer will return.

Tumor recurrence: Managing recurrent tumors after therapy is complex and has potential complications. A palpable mass or ulcerated lesion with edema or pain at the primary site after therapy strongly suggests a persistent tumor. Such patients require CT (with thin cuts) or MRI. For local recurrence after surgical treatment, all scar planes and reconstructive flaps are excised along with residual cancer. Radiation therapy, chemotherapy, or both may be done but have limited effectiveness. Patients with recurrence after radiation therapy should not receive additional radiation and are best treated with surgery.

Symptom control: Pain is a common symptom in patients with head and neck cancer and must be adequately addressed. Palliative surgery or radiation may temporarily alleviate pain, and in 30 to 50% of patients, chemotherapy can produce improvement that lasts a mean of 3 mo. A stepwise approach to pain management, as recommended by the WHO, is critical to controlling pain. Severe pain is best managed in association with a pain and palliative care specialist.

Pain, difficulty eating, choking on secretions, and other problems make adequate symptomatic treatment essential. Patient directives regarding such care should be clarified early (see p. 3471).

Adverse effects of treatment: All cancer treatments have potential complications and expected sequelae. Because many treatments have similar cure rates, the choice of modality is based largely on real, or perceived, differences in sequelae.

Although it is commonly thought that surgery requires rehabilitation for swallowing and speaking, many procedures do not require such rehabilitation. Increasingly complex reconstructive procedures and techniques, including prostheses, grafts, regional pedicle flaps, and complex free flaps, can restore function and appearance often to near normal.

Toxic effects of chemotherapy include malaise, severe nausea and vomiting, mucositis, transient hair loss, gastroenteritis, hematopoietic and immune suppression, and infection.

Therapeutic radiation for head and neck cancers has several adverse effects. The function of any salivary gland within the beam is permanently destroyed by a dose of about 40 Gy, resulting in xerostomia, which markedly increases the risk of dental caries. Newer radiation techniques, such as intensity-modulated radiation therapy, can minimize or eliminate toxic doses to the parotid glands in certain patients. Radioprotectant drugs (eg, amifostine) also can help protect salivary function. In addition, the blood supply of bone, particularly in the mandible, is compromised by doses of > 60 Gy, and osteoradionecrosis may occur (see also p. 505). In this condition, tooth extraction sites break down, sloughing bone and soft tissue. Therefore, any needed dental treatment, including scaling, fillings, and extractions, should be done before radiation therapy. Any teeth in poor condition that cannot be rehabilitated should be extracted. Radiation therapy may also cause oral mucositis and dermatitis in the overlying skin, which may result in dermal fibrosis. Loss of taste (ageusia) and impaired smell (dysosmia) often occur but are usually transient.

Prevention

Removing risk factors is critical, and all patients should cease tobacco use and limit alcohol consumption. Removing risk factors also helps prevent disease recurrence in those treated for cancer. A new primary

cancer develops in about 5% of patients/yr (to a maximum risk of about 20%); risk is lower in those who stop.

Cancer of the lower lip may be prevented by sunscreen use and tobacco cessation. Because 60% of head and neck cancers are well advanced (stage III or IV) at the time of diagnosis, the most promising strategy for reducing morbidity and mortality is diligent routine examination of the oral cavity.

Jaw Tumors

Numerous tumor types, both benign and malignant, originate in the jaw. Symptoms are swelling, pain, tenderness, and unexplained tooth mobility; some tumors are discovered on routine dental x-rays, whereas others are found on routine examinations of the oral cavity and teeth. Treatment depends on location and tumor type. Benign tumors may be observed and may not need surgical excision, although most tumors require resection with possible reconstruction.

If not initially detected on x-ray, jaw tumors are diagnosed clinically because their growth causes swelling of the face, palate, or alveolar ridge (the part of the jaw supporting the teeth). They can also cause bone tenderness and severe pain.

Bony outgrowths (torus palatinus, torus mandibularis) may develop on the palate or mandible. These are common growths and may prompt concerns about cancer, although they are benign and of concern only if they interfere with dental care or function of the submandibular gland. When on the palate, they are in the midline and have intact, smooth mucosa. Multiple osteomas seen on dental x-ray may suggest Gardner's syndrome.

The most common tumor of the mandible and maxilla is squamous cell carcinoma invading the bone through dental sockets. These can involve any portion of the intraoral mandible or maxilla.

Ameloblastoma, the most common epithelial odontogenic tumor, usually arises in the posterior mandible. It is slowly invasive and rarely metastatic. On x-ray, it typically appears as multiloculated or soap-bubble radiolucency. Treatment is wide surgical excision and reconstruction if appropriate.

Odontoma, the most common odontogenic tumor, affects the dental follicle or the dental tissues and usually appears in the mandibles of young people. Odontomas include fibrous odontomas and cementomas. A clinically absent molar tooth suggests a composite odontoma. Typically, no treatment is indicated. These tumors may be excised when the diagnosis is in doubt.

Osteosarcoma, giant cell tumor, Ewing's tumor, multiple myeloma, and metastatic tumors may affect the jaw. Treatment is the same as for those tumors in other bony sites.

Laryngeal Cancer

Ninety percent of laryngeal cancer is squamous cell carcinoma. Smoking, alcohol abuse, lower socioeconomic status, and being male and > 60 yr increase risk. Early diagnosis is common with vocal cord tumors because vocal, swallowing, or respiratory symptoms develop early. However, supraglottic tumors (above the vocal cords) and subglottic tumors (below the vocal cords) are often very large and at an advanced stage when diagnosed because they are asymptomatic until obstructive symptoms develop. Diagnosis is based on laryngoscopy and biopsy. Treatment of early-stage tumors is with surgery or radiation. Advanced-stage tumors are most often treated with chemotherapy and radiation therapy. Surgery is reserved for salvage treatment or lesions with significant extralaryngeal extension or cartilage destruction. Reestablishment of speaking ability is needed if a total laryngectomy is done.

Squamous cell carcinoma is the most common cancer of the larynx. In the US, it is 4 times more common among men and is more common among those of lower socioeconomic status. Over 95% of patients are smokers; 15 pack-years of smoking increase the risk 30-fold. The incidence of larynx cancer is decreasing, particularly among men, most likely due to changes in smoking habits.

Sixty percent of patients present with localized disease alone; 25% present with local disease and regional nodal metastatic disease; and 15% present with advanced disease, distant metastases, or both. Distant metastases occur most frequently in the lungs and liver.

Common sites of origin are the true vocal cords (glottis) and the supraglottic larynx. The least common site is the subglottic larynx, where only 1% of primary laryngeal cancers originate. Verrucous carcinoma, a rare variant of squamous cell carcinoma, usually arises in the glottic area and has a better survival rate than standard squamous cell carcinoma.

Symptoms and Signs

Symptoms and signs differ based on the involved portion of the larynx. Hoarseness is common early in glottic cancers but is a late symptom for supraglottic and subglottic cancers. Supraglottic cancer is often asymptomatic until it manifests as a mass lesion (eg, with airway obstruction, dysphagia, otalgia, or a "hot potato" voice) or with weight loss. Such patients should be referred for indirect laryngoscopy without delay.

Diagnosis

• Laryngoscopy

• Operative endoscopy and biopsy

• Imaging tests for staging

All patients who have hoarseness for > 2 to 3 wk should have their larynx examined by a head and neck specialist. Any lesions discovered require further evaluation, usually with operative endoscopy and biopsy, with concomitant evaluation of the upper airway and GI tract for coexisting cancers. The incidence of a synchronous second primary tumor may be as high as 10%.

Patients with confirmed carcinoma typically have neck CT with contrast and a chest x-ray or chest CT. Most clinicians also do PET of the neck and chest at the time of diagnosis.

Treatment

• Early-stage: Surgery or radiation therapy

• Advanced: Radiation therapy and sometimes chemotherapy

For early-stage glottic carcinoma, laser excision, radiation therapy, or occasionally open laryngeal surgery results in a 5-yr survival rate of 85 to 95%. Endoscopic laser resection and radiation therapy usually preserve a normal voice and post-treatment function and have similar cure rates.

For advanced glottic carcinoma, defined by a lack of vocal cord mobility, thyroid cartilage invasion, or extension into the tongue, most patients are treated with chemotherapy and radiation therapy. Surgery (followed by radiation therapy) is reserved for salvage situations; most such cases require total laryngectomy, although endoscopic or open partial laryngectomy may sometimes be used. Extensive local invasion, however, usually requires an initial total laryngectomy rather than nonsurgical therapy.

Early supraglottic carcinoma can be effectively treated with radiation therapy or partial laryngectomy. Laser resection has shown considerable success on early-stage supraglottic squamous cell carcinomas and minimizes functional changes after surgery. If the carcinoma is more advanced but does not affect the true vocal cords, a supraglottic partial laryngectomy can be done to preserve the voice and glottic sphincter. If the true vocal cords also are affected, a supracricoid laryngectomy or a total laryngectomy is required if surgery is chosen. As with glottic carcinoma, most advanced-stage supraglottic cancers initially are treated with chemotherapy and radiation therapy.

Treatment of hypopharyngeal carcinomas is similar to that of laryngeal cancer. Early-stage lesions usually are treated with radiation alone, although endoscopic resection is an option. However, the majority of patients with hypopharyngeal cancer have advanced-stage disease, because of the silent nature of the disease and frequent regional lymphatic spread; such patients are treated with chemotherapy and radiation therapy primarily, with surgical salvage.

Rehabilitation: Rehabilitation may be required after either surgical or nonsurgical treatment. Significant swallowing problems are common after chemotherapy and radiation therapy and may require esophageal dilation, swallowing therapy, or, in severe cases, surgical replacement of the pharynx or gastrostomy tube feedings. Swallowing also is affected by surgery and may require swallowing therapy or dilation as well.

Speech, on the other hand, is more significantly affected by surgery. After total laryngectomy, the patient requires creation of a new voice by way of

• Esophageal speech

• A tracheoesophageal puncture

• An electrolarynx

In all 3 techniques, sound is articulated into speech by the pharynx, palate, tongue, teeth, and lips.

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 puncture involves placement of a one-way valve between the trachea and esophagus to facilitate phonation. This valve forces air into the esophagus during expiration to produce a sound. Patients receive physical rehabilitation, speech therapy, and appropriate training in the maintenance and use of this valve and must be cautioned against the possible aspiration of food, fluids, and secretions.

An electrolarynx is a battery-powered sound source that is held against the neck to produce sound. Although it carries a great deal of social stigma for many patients, it has the advantage of being functional immediately with little or no training.

Nasopharyngeal Cancer

Nasopharyngeal cancers are rare in the US but common in the South China Sea region. Symptoms develop late, including unilateral bloody nasal discharge, nasal obstruction, facial swelling, and numbness. Diagnosis is based on inspection and biopsy, with CT, MRI, or PET to evaluate extent. Treatment is with radiation, chemotherapy, and, rarely, surgery.

Squamous cell carcinoma is the most common malignant tumor of the nasopharynx. It can occur in any age group and is rare in North America. It is one of the most common cancers among people of Chinese, especially southern Chinese, and Southeast Asian ancestry, including Chinese immigrants to North America. Over several generations, the prevalence among Chinese-Americans gradually decreases to that among non-Chinese Americans, suggesting an environmental component to etiology. Dietary exposure to nitrites and salted fish also is thought to increase risk. Epstein-Barr virus is a significant risk factor, and there is hereditary predisposition. Other nasopharyngeal cancers include adenoid cystic and mucoepidermoid carcinomas, malignant mixed tumors, adenocarcinomas, lymphomas, fibrosarcomas, osteosarcomas, chondrosarcomas, and melanomas.

Symptoms and Signs

The first symptom is often nasal or eustachian tube obstruction causing hearing loss due to a middle ear effusion. Other symptoms include purulent bloody rhinorrhea, frank epistaxis, cranial nerve palsies, and cervical lymphadenopathy. Cranial nerve palsies most often involve the 6th, 4th, and 3rd cranial nerves due to their location in the cavernous sinus, in close proximity to the foramen lacerum, which is the most

common route of intracranial spread for these tumors. Because lymphatics of the nasopharynx communicate across the midline, bilateral metastases are common.

Diagnosis

• Nasopharyngeal endoscopy and biopsy

• Imaging tests for staging

Patients suspected of having nasopharyngeal cancer must undergo examination with a nasopharyngeal mirror or endoscope, and lesions are biopsied. Open cervical node biopsy should not be done as the initial procedure (see Neck Mass on p. 461), although a needle biopsy is acceptable and often recommended. Gadolinium-enhanced MRI (with fat suppression) of the head with attention to the nasopharynx and skull base is done; the skull base is involved in about 25% of patients. CT also is required to accurately assess skull base bony changes, which are less visible on MRI. A PET scan also commonly is done to assess the extent of disease as well as the cervical lymphatics.

Treatment

• Chemotherapy plus radiation therapy

Because of the location and extent of involvement, nasopharyngeal cancers often are not amenable to surgical resection. They are typically treated with chemotherapy and radiation therapy, which are often followed by adjuvant chemotherapy.

Recurrent tumors can be treated with another course of radiation, commonly with brachytherapy; radionecrosis of the skull base is a risk. An alternative to radiation is skull base resection.

Oral Squamous Cell Carcinoma

Oral squamous cell carcinoma affects about 30,000 Americans each year. Over 95% smoke, drink alcohol, or both. Early, curable lesions are rarely symptomatic; thus, preventing fatal disease requires early detection by screening. Treatment is with surgery, radiation, or both. The overall 5-yr survival rate (all sites and stages combined) is > 50%.

In the US, 3% of cancers in men and 2% in women are oral squamous cell carcinomas, most of which occur after age 50. Squamous cell carcinoma is the most common oral or pharyngeal cancer (and the most common at head and neck sites in general).

The chief risk factors for oral squamous cell carcinoma are smoking (especially > 2 packs/day) and alcohol use. Risk increases dramatically when alcohol use exceeds 6 oz of distilled liquor, 6 oz of wine, or 12 oz of beer/day. The combination of heavy smoking and alcohol abuse is estimated to raise the risk 100-fold in women and 38-fold in men. Squamous cell carcinoma of the tongue may also result from any chronic irritation, such as dental caries, overuse of mouthwash, chewing tobacco, or the use of betel quid. Oral human papillomavirus (HPV), typically acquired via oral-genital contact, may have a role in etiology.

About 40% of intraoral squamous cell carcinomas begin on the floor of the mouth or on the lateral and ventral surfaces of the tongue. About 38% of all oral squamous cell carcinomas occur on the lower lip; these are usually solar-related cancers on the external surface. About 11% begin in the palate and tonsillar area. Squamous cell carcinoma of the tonsil (an oropharyngeal cancer), 2nd in frequency only to carcinoma of the larynx among cancers of the upper respiratory tract, occurs predominantly in males.

Symptoms and Signs

Oral lesions are asymptomatic initially, highlighting the need for oral screening. Most dental professionals carefully examine the oral cavity and oropharynx during routine care and may do a brush biopsy of abnormal areas. The lesions may appear as areas of erythroplakia or leukoplakia and may be exophytic

or ulcerated. Cancers are often indurated and firm with a rolled border. Tonsillar carcinoma usually manifests as an asymmetric swelling and sore throat, with pain often radiating to the ipsilateral ear. A metastatic mass in the neck may be the first symptom, particularly in tonsillar cancer.

Diagnosis

• Biopsy

• Endoscopy to detect second primary cancer

• Chest x-ray and CT of head and neck

Biopsy of suspect areas is done. Direct laryngoscopy, bronchoscopy, and esophagoscopy are done to exclude a simultaneous second primary cancer. Head and neck CT usually is done. Chest x-ray is done; chest CT is done if an advanced stage is suspected or confirmed.

Prognosis

If carcinoma of the tongue is localized (no lymph node involvement), 5-yr survival is > 50%. For localized carcinoma of the floor of the mouth, 5-yr survival is 65%. Lymph node metastasis decreases survival rate by about 50%. Metastases reach the regional lymph nodes first and later the lungs.

For lower lip lesions, 5-yr survival is 90%, and metastases are rare. Carcinoma of the upper lip tends to be more aggressive and metastatic. For carcinoma of the palate and tonsillar area, 5-yr survival is 68% if patients are treated before lymph node involvement but only 17% after involvement. The prognosis for tonsillar carcinoma is often better stage for stage than that for oral cancers. Oropharyngeal cancer associated with HPV infection may have a better prognosis.

Treatment

• Surgery or radiation therapy

Surgery and radiation therapy are the treatments of choice. Regional or distant disease necessitates a more radical treatment approach.

For tongue lesions, surgery is usually the initial treatment, particularly for early-stage disease. Selective neck dissection is indicated if the risk of nodal disease exceeds 15 to 20%. Routine surgical reconstruction is the key to reducing postoperative oral disabilities; procedures range from local tissue flaps to free tissue transfers. Speech and swallowing therapy may be required after significant resections. Radiation therapy is an alternative treatment. Chemotherapy is not used routinely but is recommended on an individual basis; rare distant metastases are present in sites where chemotherapy may be of some palliative value (eg, lung, bone, heart, pericardium).

Treatment of squamous cell carcinoma of the lip is surgical excision with reconstruction to maximize postoperative function. When large areas of the lip exhibit premalignant change, the lip can be surgically shaved, or a laser can remove all affected mucosa. Thereafter, appropriate sunscreen application is recommended.

Treatment of tonsillar carcinoma usually consists of concomitant chemotherapy and radiation therapy. Another option includes radical resection of the tonsillar fossa, sometimes with partial mandibulectomy and neck dissection.

