13

Ear, nose, and throat disorders

Introduction

Ear, nose, and throat disorders rarely prove fatal (except for those resulting from neoplasms, epiglottitis, and neck trauma), but they may cause serious social, cosmetic, and communication problems. Untreated hearing loss or deafness can drastically impair ability to interact with society. Ear disorders also can cause impaired equilibrium. Nasal disorders can cause changes in facial features and interfere with breathing and tasting. Diseases arising in the throat may threaten airway patency and interfere with speech. In addition, these disorders can cause considerable discomfort and pain for the patient and require thorough assessment and prompt treatment.

The ear

Hearing begins when sound waves reach the tympanic membrane, which then vibrates the ossicles, incus, malleus, and stapes in the middle ear cavity. The stapes transmits these vibrations to the perilymphatic fluid in the inner ear by vibrating against the oval window. The vibrations then pass across the cochlea's fluid receptor cells in the basilar membrane, stimulating movement of the hair cells of the organ of Corti. The axons of the cochlear nerve terminate around the bases of those hair cells. Sound waves, which initiate impulses, travel over the auditory nerve (made up of the cochlear nerve and the vestibular nerve) to the temporal lobe of the brain.

The inner ear structures also maintain the body's equilibrium and balance through the fluid in the semicircular canals. This fluid is set in

motion by body movement and stimulates nerve cells that line the canals. These cells, in turn, transmit impulses to the cerebellum of the brain by way of the vestibular branch of the eighth cranial nerve (the acoustic nerve).

Although the ear can respond to sounds that vibrate at frequencies from 20 to 20,000 hertz (Hz), the range of normal speech is from 250 to 4,000 Hz, with 70% falling between 500 and 2,000 Hz. The ratio between sound intensities, the decibel (dB) is the unit for expressing the relative intensity (loudness) of sounds. A faint whisper registers 10 to 15 dB; average conversation, 50 to 60 dB; a shout, 85 to 90 dB. Hearing damage may follow exposure to sounds louder than 90 dB.

Assessment

After obtaining a thorough patient history of ear disease, inspect the auricle and surrounding tissue for deformities, lumps, and skin lesions. (See *Structures of the external ear*.) Ask the patient if he has ear pain. If you see inflammation, check for tenderness by moving the auricle and pressing on the tragus and the mastoid process. Check the ear canal for excessive cerumen, discharge, or foreign bodies.

Ask the patient if he has had episodes of vertigo or blurred vision. To test for vertigo, have the patient stand on one foot and close his eyes, or have him walk a straight line with his eyes closed. Ask him if he always falls to the same side and if the room seems to be spinning.

Audiometric testing

Audiometric testing evaluates hearing and determines the type and extent of hearing loss. The simplest but least reliable method for judging hearing acuity consists of covering one of the patient's ears, standing 18″ to 24″” (46 to 61 cm) from the uncovered ear, and whispering a short phrase or series of numbers. (