Otic Tumors

A number of malignant and benign otic tumors occur, usually manifesting with hearing loss. They may also manifest with dizziness, vertigo, or imbalance. These tumors are rare and can be difficult to diagnose.

Malignant otic tumors: Basal cell and squamous cell carcinomas may arise in the ear canal. Persistent inflammation caused by 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 done when lesions are limited to the canal and have not invaded the middle ear. Deeper invasion requires a more significant temporal bone resection.

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

Nonchromaffin paragangliomas (chemodectomas) arise in the temporal bone from glomus bodies in the jugular bulb (glomus jugulare tumors) or the medial wall of the middle ear (glomus tympanicum tumors). They appear as a pulsatile red mass in the middle ear. The first symptom often is tinnitus that is synchronous with the pulse. Hearing loss develops, followed by vertigo. Cranial nerve palsies of the 9th, 10th, or 11th nerve may accompany glomus jugulare tumors that extend through the jugular foramen. Excision is the treatment of choice, and radiation is used for nonsurgical candidates.

Benign otic tumors: Sebaceous cysts, osteomas, and keloids may arise in and occlude the ear canal, causing retention of cerumen and conductive hearing loss. Excision is the treatment of choice for all benign otic tumors.

Ceruminomas occur in the outer third of the ear canal. These tumors appear benign histologically and do not metastasize regionally or distantly but they are locally invasive and potentially destructive and should be excised widely.

Salivary Gland Tumors

Most salivary gland tumors are benign and occur in the parotid glands. A painless salivary mass is the most common sign and is evaluated by fine-needle aspiration biopsy. Imaging with CT and MRI can be helpful. For malignant tumors, treatment is with excision and radiation. Long-term results are related to the grade of the cancer.

About 85% of salivary gland tumors occur in the parotid glands, followed by the submandibular and minor salivary glands, and about 1% occur in the sublingual glands. About 75 to 80% are benign, slow-growing, movable, painless, usually solitary nodules beneath normal skin or mucosa. Occasionally, when cystic, they are soft but most often they are firm.

Benign tumors: The most common type is a pleomorphic adenoma (mixed tumor). Malignant transformation is possible, resulting in carcinoma ex mixed tumor, but this usually occurs only after the benign tumor has been present for 15 to 20 yr. If malignant transformation occurs, the cure rates are very low, despite adequate surgery and adjuvant therapy.

Other benign tumors include monomorphic adenoma, oncocytoma, and papillary cystadenoma lymphomatosum (previously known as cylindroma). These tumors rarely recur and rarely become malignant.

Malignant salivary gland tumors: Malignant tumors are less common and are characterized by rapid growth or a sudden growth spurt. They are firm, nodular, and can be fixed to adjacent tissue, often with a poorly defined periphery. Pain and neural involvement are common. Eventually, the overlying skin or mucosa may become ulcerated or the adjacent tissues may become invaded. Surgery, followed by radiation therapy, is the treatment of choice for resectable disease. Currently, there is no effective chemotherapy for salivary cancer.

Mucoepidermoid carcinoma is the most common salivary gland cancer, typically occurring in people in their 20s to 50s. It can manifest in any salivary gland, often in a minor salivary gland of the palate, or it can occur deep within the bone, such as in the wall of a dentigerous cyst. Intermediate and high-grade mucoepidermoid carcinomas may metastasize to the regional lymphatics, which must be addressed with surgical dissection or postoperative radiation therapy.

Adenoid cystic carcinoma is the most common malignant tumor of minor salivary glands (and of the trachea). It is a slowly growing malignant transformation of a much more common benign cylindroma. Its peak incidence is between ages 40 and 60, and symptoms include severe pain and, often, facial nerve paralysis. It has a propensity for perineural invasion and spread, with disease potentially extending many centimeters from the main tumor mass. Lymphatic spread is not a common feature of this tumor, so elective nodal treatment is less common. Although the 5- and 10-yr survival rates are quite good, the 15- and 20-yr rates are quite poor, with most patients developing distant metastases. Pulmonary metastases are common, although patients can live quite long with them.

Acinic cell carcinoma, a common parotid tumor, occurs in people in their 40s and 50s. This carcinoma has a more indolent course, as well as an incidence of multifocality.

Carcinoma ex mixed tumor is adenocarcinoma arising in a preexisting benign carcinoma ex mixed tumor. Only the carcinomatous element metastasizes.

Symptoms and Signs

Most benign and malignant tumors manifest as a painless mass. However, malignant tumors may invade nerves, causing localized or regional pain, numbness, paresthesia, causalgia, or a loss of motor function.

Diagnosis

• Biopsy

• CT and MRI for extent of disease

CT and MRI locate the tumor and describe its extent. Biopsy confirms the cell type. A search for spread to regional nodes or distant metastases in the lung, liver, bone, or brain may be indicated before treatment is selected.

Treatment

• Surgery, sometimes plus radiation therapy

Treatment of benign tumors is surgery. The recurrence rate is high when excision is incomplete.

Treatment of mucoepidermoid carcinoma consists of wide excision and postoperative radiation. The 5-yr survival rate is 95% with the low-grade type, primarily affecting mucus cells, and 50% with the high-grade type, primarily affecting epidermoid cells. Treatment of adenoid cystic carcinoma is wide surgical excision, but local recurrence is common. Lung metastases and death are likely, although many years, to a decade or more, after the initial diagnosis and treatment. The prognosis for acinic cell carcinoma is favorable after wide excision. All surgeries are designed to spare the facial nerve, which is sacrificed only in cases of direct tumor involvement with the nerve.

Chapter 56. Approach to Dental and Oral Symptoms

Introduction

A physician should always examine the mouth and be able to recognize major oral disorders, particularly possible cancers. However, consultation with a dentist is needed to evaluate nonmalignant changes as well as patients with tooth problems. Likewise, patients with xerostomia or unexplained swelling or pain in the mouth, face, or neck require a dental consultation. Children with abnormal facies (who also may have dental malformations requiring correction) should be evaluated by a dentist. In FUO or a systemic infection of unknown cause, a dental disorder should be considered. A dental consultation is necessary before head and neck radiation therapy and is advisable before chemotherapy.

Clues suggesting systemic disease may be found in the mouth and adjacent structures (see Table 56-1). A dentist should consult a physician when a systemic disorder is suspected, when the patient is taking certain drugs (eg, warfarin, bisphosphonates), and when a patient's ability to withstand general anesthesia or extensive oral surgery must be evaluated. Patients with certain heart valve abnormalities may require antibiotic prophylaxis to help prevent bacterial endocarditis before undergoing certain dental procedures (see Tables 215-3 and 215-4 on pp. 2199 and 2200).

Common dental disorders are discussed in Ch. 57. Dental emergencies, including toothache, are discussed in Ch. 58.

Anatomy and Development

Teeth: The teeth are categorized as incisors, canines, premolars, and molars and conventionally are numbered beginning with the maxillary right 3rd molar (see Fig. 56-1).

Each tooth has a crown and a root. The canines have the largest and strongest roots. An inner pulp contains blood vessels, lymphatics, and nerves, surrounded by the hard but porous dentin, a very hard enamel coating that covers the crown. The bonelike cementum is over the root, which, when healthy, is covered by gingiva (see Fig. 56-2). Twenty deciduous

[**Table 56-1.** Oral Findings in Systemic Disorders]

teeth normally begin appearing at close to age 6 mo and should all be in place by age 30 mo (see Table 271-1 on p. 2757). These teeth are followed by 32 permanent teeth that begin to appear by about age 6. The period from age 6 to 11 is called the mixed dentition stage, in which both deciduous and permanent teeth are present. Timing of tooth eruption is one indicator of skeletal age and may identify growth retardation or establish age for forensic purposes.

Supporting tissues: The gingiva surrounds the teeth at the base of their crown. The alveolar ridges are trabecular bone containing sockets for the teeth. The periodontium consists of the tissues that support the teeth—the gingiva, epithelial attachment, connective tissue attachment, periodontal ligament, and alveolar bone. The mandible and maxilla support the alveolar ridges and house the teeth. Saliva from the salivary glands bathes and protects the teeth. The tongue directs food between the grinding surfaces and helps clean the teeth. The maxilla receives innervation from the maxillary nerve, the 2nd division of the trigeminal nerve (the 5th cranial nerve). The mandibular nerve, which is the 3rd and most inferior division of the trigeminal nerve, innervates the mandible.

In the elderly, or in some periodontal diseases, gingival recession exposes the dental root adjacent to the crown, making root caries common. If tooth destruction results and the tooth must be removed, the mechanical stimulation necessary for maintaining bone integrity ceases. Consequently, atrophy of the alveolar ridge (senile atrophy) begins when teeth are absent.

Mouth: Normally, keratinized epithelium occurs on the facial aspect of the lips, dorsum of the tongue, hard palate, and gingiva around the teeth. When healthy, the gingiva extends

[Fig. 56-1. Identifying the teeth.]

5 to 7 mm from the tooth. Nonkeratinized mucosa occurs over alveolar bone further from the teeth, inside the lips and cheeks, on the sides and undersurface of the tongue, on the soft palate, and covering the floor of the mouth. The skin and mucosa of the lips are demarcated by the vermilion border.

The buccal mucosa, including the vestibule and nonkeratinized alveolar mucosa, is usually smooth, moist, and more red than pink (as compared to healthy gingiva). Innocuous entities in this region include linea alba (a thin white line, typically bilateral, on the level of the occlusal plane, where the cheek is bitten), Fordyce's granules (aberrant sebaceous glands appearing as < 1 mm light yellow spots that also may occur on the lips), and white sponge nevus (bilateral thick white folds over most of the buccal mucosa). Recognizing these avoids needless biopsy and apprehension. The orifices of the parotid (Stensen's) ducts are opposite the maxillary 1st molar on the inside of each cheek and should not be mistaken for an abnormality.

The dorsal surface of the tongue is covered with numerous whitish elevations called the filiform papillae. Interspersed among them are isolated reddish prominences called the fungiform papillae, occurring mostly on the anterior part of the tongue. The circumvallate papillae, numbering 8 to 12, are considerably larger and lie posteriorly in a V pattern. The circumvallate papillae do not project from the tongue but instead are surrounded by a trench. The foliate papillae appear as a series of parallel, slitlike folds on the lateral borders of the tongue, near the anterior pillars of the fauces. They vary in length and can easily be confused with malignant lesions, as may the foramen cecum, median rhomboid glossitis, and, rarely, a lingual thyroid nodule. Lingual tonsils are components of Waldeyer's ring, are at the back of the tongue, and should not be mistaken for lesions. If an apparent abnormality is bilateral, it is almost always a normal variant.

Innervation is supplied by the lingual nerves (branches of the 5th cranial nerves), for general sensory innervation, and the chorda tympani fibers (of the 7th cranial nerve), which innervate the taste buds of the anterior two thirds of the tongue. Behind the circumvallate papillae, the glossopharyngeal nerves (9th cranial nerves) provide the sensations of touch and taste. The tongue has taste receptors for sweet, salty, sour, bitter, and umami (a savory

[Fig. 56-2. Section of a canine tooth.]

taste triggered by natural glutamic acid and glutamates such as the flavoring agent monosodium glutamate). Although previously thought to be isolated to particular portions of the tongue, these receptors are now known to be distributed over the surface of the tongue. The hypoglossal nerves (12th cranial nerves) control movement of the tongue.

The major salivary glands are the paired parotid, submandibular, and sublingual glands. Most oral mucosal surfaces contain many minor mucus-secreting salivary glands. Anteriorly and near the midline on each side of the floor of the mouth are the openings of Wharton's ducts, which drain the ipsilateral submandibular and sublingual glands. The parotid glands drain into the cheeks via Stensen's ducts.

Evaluation

The first routine dental examination should take place by age 1 yr or when the first tooth erupts. Subsequent evaluations should take place at 6-mo intervals or whenever symptoms develop. Examination of the mouth is part of every general physical examination. Oral findings in many systemic diseases are unique, sometimes pathognomonic, and may be the first sign of disease. Oral cancer may be detected at an early stage.

History: Important dental symptoms include bleeding, pain, malocclusion, new growths, numbness or paresthesias, and chewing problems (which may lead to weight loss—see Table 56-2). General information includes use of alcohol or tobacco (both major risk factors for head and

neck cancer) and systemic symptoms, such as fever and weight loss.

Physical examination: A thorough inspection requires good illumination, a tongue blade, gloves, and a gauze pad. Complete or partial dentures are removed so that underlying soft tissues can be seen.

Most physicians use a head-mounted light. However, because the light cannot be precisely aligned on the axis of vision, it is difficult to avoid shadowing in narrow areas. Better illumination results with a head-mounted convex mirror; the physician looks through a hole in the center of the mirror, so the illumination is always on-axis. The head mirror reflects light from a source (any incandescent light) placed behind the patient and slightly to one side and requires practice to use effectively.

The examiner initially looks at the face for asymmetry, masses, and skin lesions. Slight facial asymmetry is universal, but more marked asymmetry may indicate an underlying disorder, either congenital or acquired (see
Table 56-3).

Teeth are inspected for shape, alignment, defects, mobility, color, and presence of adherent plaque, materia alba (dead bacteria, food debris, desquamated epithelial cells), and calculus (tartar).

Teeth are gently tapped with a tongue depressor or mirror handle to assess tenderness (percussion sensitivity). Tenderness to percussion suggests deep caries that has caused a necrotic pulp with periapical abscess or severe periodontal disease. Percussion sensitivity or pain on biting also can indicate an incomplete (green stick) fracture of a tooth. Percussion tenderness in multiple adjacent maxillary teeth may result from maxillary sinusitis. Tenderness to palpation around the apices of the teeth also may indicate an abscess.

Loose teeth usually indicate severe periodontal disease but can be caused by bruxism (clenching or grinding of teeth—see p. 506) or trauma that damages periodontal tissues. Rarely, teeth become loose when alveolar bone is eroded by an underlying mass (eg, ameloblastoma, eosinophilic granuloma). A tumor or systemic cause of alveolar bone loss (eg, diabetes mellitus, hyperparathyroidism, osteoporosis, Cushing's syndrome) is suspected when teeth are loose and heavy plaque and calculus are absent.

Calculus is mineralized bacterial plaque—a concretion of bacteria, food residue, saliva,

[**Table 56-2.** Some Oral Symptoms and Possible Causes]

and mucus with Ca and phosphate salts. After a tooth is cleaned, a mucopolysaccharide coating (pellicle) is deposited almost immediately. After about 24 h, bacterial colonization turns the pellicle into plaque. After about 72 h, the plaque starts calcifying, becoming calculus. When present, calculus is deposited most heavily on the lingual (inner, or tongue) surfaces of the mandibular anterior teeth near the submandibular and sublingual duct orifices (Wharton's ducts) and on the buccal (cheek) surfaces of the maxillary molars near the parotid duct orifices (Stensen's ducts).

Caries (tooth decay—see p. 516) first appears as defects in the tooth enamel. Caries then appears as white spots, later becoming brown.

Attrition (wearing of biting surfaces) can result from chewing abrasive foods or tobacco or from the wear that accompanies aging, but it usually indicates bruxism. Another common cause is abrasion of a porcelain crown occluding against opposing enamel, because porcelain is considerably harder than enamel. Attrition makes chewing less effective and causes noncarious teeth to become

[**Table 56-3.** Some Disorders of the Oral Region by Predominant Site of Involvement]

painful when the eroding enamel exposes the underlying dentin. Dentin is sensitive to touch and to temperature changes. A dentist can desensitize such teeth or restore the dental anatomy by placing crowns or onlays over badly worn teeth. In minor cases of root sensitivity, the exposed root may be desensitized by fluoride application or dentin-bonding agents.

Deformed teeth may indicate a developmental or endocrine disorder. In Down syndrome, teeth are small. In congenital syphilis, the incisors may be small at the incisal third, causing a pegged or screwdriver shape with a notch in the center of the incisal edge (Hutchinson's incisors), and the 1st molar is small, with a small occlusal surface and roughened, lobulated, often hypoplastic enamel (mulberry molar). In ectodermal dysplasia, teeth are absent or conical, so that dentures are needed from childhood. Dentinogenesis imperfecta, an autosomal dominant disorder, causes abnormal dentin that is dull bluish brown and opalescent and does not cushion the overlying enamel adequately. Such teeth cannot withstand occlusal stresses and rapidly become worn. People with pituitary dwarfism or with congenital hypoparathyroidism have small dental roots; people with gigantism have large ones. Acromegaly causes excess cementum in the roots as well as enlargement of the jaws, so teeth may become widely spaced. Acromegaly also can cause an open bite to develop in adulthood. Congenitally narrow lateral incisors occur in the absence of systemic disease. The most commonly congenitally absent teeth are the 3rd molars, followed in frequency by the maxillary lateral incisors and 2nd mandibular premolars.

Defects in tooth color must be differentiated from the darkening or yellowing that is caused by food pigments, aging, and, most prominently, smoking. A tooth may appear gray because of pulpal necrosis, usually due to extensive caries penetrating the pulp or because of hemosiderin deposited in the dentin after trauma, with or without pulpal necrosis. Children's teeth darken appreciably and permanently after even short-term use of tetracyclines by the mother during the 2nd half of pregnancy or by the child during odontogenesis (tooth development), specifically calcification of the crowns, which lasts until age 9. Tetracyclines rarely cause permanent discoloration of fully formed teeth in adults. However, minocycline darkens bone, which can be seen in the mouth when the overlying gingiva and mucosa are thin. Affected teeth fluoresce with distinctive colors under ultraviolet light corresponding to the specific tetracycline taken. In congenital porphyria, both the deciduous and permanent teeth may have red or brownish discoloration but always fluoresce red from the pigment deposited in the dentin. Congenital hyperbilirubinemia causes a yellowish tooth discoloration. Teeth can be whitened (see Table 56-4).

Defects in tooth enamel may be caused by rickets, which results in a rough, irregular band in the enamel. Any prolonged febrile illness during odontogenesis can cause a permanent narrow zone of chalky, pitted enamel or simply white discoloration visible after the tooth erupts. Thus, the age at which the disease occurred and its duration can be estimated from the location and height of the band. Amelogenesis imperfecta, an autosomal dominant disease, causes severe enamel hypoplasia. Chronic vomiting and esophageal reflux can decalcify the dental crowns, primarily the lingual surfaces of the maxillary anterior teeth. Chronic snorting of cocaine can result in widespread decalcification of teeth, because the drug dissociates in saliva into a base and HCl. Chronic use of methamphetamines markedly increases dental caries ("meth mouth").

Swimmers who spend a lot of time in over-chlorinated pools may lose enamel from the outer facial/buccal side of the teeth, especially the maxillary incisors, canines, and 1st premolars. If Na carbonate has been added to the pool water to correct pH, then brown calculus develops but can be removed by a dental cleaning.

Fluorosis is mottled enamel that may develop in children who drink water containing > 1 ppm of fluoride during tooth development. Fluorosis depends on the amount of fluoride ingested. Enamel changes range from irregular whitish opaque areas to severe brown discoloration of the entire crown with a roughened surface. Such teeth are highly resistant to dental caries.

The lips are palpated. With the patient's mouth open, the buccal mucosa and vestibules are examined with a tongue blade; then the hard and soft palates, uvula, and oropharynx are viewed. The patient is asked to extend the tongue as far as possible, exposing the dorsum, and to move the extended tongue as far as possible to each side, so that its posterolateral surfaces can be seen. If a patient does not extend the tongue far enough to expose the circumvallate papillae, the examiner grasps the tip of the tongue with a gauze pad and extends it. Then the tongue is raised to view the ventral surface and the floor of the mouth. The teeth and gingiva are viewed. An abnormal distribution of keratinized or nonkeratinized oral mucosa demands attention. Keratinized tissue that occurs in normally nonkeratinized areas appears white. This abnormal condition, called leukoplakia, requires a biopsy because it may be cancerous or precancerous. More ominous, however, are thinned areas of mucosa. These

[Table 56-4. Tooth Whitening Procedures]

red areas, called erythroplakia, if present for at least 2 wk, especially on the ventral tongue and floor of the mouth, suggest dysplasia, carcinoma in situ, or cancer.

With gloved hands, the examiner palpates the vestibules and the floor of the mouth, including the sublingual and submandibular glands. To make palpation more comfortable, the examiner asks the patient to relax the mouth, keeping it open just wide enough to allow access.

The temporomandibular joint (TMJ) is assessed by looking for jaw deviation on opening and by palpating the head of the condyle anterior to the external auditory meatus. Examiners then place their little fingers into the external ear canals with the pads of the fingertips lightly pushing anteriorly while patients open widely and close 3 times. Patients also should be able to comfortably open wide enough to fit 3 of their fingers vertically between the incisors (typically 4 to 5 cm). Trismus, the inability to open the mouth, may indicate temporomandibular disease (the most common cause), pericoronitis, scleroderma, arthritis, ankylosis of the TMJ, dislocation of the temporomandibular disk, tetanus, or peritonsillar abscess. Unusually wide opening suggests subluxation or type III Ehlers-Danlos syndrome.

Testing: For a new patient or for someone who requires extensive care, the dentist takes a full mouth x-ray series. This series consists of 14 to 16 periapical films to show the roots and bone plus 4 bite-wing films to detect early caries between posterior teeth. Modern techniques reduce radiation exposure to a near-negligible level. Patients at high risk of caries (ie, those who have had caries detected during the clinical examination, have many restorations, or have recurrent caries on teeth previously restored) should undergo bite-wing x-rays every 12 mo. Otherwise, bite-wings are indicated every 2 to 3 yr. A panoramic x-ray can yield useful information about tooth development, cysts or tumors of the jaws, supernumerary or congenitally absent teeth, 3rd molar impaction, Eagle's syndrome (less frequently), and carotid plaques.

Geriatrics Essentials

With aging, resting salivary secretion diminishes and can be further diminished by drugs, although meal-stimulated salivary flow is usually adequate. The flattened cusps of worn teeth and weakness of the masticatory muscles may make chewing tiresome, impairing food intake. Loss of bone mass in the jaws (particularly the alveolar portion), dryness of the mouth, thinning of the oral mucosa, and impaired coordination of lip, cheek, and tongue movements may make denture retention difficult. The taste buds become less sensitive, so the elderly may add abundant seasonings, particularly salt (which is harmful for some), or they may desire very hot foods for more taste, sometimes burning the often atrophic oral mucosa. Gingival recession and xerostomia contribute to development of root caries. Despite these changes, improved dental hygiene has greatly decreased the prevalence of tooth loss, and most older people can expect to retain their teeth.

Poor oral health contributes to poor nutritional intake, which impairs general health. Dental disease (particularly periodontitis) is associated with a 2-fold increased risk of coronary artery disease. Edentulous patients cannot have periodontitis (because they do not have a periodontium), although periodontitis may have resulted in their tooth loss. Aspiration pneumonia in patients with periodontitis can involve anaerobic organisms and has a high mortality rate. Severe bacteremias secondary to acute or chronic dental infection may contribute to brain abscesses, cavernous sinus thrombosis, endocarditis, prosthetic joint infections, and unexplained fevers.

Dental Care of Patients With Systemic Disorders

Certain medical conditions (and their treatment) predispose patients to dental problems or affect dental care.

Hematologic disorders: People who have disorders that interfere with coagulation (eg, hemophilia, acute leukemia, thrombocytopenia) require medical consultation before undergoing dental procedures that might cause bleeding (eg, extraction, mandibular block). Hemophiliacs should have clotting factors

given before, during, and after an extraction. Such oral surgery should be done in the hospital in consultation with a hematologist. All patients with bleeding disorders should maintain a lifelong routine of regular dental visits, which includes cleanings, fillings, topical fluoride, and preventative sealants, to avoid the need for extractions.

Cardiovascular disorders: After an MI, dental procedures should be avoided for 6 mo, if possible, to allow damaged myocardium to become less electrically labile. Patients with pulmonary or cardiac disease who require inhalation anesthesia for dental procedures should be hospitalized.

Endocarditis prophylaxis is required before dental procedures only in patients with

• Prosthetic cardiac valves

• Previous history of bacterial endocarditis

• Cyanotic congenital defects of the heart or great vessels (if unrepaired, if completely repaired during first 6 mo after surgery, or if repaired but with residual defects)

• Cardiac transplantation recipients with a valvulopathy

The heart is better protected against low-grade bacteremias, which occur in chronic dental conditions, when dental treatment is received (with prophylaxis) than when it is not received. Patients who are to undergo cardiac valve surgery or repair of congenital heart defects should have any necessary dental treatment completed before surgery.

Although probably of marginal benefit, antibiotic prophylaxis is sometimes recommended for patients with hemodialysis shunts and within 2 yr of receipt of a major prosthetic joint (hip, knee, shoulder, elbow). The organisms causing infections at these sites are almost invariably of dermal rather than oral origin.

Epinephrine and levonordefrin are added to local anesthetics to increase the duration of anesthesia. In some cardiovascular patients, excess amounts of these drugs cause arrhythmias, myocardial ischemia, or hypertension. Plain anesthetic can be used for procedures requiring < 45 min, but in longer procedures or where hemostasis is needed, up to 0.04 mg epinephrine (2 dental cartridges with 1:100,000 epinephrine) is considered safe. Generally, no healthy patient should receive > 0.2 mg epinephrine at any one appointment. Absolute contraindications to epinephrine (any dose) are uncontrolled hyperthyroidism; pheochromocytoma; BP > 200 mm Hg systolic or > 115 mm Hg diastolic; uncontrolled arrhythmias despite drug therapy; and unstable angina, MI, or stroke within 6 mo.

Some electrical dental equipment, such as an electrosurgical cautery, a pulp tester, or an ultrasonic scaler, can interfere with early-generation pacemakers.

Cancer: Extracting a tooth adjacent to a carcinoma of the gingiva, palate, or antrum facilitates invasion of the alveolus (tooth socket) by the tumor. Therefore, a tooth should be extracted only during the course of definitive treatment. In patients with leukemia or agranulocytosis, infection may follow an extraction despite the use of antibiotics.

Immunosuppression: People with impaired immunity are prone to severe mucosal and periodontal infections by fungi, herpes and other viruses, and, less commonly, bacteria. The infections may cause hemorrhage, delayed healing, or sepsis. Dysplastic or neoplastic oral lesions may develop after a few years of immunosuppression. People with AIDS may develop Kaposi's sarcoma, non-Hodgkin lymphoma, hairy leukoplakia, candidiasis, aphthous ulcers, or a rapidly progressive form of periodontal disease.

Endocrine disorders: Dental treatment may be complicated by some endocrine disorders. For example, people with hyperthyroidism may develop tachycardia and excessive anxiety as well as thyroid storm if given epinephrine. Insulin requirements may be reduced on elimination of oral infection in diabetics; insulin dose may require reduction when food intake is limited because of pain after oral surgery. In people with diabetes, hyperglycemia with resultant polyuria may lead to dehydration, resulting in decreased salivary flow (xerostomia), which, along with elevated salivary glucose levels, contributes to

caries.

Patients receiving corticosteroids and those with adrenocortical insufficiency may require supplemental corticosteroids during major dental procedures. Patients with Cushing's syndrome or who are taking corticosteroids may have alveolar bone loss, delayed wound healing, and increased capillary fragility.

Neurologic disorders: Patients with seizures who require dental appliances should have nonremovable appliances that cannot be swallowed or aspirated. Patients unable to brush or floss effectively may use chlorhexidine 12% rinses in the morning and at bedtime.

Obstructive sleep apnea: Patients with obstructive sleep apnea who are unable to tolerate treatment with a positive airway pressure (CPAP, biPAP) mask are sometimes treated with an intraoral device that expands the oropharynx. This treatment is not as effective as CPAP, but more patients tolerate using it.

Drugs: Certain drugs, such as corticosteroids, immunosuppressants, and antineoplastics, compromise healing and host defenses. When possible, dental procedures should not be done while these drugs are being given.

Some antineoplastics (eg, doxorubicin, 5-fluorouracil, bleomycin, dactinomycin, cytosine, arabinoside, methotrexate) cause stomatitis, which is worse in patients with preexisting periodontal disease. Before such drugs are prescribed, oral prophylaxis should be completed, and patients should be instructed in proper toothbrushing and flossing.

Drugs that interfere with clotting may need to be reduced or stopped before oral surgery. Patients taking aspirin, NSAIDs, or clopidogrel should stop doing so 4 days before undergoing dental surgery and can resume taking these drugs after bleeding stops. Warfarin should be stopped 2 to 3 days before oral surgery. PT is obtained; INR of 1.5 is considered safe for surgery. For people receiving hemodialysis, dental procedures should be done the day after dialysis, when heparinization has subsided.

Phenytoin and Ca channel blockers, particularly nifedipine, contribute to gingival hyperplasia; however, this hyperplasia is minimized with excellent oral hygiene and frequent oral prophylaxes (cleanings).

Bisphosphonates, primarily when given parenterally for treatment of bone cancer, and to a much lesser degree when used orally to prevent osteoporosis, can result in osteonecrosis after an extraction (see Sidebar 39-1 on p. 363).

Radiation therapy: (CAUTION: *Extraction of teeth from irradiated tissues [particularly if the total dose was > 65 Gy, especially in the mandible] is commonly followed by osteoradionecrosis of the jaw. This is a catastrophic complication in which extraction sites break down, frequently sloughing bone and soft tissue.*) Thus, if possible, patients should have any necessary dental treatment completed before undergoing radiation therapy of the head and neck region, with time allowed for healing. Teeth that may not survive should be extracted. Necessary sealants and topical fluoride should be applied. After radiation, extraction should be avoided, if possible, by using dental restorations and root canal treatment instead.

Head and neck radiation often damages salivary glands, causing xerostomia, which promotes caries. Patients must therefore practice lifelong good oral hygiene. A fluoride gel and fluoride mouth rinse should be used daily. Rinsing with 0.12% chlorhexidine for 30 to 60 sec, if tolerated, can be done in the morning and at bedtime. Viscous lidocaine may enable a patient with sensitive oral tissues to brush and floss the teeth and eat. A dentist must be seen at 3-, 4-, or 6-mo intervals, depending on findings at the last examination. Irradiated tissue under dentures is likely to break down, so dentures should be checked and adjusted whenever discomfort is noted. Early caries may also be reversed by Ca phosphopeptides and amorphous Ca phosphate, which can be applied by a dentist or prescribed to a patient for at-home use.

Patients who undergo radiation therapy may develop oral mucosal inflammation and diminished taste as well as trismus due to fibrosis of the masticatory muscles. Trismus may be minimized by such exercises as opening and closing the mouth widely 20 times 3 or 4 times/day. Extractions of teeth in irradiated bone

should be avoided (because of possible osteoradionecrosis). Sometimes root canal therapy is done, and the tooth is ground down to the gum line. If extraction is required after radiation, 10 to 20 treatments in a hyperbaric O_2 chamber may forestall or prevent osteoradionecrosis.

Bruxism

Bruxism is clenching or grinding of teeth. Bruxism can abrade and eventually wear down dental crowns and loosen teeth. In many people, headaches, jaw pain, or both actually are the result of bruxism. The most severe and extensive grinding and clenching occurs during sleep, so the person may be oblivious to it, but family members might notice.

Treatment requires that the patient consciously try to reduce bruxism while awake. Plastic oral appliances (night guards) that prevent occlusal contact by fitting between the teeth can be used while sleeping. When symptoms are severe, a guard can be used also during the day. Usually, such devices are made by dentists. However, if the only problem is tooth wear, OTC heat-moldable devices, fitted at home, are available. Mild anxiolytics, particularly benzodiazepines, may help until a night guard is available but should not be used for extended periods.

Halitosis

(Fetor Oris; Oral Malodor)

Halitosis is a frequent or persistent unpleasant odor to the breath.

Pathophysiology

Halitosis most often results from fermentation of food particles by anaerobic gram-negative bacteria in the mouth, producing volatile sulfur compounds such as hydrogen sulfide and methyl mercaptan. Causative bacteria may be present in areas of gingival or periodontal disease, particularly when ulceration or necrosis is present. The causative organisms reside deep in periodontal pockets around teeth. In patients with healthy periodontal tissue, these bacteria may deposit on the dorsal posterior tongue.

Factors contributing to the overgrowth of causative bacteria include decreased salivary flow (eg, due to parotid disease, Sjogren's syndrome, use of anticholinergics—see p. 513), salivary stagnation, and increased salivary pH.

Certain foods or spices, after digestion, release the odor of that substance to the lungs; the exhaled odor may be unpleasant to others. For example, the odor of garlic is noted on the breath by others 2 or 3 h after consumption, long after it is gone from the mouth.

Etiology

About 85% of cases result from oral conditions. A variety of systemic and extraoral conditions account for the remainder (see
Table 56-5).

The **most common causes** overall are the following:

• Gingival or periodontal disease

• Smoking

• Ingested foods that have a volatile component

GI disorders rarely cause halitosis because the esophagus is normally collapsed. It is a fallacy that breath odor reflects the state of digestion and bowel function.

Other breath odors: Several systemic diseases produce volatile substances detectable on the breath,

although not the particularly foul, pungent odors typically considered halitosis. Diabetic ketoacidosis produces a sweet or fruity odor of acetone; liver failure produces a mousy or sometimes faintly sulfurous odor; and renal failure produces an odor of urine or ammonia.

Evaluation

History: History of present illness should ascertain duration and severity of halitosis (including whether other people have noticed or complained), adequacy of patient's oral hygiene, and the relationship of halitosis to ingestion of causative foods (see Table 56-5).

Review of systems should seek symptoms of causative disorders, including nasal discharge and face or head pain (sinusitis, nasal foreign body); productive cough and fevers (pulmonary infection); and regurgitation of undigested food when lying down or bending over (Zenker's diverticulum). Predisposing factors such as dry mouth, dry eyes, or both (Sjogren's syndrome) should be noted.

Past medical history should ask about duration and amount of use of alcohol and tobacco. Drug history should specifically ask about use of those that can cause dry mouth.

[**Table 56-5.** Some Causes of Halitosis]

Physical examination: Vital signs are reviewed, particularly for presence of fever.

The nose is examined for discharge and foreign body.

The mouth is examined for signs of gum disease, dental infection, and cancer. Signs of apparent dryness are noted (eg, whether the mucosa is dry, sticky, or moist; whether saliva is foamy, stringy, or normal in appearance).

The pharynx is examined for signs of infection and cancer.

Sniff test: A sniff test of exhaled air is conducted. In general, oral causes result in a putrefying, pungent smell, whereas systemic conditions result in a more subtle, abnormal odor. Ideally, for 48 h before the examination, the patient avoids eating garlic or onions, and for 2 h before, the patient abstains from eating, chewing, drinking, gargling, rinsing, or smoking. During the test, the patient exhales 10 cm away from the examiner's nose, first through the mouth and then with the mouth closed. A worse odor through the mouth suggests an oral etiology. A worse odor through the nose suggests a nasal or sinus etiology. Similar odor through both nose and mouth suggests a systemic or pulmonary cause. If site of origin is unclear, the posterior tongue is scraped with a plastic spoon. After 5 sec, the spoon is sniffed 5 cm from the examiner's nose.

Red flags: The following findings are of particular concern:

• Fever

• Purulent nasal discharge or sputum

• Visible or palpable oral lesions

Interpretation of findings: Because oral causes are by far the most common, any visible oral disease may be presumed to be the cause in patients with no extraoral symptoms or signs. When other disorders are involved, clinical findings often suggest a diagnosis (see Table 56-5).

In patients whose symptoms seem to be related to intake of certain substances and who have no other findings, a trial of avoidance may clarify the diagnosis.

Testing: Extensive diagnostic evaluation should not be undertaken unless the history and physical examination suggest an underlying disease (see Table 56-5). Portable sulfur monitors, gas chromatography, and chemical tests of tongue scrapings are available but best left to research protocols

or the occasional dental office that focuses on halitosis.

Treatment

Underlying diseases are treated.

If the cause is oral, the patient should see a dentist for professional cleaning and treatment of gingival disease and caries. Home treatment involves enhanced oral hygiene, including thorough flossing, toothbrushing, and brushing of the tongue with the toothbrush or a scraper. Mouthwashes are of limited benefit except to mask odor for about 20 min. Psychogenic halitosis may require psychiatric consultation.

Geriatrics Essentials

Elderly patients are more likely to take drugs that cause dry mouth, which leads to difficulties with oral hygiene and hence to halitosis, but are otherwise not more likely to have halitosis. Also, oral cancers are more common with aging and are more of a concern among elderly than younger patients.

Key Points

- Most halitosis comes from fermentation of food particles by anaerobic gram-negative bacteria in the mouth.
- Extraoral disorders may cause halitosis but are often accompanied by suggestive findings.
- It is a fallacy that breath odor reflects the state of digestion and bowel function.
- Mouthwashes provide only brief benefit.

Malocclusion

Malocclusion is abnormal contact between the maxillary and mandibular teeth.

Normally, each dental arch consists of teeth in side-by-side contact, forming a smooth curve, with the maxillary anterior teeth overlying the upper third of the mandibular anterior teeth. The buccal (outer) cusps of the maxillary posterior teeth are external to the corresponding cusps of the mandibular posterior teeth. On each side of the mouth, the anterior buccal cusp of the maxillary 1st permanent molar fits into the anterior buccal groove of the mandibular 1st molar. Because the outer parts of all maxillary teeth are normally external to the mandibular teeth, the lips and cheeks are displaced from between the teeth so that they are not bitten. The lingual (inner) surfaces of the lower teeth form a smaller arc than those of the upper teeth, confining the tongue and minimizing the likelihood of its being bitten. All the maxillary teeth should contact the corresponding mandibular teeth, so that the masticatory forces (which may be > 150 lb in the molar region and 250 lb when clenching during sleep) are widely distributed. If these forces are applied to only a few teeth, those teeth will eventually loosen.

Etiology

Malocclusion often results from jaw and tooth size discrepancies (ie, the jaw is too small or the teeth are too large for the jaw to accommodate them in proper alignment) but may be caused by a number of congenital deformities and disorders or by tooth loss. When permanent teeth are lost, adjacent teeth shift and opposing teeth extrude, causing malocclusion unless a bridge, implant, or partial denture is worn to prevent these movements. When children lose deciduous teeth prematurely, the teeth more posterior in the arch or the permanent 1st molars often drift forward, leaving insufficient space for other permanent teeth to erupt. Malocclusion after facial trauma may indicate tooth displacement or jaw fracture. In ectodermal dysplasia, malocclusion results from having too few teeth.

Evaluation

Occlusion is checked on both sides of the mouth by retracting each cheek with a tongue depressor while

telling the patient to close on the back teeth. Malocclusion sometimes is identified as early as the first dental visit. Early identification may make later treatment easier and more effective.

Treatment

Malocclusions are corrected primarily for aesthetic and psychologic reasons. However, in some cases, treatment may increase resistance to caries (in specific teeth), to anterior tooth fracture, and, possibly, to periodontal disease or stripping of the gingiva on the palate. Treatment may improve speech and mastication as well. Occlusion can be improved by aligning teeth properly, by selectively grinding teeth and restorations that contact prematurely, and by inserting crowns or onlays to build up tooth surfaces that are below the plane of occlusion.

Orthodontic appliances (braces) apply a continuous mild force to teeth to gradually remodel the surrounding alveolar bone. Extraction of one or more permanent teeth (usually a 1st premolar) may be needed to allow other teeth to be repositioned or to erupt into a stable alignment. After the teeth are properly aligned, the patient wears a plastic-and-wire retainer 24 h/day initially, then only at night for 2 to 3 yr.

When orthodontic treatment alone is insufficient, surgical correction of jaw abnormalities contributing to malocclusion (orthognathic surgery) may be indicated.

Stomatitis

Oral inflammation and ulcers, known as stomatitis, may be mild and localized or severe and widespread. They are invariably painful. Stomatitis may involve swelling and redness of the oral mucosa or discrete, painful ulcers (single or multiple). Less commonly, whitish lesions form, and, rarely, the mouth appears normal (burning mouth syndrome) despite significant symptoms. Symptoms hinder eating, sometimes leading to dehydration and malnutrition. Secondary infection occasionally occurs. Some conditions are recurrent.

Etiology

Stomatitis may be caused by local infection, systemic disease, a physical or chemical irritant, or an allergic reaction (see
Table 56-6); many cases are idiopathic. Because the normal flow of saliva protects the mucosa against many insults, xerostomia predisposes the mouth to stomatitis of any cause.

The **most common specific causes** overall include

• Recurrent aphthous stomatitis (RAS)—also called recurrent aphthous ulcers (RAU)

• Viral infections, particularly herpes simplex and herpes zoster

• Other infectious agents (*Candida albicans* and bacteria)

• Trauma

• Tobacco

• Chemotherapy and radiation therapy

Evaluation

History: History of present illness should ascertain the duration of symptoms and whether the patient ever had them previously. Presence and severity of pain should be noted. The relation of symptoms to food, drugs, and other substances (particularly occupational exposure to chemicals, metals, fumes, or dust) is sought.

Review of systems seeks symptoms of possible causes, including chronic diarrhea and weakness (inflammatory bowel disease, celiac sprue); genital lesions (Behcet's syndrome, syphilis); eye irritation (Behcet's syndrome); and weight loss, malaise, and fever (nonspecific chronic illness).

Past medical history should ascertain known conditions that cause oral lesions, including herpes simplex, Behcet's syndrome, inflammatory bowel disease, and risk factors for oral lesions, including immunocompromised state (eg, cancer, diabetes, organ transplant, use of immunosuppressants, HIV infection). Whether chemotherapy or radiation therapy has ever been used to manage cancer needs to be determined. Drug history should note all recent drugs used. History of tobacco use should be noted. Social history should include sexual contact, particularly oral sex, unprotected sex, and sex with multiple partners.

Physical examination: Vital signs are reviewed for fever. The patient's general appearance is noted for lethargy, discomfort, or other signs of significant systemic illness.

The mouth is inspected for the location and nature of any lesions.

The skin and other mucosal surfaces (including the genitals) are inspected for any lesions,

[Table 56-6. Some Causes of Stomatitis]

rash, petechiae, or desquamation. Any bullous lesions are rubbed for Nikolsky's sign (peeling of epithelium with lateral pressure).

Red flags: The following findings are of particular concern:

• Fever

• Cutaneous bullae

• Ocular inflammation

• Immunocompromise

Interpretation of findings: Occasionally, causes are obvious in the history (eg, cytotoxic chemotherapy; significant occupational exposure to chemicals, fumes, or dust). Recurrent episodes of oral lesions occur with RAS, herpes simplex, and Behcet's syndrome. History of diabetes, HIV infection or other immunocompromise, or recent antibiotic use should increase suspicion of *Candida* infection. Recent drug use (particularly sulfa drugs, other antibiotics, and antiepileptics) should increase suspicion of Stevens-Johnson syndrome (SJS).

Some causes typically have **extraoral, noncutaneous findings**, some of which suggest a cause. Recurrent GI symptoms suggest inflammatory bowel disease or celiac sprue. Ocular symptoms can occur with Behcet's syndrome and SJS. Genital lesions may occur with Behcet's syndrome and primary syphilis.

Some causes usually also have **extraoral, cutaneous findings**.

Cutaneous bullae suggest SJS, pemphigus vulgaris, or bullous pemphigoid. Prodrome of malaise, fever, conjunctivitis, and generalized macular target lesions suggests SJS. Pemphigus vulgaris starts with oral lesions, then progresses to flaccid cutaneous bullae. Bullous pemphigoid has tense bullae on normal-appearing skin. Nikolsky's sign is usually positive in SJS and pemphigus vulgaris.

Cutaneous vesicles are typical with chickenpox or herpes zoster. Unilateral lesions in a band after a dermatome suggest herpes zoster. Diffuse, scattered vesicular and pustular lesions in different stages suggest chickenpox.

Kawasaki disease usually has a macular rash, desquamation of hands and feet, and conjunctivitis; it occurs in children, usually those < 5 yr. Oral findings include erythema of the lips and oral mucosa.

Other cutaneous lesions may implicate erythema multiforme, hand-foot-and-mouth disease (from coxsackievirus), or secondary syphilis.

Some causes have **isolated oral findings**, including RAS, most viral infections, acute necrotizing ulcerative gingivitis, primary syphilis, gonorrhea, and *Candida*.

Location of oral lesions may help identify the cause. Interdental ulcers occur with primary herpes simplex or acute necrotizing ulcerative gingivitis. Lesions on keratinized surfaces suggest herpes simplex, RAS, or physical injury. Physical injury typically has an irregular appearance and occurs near projections of teeth, dental appliances, or where biting can injure the mucosa. An aspirin burn next to a tooth and pizza burn on the palate are common.

Primary herpes simplex infection causes multiple vesicular lesions on the intraoral mucosa on both keratinized and nonkeratinized surfaces and always includes the gingiva. These lesions rapidly ulcerate. Clinical manifestation occurs most often in children. Subsequent reactivations (secondary herpes simplex, cold sore) usually appear starting in puberty on the lip at the vermilion border and, rarely, on the hard palate.

Acute necrotizing ulcerative gingivitis causes severe inflammation and punched-out ulcers on the dental papillae and marginal gingivae. A severe variant called noma (gangrenous stomatitis) can cause full-thickness tissue destruction (sometimes involving the lips or cheek), typically in a debilitated patient. It begins as a gingival, buccal, or palatal (midline lethal granuloma) ulcer that becomes necrotic and spreads rapidly. Tissue sloughing may occur.

Isolated oral gonorrhea very rarely causes burning ulcers and erythema of the gingiva and tongue, as well as the more common pharyngitis. Primary syphilis chancres may appear in the mouth. Tertiary syphilis may cause oral gummas or a generalized glossitis and mucosal atrophy. The site of a gumma is the only time that squamous cell carcinoma develops on the dorsum of the tongue. A common sign of HIV becoming AIDS is hairy leukoplakia (vertical white lines on the lateral border of the tongue).

C. albicans and related species, which are normal oral flora, can overgrow in people who have taken antibiotics or corticosteroids or who are immunocompromised, such as patients with AIDS. *C. albicans* can cause whitish, cheesy plaques that leave erosions when wiped off. Sometimes only flat, erythematous areas appear (erosive form of *Candida*).

Testing: Patients with acute stomatitis and no symptoms, signs, or risk factors for systemic illness probably require no testing.

If stomatitis is recurrent, viral and bacterial cultures, CBC, serum iron, ferritin, vitamin B_{12}, folate, zinc, and endomysial antibody (for sprue) are done. Biopsy at the periphery of normal and abnormal tissue can be done for persistent lesions that do not have an obvious etiology.

Systematically eliminating foods from the diet can be useful, as can changing brands of toothpaste, chewing gum, or mouthwash.

Treatment

Specific disorders are treated, and any causative substances or drugs are avoided.

Meticulous oral hygiene (using a soft toothbrush) may help prevent secondary infection. A soft diet that does not include acidic or salty foods is followed.

Topical measures: Numerous topical treatments, alone or in combination, are used to ease symptoms. These treatments include

• Anesthetics

• Protective coatings

• Corticosteroids

• Antibiotics

• Physical measures (eg, cautery)

For topical anesthesia of discomfort that may interfere with eating and drinking, the following may be effective:

• Lidocaine rinse

• Sucralfate plus aluminum-magnesium antacid rinse

A 2-min rinse is done with 15 mL (1 tbsp) 2% viscous lidocaine q 3 h prn; patient expectorates when done (no rinsing with water and no swallowing unless the pharynx is involved). A soothing coating may be prepared with sucralfate (1-g pill dissolved in 15 mL water) plus 30 mL of aluminum-magnesium liquid antacid; the patient should rinse with or without swallowing. Many institutions and pharmacies have their own variation of this formulation (magic mouthwash), which sometimes also contains an antihistamine.

If the physician is certain the inflammation is not caused by an infectious organism, the patient can

• Rinse and expectorate after meals with dexamethasone elixir 0.5 mg/5 mL (1 tsp)

• Apply a paste of 0.1% triamcinolone in an oral emollient

• Wipe amlexanox over the ulcerated area with the tip of a finger

Chemical or physical cautery can ease pain of localized lesions. Silver nitrate sticks are not as effective as low-power (2- to 3-watt), defocused, pulsed-mode CO_2 laser treatments, after which pain relief is immediate and lesions tend not to recur locally.

Key Points

• Isolated stomatitis in patients with no other symptoms and signs or risk factors for systemic illness is usually caused by a viral infection or RAS.

• Extraoral symptoms, skin rash, or both suggest more immediate need for diagnosis.

Recurrent Aphthous Stomatitis

Recurrent aphthous stomatitis is a common condition in which round or ovoid painful ulcers recur on the oral mucosa. Etiology is unclear. Diagnosis is clinical. Treatment is symptomatic and usually includes topical corticosteroids.

Recurrent aphthous stomatitis (RAS) affects 20 to 30% of adults and a greater percentage of children at some time in their life.

Etiology

Etiology is unclear, but RAS tends to run in families. The damage is predominately cell-mediated. Cytokines, such as IL-2, IL-10, and, particularly, tumor necrosis factor-α, play a role.

Predisposing factors include

• Oral trauma

- Stress
- Foods, particularly chocolate, coffee, peanuts, eggs, cereals, almonds, strawberries, cheese, and tomatoes

Allergy does not seem to be involved.

Factors that may, for unknown reasons, be *protective* include oral contraceptives, pregnancy, and tobacco, including smokeless tobacco and nicotine-containing tablets.

Symptoms and Signs

Symptoms and signs usually begin in childhood (80% of patients are < 30 yr) and decrease in frequency and severity with aging. Symptoms may involve as few as one ulcer 2 to 4 times/yr or almost continuous disease, with new ulcers forming as old ones heal. A prodrome of pain or burning for 1 to 2 days precedes ulcers, but there are no antecedent vesicles or bullae. Severe pain, disproportionate to the size of the lesion, can last from 4 to 7 days.

Ulcers are well-demarcated, shallow, ovoid, or round and have a necrotic center with a yellow-gray pseudomembrane, a red halo, and slightly raised red margins.

Minor aphthae (Mikulicz's disease) account for 85% of cases. They occur on the floor of the mouth, lateral and ventral tongue, buccal mucosa, and pharynx; are < 8 mm (typically 2 to 3 mm); and heal in 10 days without scarring.

Major aphthae (Sutton's disease, periadenitis mucosa necrotica recurrens) constitute 10% of cases. Appearing after puberty, the prodrome is more intense and the ulcers are deeper, larger (> 1 cm), and longer lasting (weeks to months) than minor aphthae. They appear in the lips, soft palate, and throat. Fever, dysphagia, malaise, and scarring may occur.

Herpetiform ulcers (morphologically resembling but unrelated to herpesvirus) account for 5% of cases. They begin as multiple (up to 100) 1- to 3-mm crops of small, painful clusters of ulcers on an erythematous base. They coalesce to form larger ulcers that last 2 wk. They tend to occur in women and at a later age of onset than do other forms of RAS.

Diagnosis

- Clinical evaluation

Evaluation proceeds as described previously under stomatitis (see p. 509). Diagnosis is based on appearance and on exclusion, because there are no definitive histologic features or laboratory tests.

Primary oral herpes simplex may mimic RAS but usually occurs in younger children, always involves the gingiva and may affect any keratinized mucosa (hard palate, attached gingiva, dorsum of tongue), and is associated with systemic symptoms. Viral culture can be done to identify herpes simplex. Recurrent herpetic lesions are usually unilateral.

Similar recurrent episodes can occur with Behcet's syndrome, inflammatory bowel disease, sprue, HIV infection, and nutritional deficiencies; these conditions generally have systemic symptoms and signs. Isolated recurrent oral ulcers can occur with herpes infection, HIV, and, rarely, nutritional deficiency. Viral testing and serum hematologic tests can identify these conditions.

Drug reactions may mimic RAS but are usually temporally related to ingestion. However, reactions to foods or dental products may be difficult to identify; sequential elimination may be necessary.

Treatment

- Topical chlorhexidine and corticosteroids

General treatments for stomatitis (see p. 512) may help patients with RAS. Chlorhexidine gluconate mouthwashes and topical corticosteroids, the mainstays of therapy, should be used during the prodrome, if possible. The corticosteroid can be dexamethasone 0.5 mg/5 mL tid used as a rinse and then expectorated or clobetasol ointment 0.05% or fluocinonide ointment 0.05% in carboxymethylcellulose mucosal protective paste (1:1) applied tid. Patients using these corticosteroids should be monitored for candidiasis. If topical corticosteroids are ineffective, prednisone (eg, 40 mg po once/day) may be needed for ≤ 5 days. Continuous or particularly severe RAS is best treated by a specialist in oral medicine. Treatment may require prolonged use of systemic corticosteroids, azathioprine or other immunosuppressants, pentoxifylline, or thalidomide. Intralesional injections can be done with betamethasone, dexamethasone, or triamcinolone. Supplemental B_1, B_2, B_6, B_{12}, folate, or iron lessens RAS in some patients.

Xerostomia

Xerostomia is dry mouth caused by reduced or absent flow of saliva. This condition can result in discomfort, interfere with speech and swallowing, make wearing dentures difficult, cause halitosis, and impair oral hygiene by causing a decrease in oral pH and an increase in bacterial growth. Longstanding xerostomia can result in severe tooth decay and oral candidiasis. Xerostomia is a common complaint among older adults, affecting about 20% of the elderly.

Pathophysiology

Stimulation of the oral mucosa signals the salivatory nuclei in the medulla, triggering an efferent response. The efferent nerve impulses release acetylcholine at salivary gland nerve terminals, activating muscarinic receptors (M_3), which increase saliva production and flow. Medullary signals responsible for salivation may also be modulated by cortical inputs from other stimuli (eg, taste, smell, anxiety).

Etiology

Xerostomia is usually caused by the following:

• Drugs

• Radiation to the head and neck (for cancer treatment)

Systemic disorders are less commonly the cause, but xerostomia is common in Sjogren's syndrome and may occur in HIV/AIDS, uncontrolled diabetes, and certain other disorders.

Drugs: Drugs are the most common cause (see Table 56-7); about 400 prescription drugs

[**Table 56-7.** Some Causes of Xerostomia]

and many OTC drugs cause decreased salivation. The most common include the following:

• Anticholinergics

• Antiparkinsonians

• Antineoplastics (chemotherapy)

Chemotherapy drugs cause severe dryness and stomatitis while they are being taken; these problems usually end after therapy is stopped.

Other common drug classes that cause xerostomia include antihypertensives, anxiolytics, and antidepressants (less severe with SSRIs than with tricyclics).

The rise of illicit methamphetamine use has resulted in an increasing incidence of meth mouth, which is severe tooth decay caused by methamphetamine-induced xerostomia. The damage is exacerbated by the bruxing and clenching caused by the drug. This combination causes very rapid destruction of teeth. Tobacco use usually causes a decrease of saliva.

Radiation: Incidental radiation to the salivary glands during radiation therapy for head and neck cancer often causes severe xerostomia (5200 cGy causes severe, permanent dryness, but even low doses can cause temporary drying).

Evaluation

History: History of present illness should include acuity of onset, temporal patterns (eg, constant vs intermittent, presence only on awakening), provoking factors, including situational or psychogenic factors (eg, whether xerostomia occurs only during periods of psychologic stress or certain activities), assessment of fluid status (eg, fluid intake habits, recurrent vomiting or diarrhea), and sleeping habits. Use of recreational drugs should be specifically elicited.

Review of systems should seek symptoms of causative disorders, including dry eyes, dry skin, rashes, and joint pain (Sjogren's syndrome).

Past medical history should inquire about conditions associated with xerostomia, including Sjogren's syndrome, history of radiation treatment, head and neck trauma, and a diagnosis of or risk factors for HIV infection. Drug profiles should be reviewed for potential offending drugs (see Table 56-7).

Physical examination: Physical examination is focused on the oral cavity, specifically any apparent dryness (eg, whether the mucosa is dry, sticky, or moist; whether saliva is foamy, stringy, or normal in appearance), the presence of any lesions caused by *Candida albicans*, and the condition of the teeth.

The presence and severity of xerostomia can be assessed at the bedside in several ways. For example, a tongue blade can be held against the buccal mucosa for 10 sec. If the tongue blade falls off immediately when released, salivary flow is normal. The more difficulty encountered removing the tongue blade, the more severe the xerostomia. In women, the lipstick sign, where lipstick adheres to the front teeth, may be a useful indicator of xerostomia.

If there appears to be dryness, the submandibular, sublingual, and parotid glands should be palpated while observing the ductal openings for saliva flow. The openings are at the base of the tongue anteriorly for the submandibular glands and on the middle of the inside of the cheek for the parotid glands. Drying the duct openings with a gauze square before palpation aids observation. If a graduated container is available, the patient can expectorate once to empty the mouth and then expectorate all saliva into the container. Normal production is 0.3 to 0.4 mL/min. Significant xerostomia is 0.1 mL/min.

Dental caries may be sought at the margins of restorations or in unusual places (eg, at the neck or tip of the tooth).

The most common manifestation of *C. albicans* infection is areas of erythema and atrophy (eg, on the dorsum of the tongue). Less common is the better-known white, cheesy curd that bleeds when wiped off.

Red flags: The following findings are of particular concern:

• Extensive tooth decay

• Concomitant dry eyes, dry skin, rash, or joint pain

• Risk factors for HIV

Interpretation of findings: Xerostomia is diagnosed by symptoms, appearance, and absence of salivary flow when massaging the salivary glands.

No further assessment is required when xerostomia occurs after initiation of a new drug and stops after cessation of that drug or when symptoms appear within several weeks of irradiation of the head and neck. Xerostomia that occurs with abrupt onset after head and neck trauma is caused by nerve damage.

Concomitant presence of dry eyes, dry skin, rash, or joint pain, particularly in a female patient, suggests a diagnosis of Sjogren's syndrome. Severe tooth decay, out of proportion to expected findings, may be indicative of illicit drug use, particularly methamphetamines. Xerostomia that occurs only during nighttime or that is noted only on awakening may be indicative of excessive mouth breathing in a dry environment.

Testing: For those in whom the presence of xerostomia is unclear, sialometry can be conducted by placing collection devices over the major duct orifices and then stimulating salivary production with citric acid or by chewing paraffin. Normal parotid flow is 0.4 to 1.5 mL/min/gland. Flow monitoring can also help determine response to therapy.

The cause of xerostomia is often apparent, but if the etiology is unclear and systemic disease is considered possible, further assessment should be pursued with biopsy of a minor salivary gland (for detection of Sjogren's syndrome, sarcoidosis, amyloidosis, TB, or cancer) and HIV testing.

Treatment

When possible, the cause of xerostomia should be addressed and treated.

For patients with drug-related xerostomia whose therapy cannot be changed to another drug, drug schedules should be modified to achieve maximum drug effect during the day, because nighttime xerostomia is more likely to cause caries. For all drugs, easy-to-take formulations, such as liquids, should be considered, and sublingual dosage forms should be avoided. The mouth and throat should be lubricated with water before swallowing capsules and tablets or before using sublingual nitroglycerin. Patients should avoid decongestants and antihistamines.

Patients using continuous positive airway pressure for obstructive sleep apnea may benefit from humidifying the source air (room humidifier for those using oral appliance therapy).

Symptom control: Symptomatic treatment consists of measures that do the following:

• Increase existing saliva

• Replace lost secretions

• Control caries

Drugs that augment saliva production include cevimeline and pilocarpine, both cholinergic agonists. Cevimeline (30 mg po tid) has less M_2 (cardiac) receptor activity than pilocarpine and a longer half-life. The main adverse effect is nausea. Pilocarpine (5 mg po tid) may be given after ophthalmologic and cardiorespiratory contraindications are excluded; adverse effects include sweating, flushing, and polyuria.

Sipping sugarless fluids frequently, chewing xylitol-containing gum, and using an OTC saliva substitute containing carboxymethylcellulose or hydroxyethylcellulose may help. Petroleum jelly can be applied to the lips and under dentures to relieve drying, cracking, soreness, and mucosal trauma. A cold-air humidifier may aid mouth breathers who typically have their worst symptoms at night.

Meticulous oral hygiene is essential. Patients should brush and floss regularly and use fluoride rinses or gels daily; using newer toothpastes with added Ca and phosphorous also may help avoid rampant caries. An increased frequency of preventive dental visits with plaque removal is advised. The most effective way to prevent caries is to sleep with individually fitted carriers containing 1.1% Na fluoride or 0.4% stannous fluoride. If 2 carriers cannot be worn at once, then each arch should be covered every other night. In addition, a dentist can apply a 5% Na fluoride varnish 2 to 4 times/yr.

Patients should avoid sugary or acidic foods and beverages and any irritating foods that are dry, spicy,

astringent, or excessively hot or cold.

Geriatrics Essentials

Although dry mouth becomes more common among the elderly, this is probably due to the many drugs typically used by the elderly rather than aging itself.

Key Points

- Drugs are the most common cause, but systemic diseases (most commonly Sjogren's syndrome or HIV) and radiation therapy also can cause xerostomia.
- Symptomatic treatment includes increasing existing saliva flow with stimulants or drugs, and artificial saliva replacement. Xylitol-containing gum and candy may be useful.
- Patients with xerostomia are at high risk of tooth decay; meticulous oral hygiene and professionally applied fluorides are essential.

Chapter 57. Common Dental Disorders

Introduction

Common dental disorders include caries, gingivitis, periodontitis, and pulpitis. Dental emergencies, such as toothache, fractured or avulsed teeth, and postextraction complications, are discussed in Ch. 58.

Caries

Caries is tooth decay, commonly called cavities. The symptoms—tender, painful teeth—appear late. Diagnosis is based on inspection, probing of the enamel surface with a fine metal instrument, and dental x-rays. Treatment involves removing affected tooth structure and restoring it with various materials. Fluoride, diligent dental hygiene, sealants, and proper diet can prevent virtually all caries.

Etiology

Caries is caused by acids produced by bacteria in dental plaque. Plaque is, at first, a soft, thin film of bacteria, mucin, dead epithelial cells, and food debris that develops on the tooth surface within about 24 h after the tooth is cleaned. *Mutans streptococci* is a group of related bacteria that grow in plaque and can cause caries. Some strains are more cariogenic than others. Eventually (commonly, after 72 h), soft plaque mineralizes, mainly with Ca, phosphate, and other minerals, becoming calculus (hard plaque or tartar), which cannot easily be removed with a toothbrush.

Risk factors: There are several risk factors for caries:

• Dental defects

• High-acid or low-fluoride environment

• Reduced salivary flow

Many teeth have open enamel pits, fissures, and grooves, which may extend from the surface to the dentin. These defects may be wide enough to harbor bacteria but too narrow to clean effectively. They predispose teeth to caries. Large amounts of sugar in the diet provide nutrients for plaque-forming bacteria.

A tooth surface is more susceptible to caries when it is poorly calcified, has low fluoride exposure, or is in an acidic environment. Typically, decalcification begins when the pH at the tooth falls below 5.5 (eg, when lactic acid-producing bacteria colonize the area or when people drink cola beverages, which contain phosphoric acid).

Rampant caries in deciduous teeth suggests prolonged contact with infant formula, milk, or juice, typically when an infant goes to bed with a bottle (baby or nursing bottle caries). Thus, bedtime bottles should contain only water.

The elderly often take drugs that reduce salivary flow, predisposing to caries. The elderly also have a higher incidence of root caries because of gingival recession, exposure of root surfaces, and declining manual dexterity.

Complications: Untreated caries leads to tooth destruction, infections, and the need for extractions and replacement prostheses. Premature loss of deciduous teeth may shift the adjacent teeth, hindering eruption of their permanent successors.

Symptoms and Signs

Caries initially involves only the enamel and causes no symptoms. A cavity that invades the dentin causes pain, first when hot, cold, or sweet foods or beverages contact the involved tooth, and later with chewing

or percussion. Pain can be intense and persistent when the pulp is severely involved (see Pulpitis on p. 522).

Diagnosis

• Direct inspection

• Sometimes use of x-rays or special testing instruments

Routine, frequent (q 6 to 12 mo) clinical evaluation identifies early caries at a time when minimal intervention prevents its progression. A thin probe, sometimes special dyes, and transillumination by fiberoptic lights are used, frequently supplemented by new devices that detect caries by changes in electrical conductivity or laser reflectivity. However, x-rays are still important for detecting caries, determining the depth of involvement, and identifying caries under existing restorations.

Treatment

• Restorative therapy

• Sometimes a root canal and crown

Incipient caries (which is confined to the enamel) should be remineralized through improved home care (brushing and flossing), cleanings, prescriptions for high-fluoride toothpastes, and multiple fluoride applications at the dental office.

The primary treatment of caries that has entered dentin is removal by drilling, followed by filling of the resultant defect. For very deep cavities, a temporary filling may be left in place 6 to 10 wk in the hope that a tooth will deposit reparative dentin, preventing exposure of the pulp, which necessitates root canal treatment.

Fillings for occlusal surfaces of posterior teeth, which bear the brunt of mastication, must be composed of strong materials. The most common material has been silver amalgam, which combines silver, mercury, copper, tin, and, occasionally, zinc, palladium, or indium. Amalgam is inexpensive and lasts an average of 14 yr. However, if oral hygiene is good and if amalgam was placed using a rubber dam for isolation from saliva, many amalgam fillings last > 40 yr. Although concern has been raised about mercury poisoning, the number of amalgam fillings a person has bears no relationship to blood mercury levels. Replacing amalgam is not recommended because it is expensive, damages tooth structure, and actually increases patient exposure to mercury.

Composite resins, which have a more acceptable appearance, have long been used in anterior teeth, where aesthetics are primary and the forces of chewing are minimal. Some patients request them in posterior teeth as well, and they are becoming common there. However, composite resins under high occlusal stress generally last less than half as long as amalgam and tend to develop recurrent decay because the composite resin shrinks when it hardens and expands and contracts with heat and cold more than the tooth or other filling materials. The current generation of composites also closely resemble enamel but do not appear to have the same incidence of recurrent caries as earlier materials and may also last longer. However, although long-term results with these newer amalgam substitutes appear good, data equivalent in numbers and duration to those with amalgam are not yet available.

If decay leaves too little dentin to hold a restoration, a dentist replaces the missing dentin with cement, amalgam, composite, or other materials. Sometimes a post must be inserted into one or more roots to support a gold, silver, or composite core, which replaces the coronal dentin. This procedure necessitates a root canal filling, in which an opening is made in the tooth and the pulp is removed. The root canal system is thoroughly debrided, shaped, and then filled with gutta-percha. The outer tooth surfaces (what would have been the enamel) are then reduced so that an artificial crown, usually made of gold, porcelain, or both, can be placed. Crowns for anterior teeth consist of, or are covered with, porcelain.

Prevention

• Regular brushing and flossing

• Fluoride in water, toothpaste, or both

• Regular professional cleanings

• Rarely chlorhexidine rinses and topical fluoride applications

For most people, caries is preventable. Cavities first form on permanent teeth in the early teens to late 20s. Caries-prone people typically have low exposure to fluoride and a relatively cariogenic microflora acquired from their mothers and through social contact. Maintaining good oral hygiene and minimizing sugar intake are especially important.

Removal of plaque at least q 24 h, usually by brushing and flossing, helps prevent dental caries. The gingival third of the tooth is the most important area to clean but is the area most often neglected. Electric and electronic toothbrushes are excellent, but a manual soft toothbrush, used for an average of 3 to 4 min, suffices. Using excess toothpaste, particularly an abrasive type, may erode the teeth. Dental floss is placed between each of the teeth, curved against the side of each tooth, and moved up and down 3 times, going just beneath the gingival margin. Flosses that are very thin (dental tape) or coated with wax or polytetraethylene can be used for exceptionally tight contacts between teeth or rough filling margins.

Teeth with fluoride incorporated into their enamel are more resistant to acidic decalcification and more readily recalcify when pH increases. If drinking water is not adequately fluoridated, fluoride supplements are recommended for children from shortly after birth through age 8 yr and for pregnant women beginning at 3 mo gestation (when teeth are forming in the fetus). The dose must be selected according to the amount of fluoride present in the drinking water and the age of the child. The total dose should not be so high as to cause dental fluorosis (see p. 52). Fluoridated toothpaste should also be used by people of all ages.

Fluoridation offers less protection against caries in pits and fissures than against those on smooth surfaces. Pits and fissures require use of sealants (plastic materials that adhere tightly to the surface of the enamel) to prevent nutrients from reaching bacteria, reducing their growth and acid production.

If these measures do not decrease cavity formation, more intensive therapy is aimed at changing the flora. After cavities are treated, pits and fissures, which can harbor *M. streptococci*, are sealed. This treatment is followed by 60-sec mouth rinses using 0.12% chlorhexidine bid for 2 wk, which may reduce the cariogenic bacteria in plaque and allow repopulation with less cariogenic strains of *M. streptococci*. To encourage this repopulation, xylitol in the form of hard candy or chewing gum is used for 5 min tid. Additionally, topical fluoride may be applied by a dentist or used at night in a custom-made fluoride carrier.

For pregnant women with a history of severe caries, the above regimen may be used before the child's teeth erupt. If this is not feasible, the mother can use xylitol, as mentioned above, from the time of the baby's birth to the age at which the mother no longer samples the child's food (the hypothesized mode of transfer).

For prevention of caries in deciduous teeth (once they have erupted) in infants, bedtime bottles should contain only water.

Gingivitis

Gingivitis is inflammation of the gingivae, causing bleeding with swelling, redness, exudate, a change of normal contours, and, occasionally, discomfort. Diagnosis is based on inspection. Treatment involves professional teeth cleaning and intensified home dental hygiene. Advanced cases may require antibiotics or surgery.

Normally, the gingivae are firm, tightly adapted to the teeth, and contoured to a point. Keratinized gingiva near the crowns is pink stippled tissue. This tissue should fill the entire space between the crowns. The

gingiva farther from the crowns, called alveolar mucosa, is nonkeratinized, highly vascular, red, movable, and continuous with the buccal mucosa. A tongue depressor should express no blood or pus from normal gingiva.

Inflammation, or gingivitis, the most common gingival problem, may evolve into periodontitis (see p. 520).

Etiology

The most common cause of gingivitis is poor oral hygiene.

Poor oral hygiene allows plaque to accumulate between the gingiva and the teeth; gingivitis does not occur in edentulous areas. Irritation due to plaque deepens the normal crevice between the tooth and gingiva, creating gingival pockets. These pockets contain bacteria that may cause both gingivitis and root caries. Other local factors, such as malocclusion, dental calculus, food impaction, faulty dental restorations, and xerostomia, play a secondary role.

Systemic causes: Gingivitis also commonly occurs at puberty, during menstruation and pregnancy, and at menopause, presumably because of hormonal changes. Similarly, oral contraceptives may exacerbate inflammation.

Gingivitis may be an early sign of a systemic disorder, particularly those that affect the response to infection (eg, diabetes, AIDS, vitamin deficiency, leukopenia), particularly if it occurs in patients with minimal dental plaque. Some patients with Crohn's disease have a cobblestone area of granulomatous gingival hypertrophy when intestinal flare-ups occur. Exposure to heavy metals (eg, lead, bismuth) may cause gingivitis and a dark line at the gingival margin. Severe deficiency of niacin or vitamin C can cause gingivitis.

Symptoms and Signs

Simple gingivitis first causes a deepening of the sulcus (gingival crevice) between the tooth and the gingiva, followed by a band of red, inflamed gingiva along one or more teeth, with swelling of the interdental papillae and easy bleeding. Pain is usually absent. It may resolve, remain superficial for years, or occasionally progress to periodontitis.

Pericoronitis is acute, painful inflammation of the gingival flap over a partly erupted tooth, usually around mandibular 3rd molars (wisdom teeth). Infection is common, and an abscess may develop. Pericoronitis often recurs as food gets trapped beneath the flap. The gingival flap disappears when the tooth is fully erupted.

Desquamative gingivitis may occur during menopause. It is characterized by deep red, painful gingival tissue that bleeds easily. Vesicles may precede desquamation. The gingivae are soft because the keratinized cells that resist abrasion by food particles are absent. A similar gingival lesion may be associated with pemphigus vulgaris, bullous pemphigoid, benign mucous membrane pemphigoid, or atrophic lichen planus.

During pregnancy, swelling, especially of the interdental papillae, is likely to occur. Pedunculated gingival growths often arise in the interdental papillae during the 1st trimester, may persist throughout pregnancy, and may or may not subside after delivery. Pregnancy tumors are soft reddish masses that are, histologically, pyogenic granulomas. They develop rapidly and then remain static. An underlying irritant is common, such as calculus or a restoration with a rough margin.

Uncontrolled diabetes can exaggerate the effects of gingival irritants, making secondary infections and acute gingival abscesses common.

In leukemia, the gingivae may become engorged with a leukemic infiltrate, exhibiting clinical symptoms of edema, pain, and easily induced bleeding.

In scurvy (vitamin C deficiency), the gingivae are inflamed, hyperplastic, and engorged, bleeding easily.

Petechiae and ecchymoses may appear throughout the mouth.

In pellagra (niacin deficiency), the gingivae are inflamed, bleed easily, and are susceptible to secondary infection. Additionally, the lips are reddened and cracked, the mouth feels scalded, the tongue is smooth and bright red, and the tongue and mucosa may have ulcerations.

Diagnosis

• Clinical evaluation

Finding erythematous, friable tissue at the gum lines confirms the diagnosis. To detect early gingival disease, some dentists frequently measure the depth of the pocket around each tooth. Depths < 3 mm are normal; deeper pockets are at high risk of gingivitis and periodontitis.

Treatment

• Regular oral hygiene and professional cleaning

Simple gingivitis is controlled by proper oral hygiene with or without an antibacterial mouth rinse. Thorough scaling (professional cleaning with hand or ultrasonic instruments) should be done. If appropriate, poorly contoured restorations are reshaped or replaced and local irritants are removed. Excess gingiva, if present, can be excised. Drugs causing gingival hyperplasia should be stopped if possible; if not, improved home care and more frequent professional cleanings (at least every 3 mo) usually reduce the hyperplasia. Pregnancy tumors are excised.

Treatment of pericoronitis consists of

• Removal of debris from under the gingival flap

• Irrigation with saline, 1.5% hydrogen peroxide, or 0.12% chlorhexidine

• Particularly when episodes recur, extraction

If severe infection develops, antibiotics may be given for a day before extraction and continued during healing. A common regimen is amoxicillin 500 mg po q 6 h for 10 days (or until 3 days after all inflammation has subsided). Abscesses associated with pericoronitis require localized incision and drainage, a periodontal flap and root debridement, or extraction.

In gingivitis caused by systemic disorders, treatment is directed at the cause. In desquamative gingivitis during menopause, sequential administration of estrogens and progestins may be beneficial, but adverse effects of this therapy (see p. 2519) limit recommendations for its use. Otherwise, dentists may prescribe a corticosteroid rinse or a corticosteroid paste that is applied directly to the gums. Gingivitis caused by pemphigus vulgaris (see p. 658) and similar mucocutaneous conditions may require systemic corticosteroid therapy.

Prevention

Daily removal of plaque with dental floss and a toothbrush and routine cleaning by a dentist or hygienist at 6-mo to 1-yr intervals can help minimize gingivitis. Patients with systemic disorders predisposing to gingivitis require more frequent professional cleanings (from q 2 wk to 4 times/yr).

Acute Necrotizing Ulcerative Gingivitis

(Fusospirochetosis; Trench Mouth; Vincent's Infection or Angina)

Acute necrotizing ulcerative gingivitis is a painful infection of the gums. Symptoms are acute pain, bleeding, and foul breath. Diagnosis is based on clinical findings. Treatment is gentle debridement, improved oral hygiene, mouth rinses, supportive care, and, if debridement must

be delayed, antibiotics.

Acute necrotizing ulcerative gingivitis occurs most frequently in smokers and debilitated patients who are under stress. Other risk factors are poor oral hygiene, nutritional deficiencies, and sleep deprivation.

Symptoms and Signs

The usually abrupt onset may be accompanied by malaise or fever. The chief manifestations are acutely painful, bleeding gingivae; excessive salivation; and overwhelmingly foul breath (fetor oris). Ulcerations, which are pathognomonic, are present on the dental papillae and marginal gingiva; these have a characteristically punched-out appearance and are covered by a gray pseudomembrane. Similar lesions on the buccal mucosa and tonsils are rare. Swallowing and talking may be painful. Regional lymphadenopathy often is present.

Diagnosis

• Clinical evaluation

Rarely, tonsillar or pharyngeal tissues are affected, and diphtheria or infection due to agranulocytosis must be ruled out by throat culture and CBC.

Treatment

• Debridement

• Rinses (eg, hydrogen peroxide, chlorhexidine)

• Improved oral hygiene

• Sometimes oral antibiotics

Treatment consists of gentle debridement with a hand scaler or ultrasonic device. Debridement is done over several days. The patient uses a soft toothbrush to wipe the teeth. Rinses at hourly intervals with warm normal saline or twice/day with 1.5% hydrogen peroxide or 0.12% chlorhexidine may help during the first few days after initial debridement. Essential supportive measures include improved oral hygiene (done gently at first), adequate nutrition, high fluid intake, rest, analgesics as needed, and avoiding irritation (eg, caused by smoking or hot or spicy foods). Marked improvement usually occurs within 24 to 48 h, after which debridement can be completed. If debridement is delayed (eg, if a dentist or the instruments necessary for debridement are unavailable), oral antibiotics (eg, amoxicillin 500 mg, erythromycin 250 mg, or tetracycline 250 mg q 6 h) provide rapid relief and can be continued until 72 h after symptoms resolve. If the gingival contour inverts (ie, if the tips of papillae are lost) during the acute phase, surgery is eventually required to prevent subsequent periodontitis.

Other Gingival Disorders

Hyperplasia of gingival tissues may occur without inflammation in response to various drugs, particularly phenytoin, cyclosporine, and nifedipine or, less commonly, other Ca channel blockers. Hyperplasia is characterized by diffuse, relatively avascular smooth or nodular enlargement of the gingiva, which may almost cover some teeth. The hypertrophied tissue is often excised. If possible, substitutions are made for the offending drugs. Scrupulous oral hygiene may minimize recurrence.

Carcinoma can also originate in the gingiva and spread to regional lymph nodes.

Periodontitis

Periodontitis is an infection of the periodontium—causing inflammation of the periodontal ligament, gingiva, cementum, and alveolar bone. It usually manifests as a worsening of gingivitis. Symptoms are rare except with HIV or when abscesses develop, in which case pain

and swelling are common. Diagnosis is based on inspection, periodontal probing, and x-rays. Treatment involves dental cleaning that extends under the gums and a vigorous home hygiene program. Advanced cases may require antibiotics and surgery.

Etiology

Periodontitis usually develops when gingivitis, usually with abundant plaque and calculus beneath the gingival margin, has not been adequately treated. In periodontitis, the deep pockets can harbor anaerobic organisms that do more damage than those usually present in simple gingivitis. The gingiva progressively loses its attachment to the teeth, periodontal pockets deepen, and bone loss begins. With progressive bone loss, teeth may loosen, and gingiva recedes. Tooth migration is common in later stages.

Systemic causes: Systemic diseases that predispose patients to periodontitis include diabetes (especially type 1); acquired, familial, and cyclic neutropenia; leukemia; Down syndrome; leukocyte adhesion deficiency syndromes; Papillon-Lefevre syndrome; Crohn's disease; histiocytosis syndromes; agranulocytosis; lazy leukocyte syndrome; hypogammaglobulinemia; Chediak-Higashi syndrome; glycogen storage disease; infantile genetic agranulocytosis; Ehlers-Danlos syndrome (types IV and VIII); vitamin C deficiency (scurvy); and hypophosphatasia. Faulty occlusion, causing an excessive functional load on teeth, may contribute to progression of a particular type of periodontitis characterized by angular bony defects.

Pathophysiology

Periodontitis is usually chronic and characterized by periods of exacerbation and remission. Chronic periodontitis (formerly adult periodontitis) occurs in localized and generalized forms, and people with significant disease tend to be > 35 yr. About 85% of the population is affected to a mild degree, but the most advanced cases are seen in less than 5% of the population.

Aggressive periodontitis: Several more rapidly progressive subtypes of chronic periodontitis exist, collectively known as aggressive periodontitis. Aggressive periodontitis may develop as early as childhood, sometimes before age 3 yr. Patients may have severe bone loss, even tooth loss, by age 20. Neutrophil function may be defective in aggressive periodontitis; its clinical significance is unknown.

In one type of aggressive periodontitis that occurs in healthy adolescents (formerly called localized juvenile periodontitis), patients often have significant colonization of *Actinobacillus actinomycetemcomitans*. Typically, the signs of inflammation are minor. The disease is detected by periodontal probing or x-rays, which show localized, deep (vertical) bone loss, commonly limited to the 1st molars and incisors. Bone loss progresses faster than in adult periodontitis, often at a rate of 3 to 4 µm/day.

An uncommon type of aggressive periodontitis (formerly called prepubertal periodontitis) affects deciduous teeth, usually shortly after eruption. Generalized acute proliferative gingivitis and rapid alveolar bone destruction are its hallmarks. Patients also have frequent bouts of otitis media and are usually diagnosed by age 4 yr. In some patients, the disease resolves before the permanent teeth erupt. Treatment regimens are under study.

Prototypical aggressive periodontitis (formerly called rapidly progressive periodontitis) occurs in patients aged 20 to 35 yr. It is often associated with *A. actinomycetemcomitans*, *Porphyromonas gingivalis*, *Eikenella corrodens*, and many gram-negative bacilli, but cause and effect are not clear. Some cases result from undiagnosed localized juvenile periodontitis or prepubertal periodontitis, but others appear independently.

HIV-associated periodontitis is a particularly virulent, rapidly progressing disease. Clinically, it resembles acute necrotizing ulcerative gingivitis (see p. 520) combined with rapidly progressive periodontitis. Patients may lose 9 to 12 mm of attachment in as little as 6 mo.

Symptoms and Signs

Pain is usually absent unless an acute infection forms in one or more periodontal pockets or if HIV-associated periodontitis is present. Impaction of food in the pockets can cause pain at meals. Abundant plaque along with redness, swelling, and exudate are characteristic. Gums may be tender and bleed easily, and breath may be foul.

Diagnosis

• Clinical evaluation

• Sometimes dental x-rays

Inspection of the teeth and gingiva combined with probing of the pockets and measurement of their depth are usually sufficient for diagnosis. Pockets deeper than 4 mm indicate periodontitis. Dental x-rays reveal alveolar bone loss adjacent to the periodontal pockets.

Treatment

• Scaling and root planing

• Sometimes oral antibiotics, antibiotic packs, or both

• Surgery or extraction

For all forms of periodontitis, the first phase of treatment consists of thorough scaling and root planing (ie, removal of diseased or toxin-affected cementum and dentin followed by smoothing of the root) to remove plaque and calculus deposits. Thorough home oral hygiene is necessary. The patient is reevaluated after 3 wk. If pockets are no deeper than 4 mm at this point, the only treatment needed is regular cleanings.

If deeper pockets persist, systemic antibiotics can be used. A common regimen is amoxicillin 500 mg po qid for 10 days. In addition, a gel containing doxycycline or microspheres of minocycline can be placed into isolated recalcitrant pockets. These are resorbed in 2 wk.

Another approach is to surgically eliminate the pocket and recontour the bone so that the patient can clean the depth of the sulcus (pocket reduction/elimination surgery). In selected situations, regenerative surgery and bone grafting are done to encourage alveolar bone growth. Splinting of loose teeth and selective reshaping of tooth surfaces to eliminate traumatic occlusion may be necessary. Extractions are often necessary in advanced disease. Contributing systemic factors should be controlled before initiating periodontal therapy.

Ninety percent of patients with HIV-associated periodontitis respond to irrigation of the sulcus with povidone-iodine (which the dentist applies with a syringe), regular use of chlorhexidine mouth rinses, and systemic antibiotics, usually metronidazole 250 mg po tid for 14 days.

Localized juvenile periodontitis requires periodontal surgery plus oral antibiotics (eg, amoxicillin 500 mg qid or metronidazole 250 mg tid for 14 days).

Pulpitis

Pulpitis is inflammation of the dental pulp resulting from untreated caries, trauma, or multiple restorations. Its principal symptom is pain. Diagnosis is based on clinical findings and is confirmed by x-ray. Treatment involves removing decay, restoring the damaged tooth, and sometimes doing root canal therapy or extracting the tooth.

Pulpitis can occur when

• Caries progresses deeply into the dentin

• A tooth requires multiple invasive procedures

• Trauma disrupts the lymphatic and blood supply to the pulp

Pulpitis begins as a reversible condition in which the tooth can be saved by a simple filling. It becomes irreversible as swelling inside the rigid encasement of the dentin compromises circulation, making the pulp necrotic, which predisposes to infection.

Complications: Infectious sequelae of pulpitis include apical periodontitis, periapical abscess, cellulitis, and osteomyelitis of the jaw. Spread from maxillary teeth may cause purulent sinusitis, meningitis, brain abscess, orbital cellulitis, and cavernous sinus thrombosis. Spread from mandibular teeth may cause Ludwig's angina, parapharyngeal abscess, mediastinitis, pericarditis, empyema, and jugular thrombophlebitis.

Symptoms and Signs

In reversible pulpitis, pain occurs when a stimulus (usually cold or sweet) is applied to the tooth. When the stimulus is removed, the pain ceases within 1 to 2 sec.

In irreversible pulpitis, pain occurs spontaneously or lingers minutes after the stimulus is removed. A patient may have difficulty locating the tooth from which the pain originates, even confusing the maxillary and mandibular arches (but not the left and right sides of the mouth). The pain may then cease for several days because of pulpal necrosis. As infection develops and extends through the apical foramen, the tooth becomes exquisitely sensitive to pressure and percussion. A periapical (dentoalveolar) abscess elevates the tooth from its socket and feels "high" when the patient bites down.

Diagnosis

• Clinical evaluation

• Sometimes dental x-rays

Diagnosis is based on the history and physical examination, which makes use of provoking stimuli (application of heat, cold, percussion). X-rays help determine whether inflammation has extended beyond the tooth apex and help exclude other conditions.

Treatment

• Drilling and filling for reversible pulpitis

• Root canal and crown or extraction for irreversible pulpitis

• Antibiotics (eg, amoxicillin) for infection

In reversible pulpitis, pulp vitality can be maintained if the tooth is treated, usually by caries removal, and then restored.

Irreversible pulpitis and its sequelae require endodontic (root canal) therapy or tooth extraction. In endodontic therapy, an opening is made in the tooth and the pulp is removed. The root canal system is thoroughly debrided, shaped, and then filled with gutta-percha. After root canal therapy, adequate healing is manifested clinically by resolution of symptoms and radiographically by bone filling in the radiolucent area at the root apex over a period of months. If patients have systemic signs of infection (eg, fever), an oral antibiotic is prescribed (amoxicillin 500 mg q 8 h; for patients allergic to penicillin, clindamycin 150 mg or 300 mg q 6 h). If symptoms persist or worsen, root canal therapy is usually repeated in case a root canal was missed, but alternative diagnoses (eg, temporomandibular disorder, occult tooth fracture, neurologic disorder) should be considered.

Very rarely, subcutaneous or mediastinal emphysema develops after compressed air or a high-speed air turbine dental drill has been used during root canal therapy or extraction. These devices can force air into

the tissues around the tooth socket that dissects along fascial planes. Acute onset of jaw and cervical swelling with characteristic crepitus of the swollen skin on palpation is diagnostic. Treatment usually is not required, although prophylactic antibiotics are sometimes given.

Dental Appliances

Teeth may be lost to dental caries, periodontal disease, or trauma or may be removed when treatment fails. Missing teeth may cause cosmetic, phonation, and occlusal problems and may allow movement of remaining teeth.

Types: Dental appliances include fixed bridges, removable partial or complete dentures, and osseo-integrated implants.

A **bridge** (fixed partial denture) is composed of false teeth cast or soldered to each other and, at each end, to a crown that is cemented to natural (abutment) teeth, which bear all stress of biting. A bridge is not removed. A bridge is smaller than a removable partial denture, but one or multiple bridges can be made to replace many of the teeth in a dental arch.

A **removable partial denture**, typically an appliance with clasps that snap over abutment teeth, may be removed for cleaning and during sleep. Part of the occlusal stress may be borne by the soft tissues under the denture, often on both sides of the jaw. This appliance commonly is used when many teeth have to be replaced and bridges or implants are not feasible or affordable.

Complete dentures are removable appliances used when no teeth remain. They help a patient chew and improve speech and appearance but do not provide the efficiency or sensation of natural dentition. When teeth are absent, the mandible slowly resorbs, resulting in ill-fitting dentures that require revision (called reline or rebase) or replacement. Alternatives are oral surgical procedures to enlarge the alveolar ridge or dental implants to replace missing teeth.

An **implant** is typically a titanium cylinder or screw that replaces a tooth root. One or more implants are placed into the alveolar bone, where they ankylose. After 4 to 6 mo, artificial teeth are attached to the implants. Implants are not readily removable, although the prostheses they support can be. The potential for infection at these sites warrants scrupulous attention to oral hygiene.

Dental appliances and surgery: Generally, all removable dental appliances are removed before general anesthesia, throat surgery, or convulsive therapy to prevent their breakage or aspiration. They are stored in water to prevent changes in shape. However, some anesthesiologists believe that leaving appliances in place aids the passage of an airway tube, keeps the face in a more normal shape so that the anesthetic mask fits better, prevents natural teeth from injuring the opposing gingiva of a completely edentulous jaw, and does not interfere with laryngoscopy.

Denture problems: Occasionally, the mucosa beneath a denture becomes inflamed (denture sore mouth, inflammatory papillary hyperplasia). Contributing factors to this usually painless condition include candidal infections, poor denture fit, poor hygiene, excessive movement of the denture, and, most frequently, wearing a denture 24 h/day. The mucosa appears red and velvety. Candidal overgrowth may be indicated by adherent cottonlike patches or, more commonly, erosive lesions on the mucosa. The presence of *Candida* can be confirmed by the microscopic appearance of typical branching hyphae. Without *Candida,* inflammatory papillary hyperplasia is unlikely.

A new well-made denture almost always improves the situation. Other treatments consist of improving oral and denture hygiene, refitting the existing denture, removing the denture for extended periods, and using anti-fungal therapy (nystatin rinses for the mouth and overnight nystatin soaks for the denture). Soaking the denture in a commercial cleanser is sometimes helpful. Other options are applying nystatin suspension to the tissue surface of the denture and clotrimazole troches 10 mg 5 times/day. Ketoconazole 200 mg po once/day may be required. If inflammation persists, biopsy is indicated, and systemic conditions should be ruled out.

Chapter 58. Dental Emergencies

Introduction

Emergency dental treatment by a physician is sometimes required when a dentist is unavailable.

Oral analgesics effective for most dental problems include acetaminophen 650 to 1000 mg q 6 h and NSAIDs such as ibuprofen 400 to 800 mg q 6 h. For severe pain, these drugs may be combined with opioids such as codeine 60 mg; hydrocodone 5 mg, 7.5 mg, or 10 mg; or oxycodone 5 mg.

Antibiotics for dental infections include penicillin VK 500 mg po q 6 h and clindamycin 300 mg po q 8 h.

Prophylactic antibiotics: Current American Heart Association guidelines (2007) recommend far fewer people use prophylactic antibiotics for prevention of infective endocarditis (IE—see p. 2199).

Coverage for dental procedures is recommended only for patients with prosthetic cardiac valves, previous IE, specific congenital heart diseases, and for cardiac transplant recipients with heart valve problems (valvulopathy). Dental procedures requiring prophylaxis are those that require manipulation or perforation of gingival or oral mucosa or that involve the root end area of the teeth (ie, those most likely to cause bacteremia). The preferred drug is amoxicillin 2 g po 30 to 60 min before the procedure. For those who cannot tolerate penicillins, alternatives include clindamycin 600 mg or cephalexin 2 g.

Fractured and Avulsed Teeth

Tooth fracture: Fractures are divided by depth into those that

• Affect only the enamel

• Expose the dentin

• Expose the pulp

If the fracture involves only the enamel, patients notice rough or sharp edges but are asymptomatic. Dental treatment to smooth the edges and improve appearance is elective.

If dentin is exposed but not the dental pulp, patients usually exhibit sensitivity to cold air and water. Treatment is a mild analgesic and referral to a dentist. Dental treatment consists of restoration of the tooth by a composite (white filling) or, if the fracture is extensive, a dental crown, to cover the exposed dentin.

If the pulp is exposed (indicated by bleeding from the tooth) or if the tooth is mobile, dental referral is urgent. Dental treatment usually involves a root canal.

Root fractures and alveolar fractures are not visible, but the tooth (or several teeth) may be mobile. Dental referral is also urgent for stabilization by bonding an orthodontic arch wire or polyethylene line onto several adjacent teeth.

Tooth avulsion: Avulsed primary teeth are not replaced because they typically become necrotic, then infected. They may also become ankylosed and do not exfoliate, thereby interfering with the eruption of the permanent tooth.

If a secondary tooth is avulsed, the patient should replace it in its socket immediately and seek dental care to stabilize it. If this cannot be done, the tooth should be kept immersed in milk or wrapped in a moistened paper towel and brought to a dentist for replacement and stabilization. The tooth should not be scrubbed, because scrubbing may remove viable periodontal ligament fibers, which aid in reattachment. A patient with an avulsed tooth should take an antibiotic for several days. If the avulsed tooth cannot be found, it may have been aspirated, embedded in soft tissue, or swallowed. A chest x-ray may be needed to rule out aspiration, but a swallowed tooth is harmless.

A partially avulsed tooth that is repositioned and stabilized quickly usually is permanently retained. A completely avulsed tooth may be permanently retained if replaced in the socket with minimal handling within 30 min to 1 h. Both partial and complete avulsions usually ultimately require root canal therapy because the pulp tissue becomes necrotic. When replacement of the tooth is delayed, the long-term retention rate drops, and root resorption eventually occurs. Nevertheless, a patient may be able to use the tooth for several years.

Mandibular Dislocation

Spontaneous mandibular dislocation usually occurs in people with a history of such dislocations. Although a dislocated mandible is occasionally caused by trauma, the initiating episode is typically a wide opening followed by biting pressure (eg, biting into a large

[
Fig. 58-1. Mandibular reduction.]

sandwich with hard bread), a wide yawn, or a dental procedure. People prone to dislocation may have naturally loose temporomandibular joint (TMJ) ligaments.

Patients present with a wide-open mouth that they are unable to close. Pain is secondary to patients' attempts to close the mouth. If the mandibular midline deviates to one side, the dislocation is unilateral. Although rarely used, a local anesthetic (eg, 2% lidocaine 2 to 5 mL) injected into the ipsilateral joint and into the adjacent area of insertion of the lateral pterygoid muscle may allow the mandible to reduce spontaneously.

Manual reduction may be necessary (see Fig. 58-1). Premedication may be used (eg, diazepam 5 to 10 mg IV at 5 mg/min or midazolam 3 to 5 mg IV at 2 mg/min and an opioid such as meperidine 25 mg IV or fentanyl 0.5 to 1 µg/kg IV) but is usually unnecessary, especially if time will be lost preparing the IV. The longer the mandible is dislocated, the more difficult it is to reduce and the greater the likelihood that dislocation will recur.

Barton's bandage may be needed for 2 or 3 days. Most importantly, the patient must avoid opening the mouth wide for at least 6 wk. When anticipating a yawn, the patient should place a fist under the chin to prevent wide opening. Food must be cut into small pieces. If the patient suffers from chronic dislocations and more conservative treatment modalities have been exhausted, an oral and maxillofacial surgeon may be consulted. As last-resort treatments, the ligaments around the TMJ can be surgically tightened (shortened) in an attempt to stabilize the joint or the articular eminence can be reduced (eminectomy).

Postextraction Problems

Pain and swelling: Swelling is normal after oral surgery and is proportional to the degree of manipulation and trauma. If swelling does not begin to subside by the 3rd postoperative day, infection is likely and an antibiotic may be given (eg, penicillin VK 500 mg po q 6 h until 72 h after symptoms subside).

Postoperative pain varies from moderate to severe and is treated with analgesics (see p. 1623).

Alveolitis and osteomyelitis: Postextraction alveolitis (dry socket) is pain emanating from bare bone if the socket's clot lyses. Although assumed to be due to bacterial action, it is much more common among smokers and oral contraceptive users. It is peculiar to the removal of mandibular molars, usually wisdom teeth. Typically, the pain begins on the 2nd or 3rd postoperative day, is referred to the ear, and lasts from a few days to many weeks. Alveolitis is best treated with topical analgesics: a 1- to 2-in iodoform gauze strip saturated in eugenol or coated with an anesthetic ointment, such as lidocaine 2.5% or tetracaine 0.5%, is placed in the socket. The gauze is changed every 1 to 3 days until symptoms do not return after the gauze is left out for a few hours. This procedure eliminates the need for systemic analgesics.

Osteomyelitis, which in rare cases is confused with alveolitis, is differentiated by fever, local tenderness,

and swelling. If symptoms last a month, a sequestrum, which is diagnostic of osteomyelitis, should be sought by x-ray. Osteomyelitis requires long-term treatment with antibiotics effective against both gram-positive and gram-negative organisms and referral for definitive care.

Osteonecrosis of the jaw (ONJ): ONJ (see also Sidebar 39-1 on p. 363) is an oral lesion involving persistent exposure of mandibular or maxillary bone, which usually manifests with pain, loosening of teeth, and purulent discharge. ONJ may occur after dental extraction but also may develop after trauma or radiation therapy to the head and neck. Recently, an association has been discovered between IV bisphosphonate (BP) use and ONJ. However, oral BP therapy seems to pose very low risk of ONJ. Stopping oral BP therapy is unlikely to reduce this already low rate of ONJ, and maintaining good oral hygiene is a more effective preventative measure than stopping oral BP before dental procedures. Management of ONJ is challenging and typically involves limited debridement, antibiotics, and oral rinses.

Bleeding: Postextraction bleeding usually occurs in the small vessels. Any clots extending out of the socket are removed with gauze, and a 4-in gauze pad (folded) or a tea bag is placed over the socket. Then the patient is instructed to apply continuous pressure by biting for 1 h. The procedure may have to be repeated 2 or 3 times. Patients are told to wait at least 1 h before checking the site so as not to disrupt clot formation. They also are informed that a few drops of blood diluted in a mouth full of saliva appear to be more blood than is actually present. If bleeding continues, the site may be anesthetized by nerve block or local infiltration with 2% lidocaine containing 1:100,000 epinephrine. The socket is then curetted to remove the existing clot and to freshen the bone and is irrigated with normal saline. Then the area is sutured under gentle tension. Local hemostatic agents, such as oxidized cellulose, topical thrombin on a gelatin sponge, or microfibrillar collagen, may be placed in the socket before suturing.

If possible, patients taking low-dose anticoagulants (eg, aspirin, clopedigrol, warfarin) should stop therapy 3 to 4 days before surgery. Therapy can be reinstated that evening. If these measures fail, a systemic cause (eg, bleeding diathesis) is sought.

Toothache and Infection

Pain in and around the teeth is a common problem, particularly among those with poor oral hygiene. Pain may be constant, felt after stimulation (eg, heat, cold, sweet food or drink, chewing, brushing), or both.

Etiology

The most common causes of toothache (see Table 58-1) are

- Dental caries
- Pulpitis
- Trauma
- Erupting wisdom tooth (causing pericoronitis)

Toothache is usually caused by dental caries and its consequences.

Caries causes pain when the lesion extends through the enamel into dentin. Pain usually occurs after stimulation from cold, heat, sweet food or drink, or brushing; these stimuli cause fluid to move along dentinal tubules to the pulp. As long as the discomfort does not persist after the stimulus is removed, the pulp is likely healthy enough to be maintained. This is referred to as normal dentinal sensitivity, reversible pulpalgia, or reversible pulpitis.

Pulpitis is inflammation of the pulp, typically due to advancing caries, cumulative minor pulp damage from previous large restorations, a defective restoration, or trauma. It may be reversible or irreversible. Pressure necrosis frequently results from pulpitis, because the pulp is encased in a rigid compartment. Pain may be spontaneous or in response to stimulation. In both cases, pain lingers for a minute or longer.

Once the pulp becomes necrotic, pain ends briefly (hours to weeks). Subsequently, periapical inflammation (apical periodontitis) or an abscess develops. The tooth is exquisitely sensitive to percussion (tapped with a metal dental probe or tongue blade) and chewing.

Periapical abscess may follow untreated caries or pulpitis. The abscess may point intraorally and eventually drain or may become a cellulitis.

Tooth trauma can damage the pulp. The damage may manifest soon after the injury or up to decades later.

Pericoronitis is inflammation and infection of the tissue between the tooth and its overlying flap of gingiva (operculum). It usually occurs in an erupting wisdom tooth (almost always a lower one).

Complications: Rarely, sinusitis results from untreated maxillary dental infection. More commonly, pain from a sinus infection is perceived as originating in the (unaffected) teeth, mistakenly creating the impression of a dental origin.

Rarely, cavernous sinus thrombosis (see p. 624) or Ludwig's angina (submandibular space infection—see p. 470) develops; these conditions are life threatening and require immediate intervention.

[**Table 58-1.** Some Causes of Toothache]

Evaluation

History: History of present illness should identify the location and duration of the pain and whether it is constant or present only after stimulation. Specific triggering factors to review include heat, cold, sweet food or drink, chewing, and brushing. Any preceding trauma or dental work should be noted.

Review of systems should seek symptoms of complications, including face pain, swelling, or both (dental abscess, sinusitis); pain below the tongue and difficulty swallowing (submandibular space infection); pain with bending forward (sinusitis); and retro-orbital headache, fever, and vision symptoms (cavernous sinus thrombosis).

Past medical history should note previous dental problems and treatment.

Physical examination: Vital signs are reviewed for fever.

The examination focuses on the face and mouth. The face is inspected for swelling and is palpated for induration and tenderness.

The oral examination includes inspection for gum inflammation and caries and any localized swelling at the base of a tooth that may represent a pointing apical abscess. If no tooth is clearly involved, teeth in the area of pain are percussed for tenderness with a tongue depressor. Also, an ice cube can be applied briefly to each tooth, removing it immediately once pain is felt. In healthy teeth, the pain stops almost immediately. Pain lingering more than a few seconds indicates pulp damage (eg, irreversible pulpitis, necrosis). The floor of the mouth is palpated for induration and tenderness, suggesting a deep space infection.

Neurologic examination, concentrating on the cranial nerves, should be done in those with fever, headache, or facial swelling.

Red flags: Findings of particular concern are

• Headache

• Fever

• Swelling or tenderness of floor of the mouth

• Cranial nerve abnormalities

Interpretation of findings: Red flag finding of headache suggests sinusitis, particularly if multiple upper molar and premolar (back) teeth are painful. However, presence of vision symptoms or abnormalities of the pupils or of ocular motility suggests cavernous sinus thrombosis.

Fever is unusual with routine dental infection unless there is significant local extension. Bilateral tenderness of the floor of the mouth suggests Ludwig's angina.

[
Table 58-2. Characteristics of Pain in Toothache]

Difficulty opening the mouth (trismus) can occur with any lower molar infection but is common only with pericoronitis.

Isolated dental condition: Patients without red flag findings or facial swelling likely have an isolated dental condition, which, although uncomfortable, is not serious. Clinical findings, particularly the nature of the pain, help suggest a cause (see Tables 58-1 and 58-2). Because of its innervation, the pulp can perceive stimuli (eg, heat, cold, sweets) only as pain. An important distinction is whether there is continuous pain or pain only on stimulation and, if pain is only on stimulation, whether the pain lingers after the stimulus is removed.

Swelling at the base of a tooth, on the cheek, or both indicates infection, either cellulitis or abscess. A tender, fluctuant area at the base of a tooth suggests a pointing abscess.

Testing: Dental x-rays are the mainstay of testing but can be deferred to a dentist.

The rare cases in which cavernous sinus thrombosis or Ludwig's angina are suspected require imaging studies, typically CT or MRI.

Treatment

Analgesics (see p. 1623) are given pending dental evaluation and definitive treatment. A patient who is seen frequently for emergencies but who never receives definitive dental treatment despite availability may be seeking opioids.

Antibiotics directed at oral flora are given for most disorders beyond irreversible pulpitis (eg, necrotic pulp, apical periodontitis, abscess, cellulitis). The patient with pericoronitis also should receive an antibiotic. However, antibiotics can be deferred if the patient can be seen the same day by a dentist, who may be able to treat the infection by removing the source (eg, by extraction, pulpectomy, or curettage). When antibiotics are used, penicillin is preferred, with clindamycin the alternative.

An **abscess** associated with well-developed (soft) fluctuance is typically drained through an incision with a #15 scalpel blade at the most dependent point of the swelling. A rubber drain, held by a suture, is often placed.

Pericoronitis or erupting 3rd molars are treated with chlorhexidine 0.12% rinses or hypertonic saltwater soaks (1 tbsp salt mixed in a glass of hot water—no hotter than the coffee or tea a patient normally drinks). The salt water is held in the mouth on the affected side until it cools and then is expectorated and immediately replaced with another mouthful. Three or 4 glasses of salt water a day usually control inflammation and pain pending dental evaluation.

Teething pain in young children may be treated with weight-based doses of acetaminophen or ibuprofen. Topical treatments can include chewing hard crackers (eg, biscotti), applying 7.5% or 10% benzocaine gel qid (provided there is no family history of methemoglobinemia), and chewing on anything cold (eg, gel-containing teething rings).

The rare patient with cavernous sinus thrombosis or Ludwig's angina requires immediate hospitalization, removal of the infected tooth, and culture-guided parenteral antibiotics.

Geriatrics Essentials

The elderly are more prone to caries of the root surfaces, usually because of gingival recession. Periodontitis often begins in young adulthood; if untreated, tooth pain and loss are common in old age.

Key Points

- Most toothache involves dental caries or its complications (eg, pulpitis, abscess).
- Symptomatic treatment and dental referral are usually adequate.
- Antibiotics are given if signs of a necrotic pulp or more severe conditions are present.
- Very rare but serious complications include extension of dental infection to the floor of the mouth or to the cavernous sinus.
- Dental infections rarely cause sinusitis, but sinus infection may cause pain perceived as originating in the teeth.

Chapter 59. Temporomandibular Disorders

Introduction

(See also Mandibular Dislocation on p. 524, Temporal Bone Fractures on p. 3234, and Jaw Tumors on p. 489.)

The term temporomandibular disorders is an umbrella term for conditions causing dysfunction of the jaw joint or pain in the jaw and face, often in or around the temporomandibular joint (TMJ), including masticatory and other muscles of the head and neck, the fascia, or both. A person is considered to have a temporomandibular disorder only if pain or limitation of motion is severe enough to require professional care.

Temporomandibular disorders typically are multifactorial, but most are related to problems with muscles or joints. Internal derangements of the TMJ cause disturbed movement of the mandibular condyle in the glenoid fossa or against the cartilaginous articular disk (see Fig. 59-1). This disk, shaped like a donut with a closed hole or like a mature red blood cell, serves as a cushion between joint surfaces. Causes for this disturbed movement include clenching and grinding of the teeth, trauma, arthritis, and malocclusion and missing teeth. Even the trauma of persistent gum chewing can be enough to damage the joint.

Diagnosis

Disorders of the TMJ must be distinguished from the many conditions that mimic them (see Table 59-1). Pain exacerbated by finger pressure on the joint when the mouth is opened implicates the TMJ.

Patients are asked to describe the pain and designate painful areas. The cervical and occipital muscles and each of the major muscle groups involved in mastication are palpated for general tenderness and trigger points (spots

[**Fig. 59-1.** The temporomandibular joint.]

that radiate pain to another area). Patients are observed opening the mouth as wide as is comfortable. When patients open and close their mouth with the junction of the maxillary and mandibular central incisors (normally in the midline) lined up against a vertical straight edge, the mandibular midline typically deviates toward the painful side. Palpation and auscultation of the joint during opening and closing may reveal tenderness, catching, clicking, or popping. Condylar motion can best be palpated by placing the 5th fingers into the external ear canals and exerting very gentle forward pressure as patients move the jaw. The average-sized patient can open the mouth at least 40 to 45 mm (measured between upper and lower central incisors). To account for differences in patient size, a patient should be able to fit 3 fingers (index, middle, ring) in the mouth on top of each other.

Ankylosis of the Temporomandibular Joint

Ankylosis of the TMJ is immobility or fusion of the joint.

Ankylosis of the TMJ most often results from trauma or infection, but it may be congenital or a result of RA. Chronic, painless limitation of motion occurs. When ankylosis leads to arrest of condylar growth, facial asymmetry is common (see Condylar Hyperplasia on p. 532). Intra-articular (true) ankylosis must be distinguished from extra-articular (false) ankylosis, which may be caused by enlargement of the coronoid process, depressed fracture of the zygomatic arch, or scarring resulting from surgery, irradiation, or infection. In most cases of true ankylosis, x-rays of the joint show loss of normal bony architecture.

Treatment may include a condylectomy if the ankylosis is intra-articular or an ostectomy of part of the ramus if the coronoid process and zygomatic arch are also affected. Jaw-opening exercises must be done for months to years to maintain the surgical correction, but forced opening of the jaws without surgery is generally ineffective because of bony fusion.

Arthritis of the Temporomandibular Joint

Infectious arthritis, traumatic arthritis, osteoarthritis, RA, and secondary degenerative arthritis can affect the TMJ.

Infectious arthritis: Infection of the TMJ may result from direct extension of adjacent infection or hematogenous spread of blood-borne organisms (see also Acute Infectious Arthritis on p. 365). The area is inflamed, and jaw movement is limited. Local signs of infection associated with evidence of a systemic disease or with an adjacent infection suggest the diagnosis. X-ray results are negative in the early stages but may show bone destruction later. If suppurative arthritis is suspected, the joint is aspirated to confirm the diagnosis and to identify the causative organism. Diagnosis must be made rapidly to prevent permanent joint damage.

[Table 59-1. Some Conditions that Mimic Temporomandibular Disorders]

Treatment includes antibiotics, proper hydration, pain control, and motion restriction. Parenteral penicillin G is the drug of choice until a specific bacteriologic diagnosis can be made on the basis of culture and sensitivity testing. Suppurative infections are aspirated or incised. Once the infection is controlled, jaw-opening exercises help prevent scarring and limitation of motion.

Traumatic arthritis: Rarely, acute injury (eg, due to difficult tooth extraction or endotracheal intubation) may lead to arthritis of the TMJ. Pain, tenderness, and limitation of motion occur. Diagnosis is based primarily on history. X-ray results are negative except when intra-articular edema or hemorrhage widens the joint space. Treatment includes NSAIDs, application of heat, a soft diet, and restriction of jaw movement.

Osteoarthritis: The TMJ may be affected, usually in people > 50 yr. Occasionally, patients complain of stiffness, grating, or mild pain. Crepitus results from a hole worn through the disk, causing bone to grate on bone. Joint involvement is generally bilateral. X-rays or CT may show flattening and lipping of the condyle, suggestive of dysfunctional change. Treatment is symptomatic.

Rheumatoid arthritis: The TMJ is affected in > 17% of adults and children with RA, but it is usually among the last joints involved. Pain, swelling, and limited movement are the most common findings. In children, destruction of the condyle results in mandibular growth disturbance and facial deformity. Ankylosis may follow. X-rays of the TMJ are usually negative in early stages but later show bone destruction, which may result in an anterior open-bite deformity. The diagnosis is suggested by TMJ inflammation associated with polyarthritis and is confirmed by other findings typical of the disease.

Treatment is similar to that of RA in other joints. In the acute stage, NSAIDs may be given, and jaw function should be restricted. A night guard or splint is often helpful. When symptoms subside, mild jaw exercises help prevent excessive loss of motion. Surgery is necessary if ankylosis develops but should not be done until the condition is quiescent.

Secondary degenerative arthritis: This type of arthritis usually develops in people aged 20 to 40 after trauma or in people with persistent myofascial pain syndrome (see p. 533). It is characterized by limited opening of the mouth, unilateral pain during jaw movement, joint tenderness, and crepitus. When it is associated with the myofascial pain syndrome, symptoms wax and wane. Diagnosis is based on x-rays, which generally show condylar flattening, lipping, spurring, or erosion. Unilateral joint involvement helps distinguish secondary degenerative arthritis from osteoarthritis.

Treatment is conservative, as it is for myofascial pain syndrome, although arthroplasty or high condylectomy may be necessary. An occlusal splint (mouth guard) usually relieves symptoms. The splint is worn constantly, except during meals, oral hygiene, and appliance cleaning. When symptoms resolve, the length of time that the splint is worn each day is gradually reduced. Intra-articular injection of corticosteroids may relieve symptoms but may harm the joint if repeated often.

Condylar Hyperplasia

Condylar hyperplasia is a disorder of unknown etiology characterized by persistent or accelerated growth of the condyle when growth should be slowing or ended. Growth eventually stops without treatment.

Slowly progressive unilateral enlargement of the head and neck of the condyle causes crossbite malocclusion, facial asymmetry, and shifting of the midpoint of the chin to the unaffected side. The patient may appear prognathic. The lower border of the mandible is often convex on the affected side. Chondroma and osteochondroma may cause similar symptoms and signs, but they grow more rapidly and may cause even greater asymmetric condylar enlargement.

Diagnosis

• Plain x-rays

• Usually CT

On x-ray, the temporomandibular joint may appear normal, or the condyle may be proportionally enlarged and the mandibular neck elongated. CT is usually done to determine whether bone growth is generalized, which confirms the diagnosis, or localized to part of the condylar head. If growth is localized, a biopsy may be necessary to distinguish between tumor and hyperplasia.

Treatment

• During active growth, usually condylectomy

• After growth cessation, orthodontics or surgical mandibular repositioning

Treatment usually includes condylectomy during the period of active growth. If growth has stopped, orthodontics and surgical mandibular repositioning are indicated. If the height of the mandibular body is greatly increased, facial symmetry can be further improved by reducing the inferior border.

Condylar Hypoplasia

Condylar hypoplasia is facial deformity caused by a short mandibular ramus.

This condition usually results from trauma, infection, or irradiation occurring during the growth period but may be idiopathic. The deformity involves fullness of the face, deviation of the chin to the affected side, an elongated mandible, and flatness of the face on the unaffected side. (The side to which the ramus is short causes muscles to appear fuller; the muscles on the unaffected side are stretched so that side appears flatter.) Mandibular deviation causes malocclusion.

Diagnosis is based on a history of progressive facial asymmetry during the growth period, x-ray evidence of condylar deformity and antegonial notching (a depression in the inferior border of the mandible just anterior to the angle of the mandible), and, frequently, a causative history.

Treatment consists of surgical shortening of the unaffected side of the mandible or lengthening of the affected side. Presurgical orthodontic therapy helps optimize results.

Internal Joint Derangement

The most common form of internal joint derangement is anterior misalignment or displacement of the articular disk above the condyle. Symptoms are localized joint pain and popping on jaw movement. Diagnosis is based on history and physical examination. Treatment is with analgesics, jaw rest, muscle relaxation, physical therapy, and bite splinting. If these methods fail, surgery may be necessary. Early treatment greatly improves results.

The superior head of the lateral pterygoid muscle may pull the articular disk out of place when abnormal

jaw mechanics place unusual stress on the joint. Abnormal jaw mechanics can be due to congenital or acquired asymmetries or to the sequelae of trauma or arthritis. If the disk remains anterior, the derangement is said to be without reduction. Restricted jaw opening (locked jaw) and pain in the ear and around the temporomandibular joint result. If at some point in the joint's excursion the disk returns to the head of the condyle, it is said to be with reduction. Derangement with reduction occurs in about one third of the population at some point. All types of derangement can cause capsulitis (or synovitis), which is inflammation of the tissues surrounding the joint (eg, tendons, ligaments, connective tissue, synovium). Capsulitis can also occur spontaneously or result from arthritis, trauma, or infection.

Symptoms and Signs

Derangement with reduction often causes a clicking or popping sound when the mouth is opened. Pain may be present, particularly when chewing hard foods. Patients are often embarrassed because they think others can hear noise when they chew. Indeed, although the sound seems louder to the patient, others can sometimes hear it.

Derangement without reduction usually causes no sound, but maximum opening between the tips of the upper and lower incisors is reduced from the normal 40 to 45 mm to ≤ 30 mm. Pain and a change in the patients' perception of their bite generally result.

Capsulitis results in localized joint pain, tenderness, and, sometimes, restricted opening.

Diagnosis

• Clinical evaluation

Diagnosis of derangement with reduction requires observation of the jaw when the mouth is opened. When the jaw is opened > 10 mm (measured between upper and lower incisors), a click or pop is heard or a catch is felt as the disk pops back over the head of the condyle. The condyle remains on the disk during further opening. Usually, another click is heard during closing when the condyle slips over the posterior rim of the disk and the disk slips forward (reciprocal clicking).

Diagnosis of derangement without reduction requires that the patient open as wide as possible. The opening is measured, and gentle pressure is then exerted to open the mouth a little wider. Normally, the jaw opens about 45 to 50 mm; if the disk is deranged, it will open about 20 mm. Closing or protruding the jaw against resistance worsens the pain.

MRI is usually done to confirm presence of derangement or to determine why a patient is not responding to treatment.

Capsulitis is often diagnosed based on a history of injury or infection along with exquisite tenderness over the joint and by exclusion when pain remains after treatment for myofascial pain syndrome, disk derangement, arthritis, and structural asymmetries. However, capsulitis may be present with any of these conditions.

Treatment

• Analgesics as needed

• Sometimes repositioning splint or surgery

• Sometimes corticosteroid injection for capsulitis

Derangement with reduction does not require treatment if the patient can open reasonably wide (about 40 mm or the width of the index, middle, and ring fingers) without discomfort. If pain occurs, mild analgesics, such as NSAIDs (ibuprofen 400 mg po q 6 h), can be used. If onset is < 6 mo, an anterior repositioning splint may be used to position the mandible forward and on the disk. The splint is a horseshoe-shaped appliance of hard, transparent acrylic (plastic) made to fit snugly over the teeth of one arch. Its chewing

surface is designed to hold the mandible forward when the patient closes on the splint. In this position, the disk is always on the head of the condyle. The splint is gradually adjusted to allow the mandible to move posteriorly. If the disk stays with the condyle as the superior head of the external pterygoid stretches, the disk is said to be captured. The longer the disk is displaced, the more deformed it becomes and the less likely repositioning will succeed. Surgical plication of the disk may be done, with variable success.

Derangement without reduction may not require treatment other than analgesics. Splints may help if the articular disk has not been significantly deformed, but long-term use may result in irreversible changes in oral architecture. In some cases, the patient is instructed to slowly stretch the disk out of position, which allows the jaw to open normally. Various arthroscopic and open surgical procedures are available when conservative treatment fails.

Capsulitis is initially treated with NSAIDs, jaw rest, and muscle relaxation. If these treatments are unsuccessful, corticosteroids may be injected into the joint, or arthroscopic joint lavage and debridement are used.

Myofascial Pain Syndrome

Myofascial pain syndrome can occur in patients with a normal temporomandibular joint. It is caused by tension, fatigue, or spasm in the masticatory muscles (medial or internal and lateral or external pterygoids, temporalis, and masseter). Symptoms include bruxism, pain and tenderness in and around the masticatory apparatus or referred to other locations in the head and neck, and, often, abnormalities of jaw mobility. Diagnosis is based on history and physical examination. Conservative treatment, including analgesics, muscle relaxation, habit modification, and bite splinting, usually is effective.

This syndrome is the most common disorder affecting the temporomandibular region. It is more common among women and has a bimodal age distribution in the early 20s and around menopause. The muscle spasm causing the disorder usually is the result of nocturnal bruxism (clenching or grinding of the teeth). Whether bruxism is caused by irregular tooth contacts, emotional stress, or sleep disorders is controversial. Bruxism usually has a multifactorial etiology. Myofascial pain syndrome is not limited to the muscles of mastication. It can occur anywhere in the body, most commonly involving muscles in the neck and back.

Symptoms and Signs

Symptoms include pain and tenderness of the masticatory muscles and often pain and limitation of jaw excursion. Nocturnal bruxism may lead to headache that is more severe on awakening and that gradually subsides during the day. Such pain should be distinguished from temporal arteritis. Daytime symptoms, including headache, may worsen if bruxism continues throughout the day.

The jaw deviates when the mouth opens but usually not as suddenly or always at the same point of opening as it does with internal joint derangement (see p. 532). Exerting gentle pressure, the examiner can open the patient's mouth another 1 to 3 mm beyond unaided maximum opening.

Diagnosis

• Clinical evaluation

A simple test may aid the diagnosis: Tongue blades of 2 or 3 thicknesses are placed between the rear molars on each side, and the patient is asked to bite down gently. The distraction produced in the joint space may ease the symptoms. X-rays usually do not help except to rule out arthritis. If temporal arteritis is suspected, ESR is measured.

Treatment

• Mild analgesics

- Splint or mouth guard
- An anxiolytic at bedtime considered
- Physical therapy modalities considered

A plastic splint or mouth guard from the dentist can keep teeth from contacting each other and prevent the damages of bruxism. Comfortable, heat-moldable splints are available from many sporting goods stores or drugstores. Low doses of a benzodiazepine at bedtime are often effective for acute exacerbations and temporary relief of symptoms. Mild analgesics, such as NSAIDs or acetaminophen, are indicated. Cyclobenzaprine may help muscle relaxation in some people. Because the condition is chronic, opioids should not be used, except perhaps briefly for acute exacerbations. The patient must learn to stop clenching the jaw and grinding the teeth. Hard-to-chew foods and chewing gum should be avoided. Physical therapy, biofeedback to encourage relaxation, and counseling help some patients. Physical modalities include transcutaneous electric nerve stimulation and "spray and stretch," in which the jaw is stretched open after the skin over the painful area has been chilled with ice or sprayed with a skin refrigerant, such as ethyl chloride. Botulinum toxin has recently been used successfully to relieve muscle spasm in myofascial pain syndrome. Most patients, even if untreated, stop having significant symptoms within 2 to 3 yr.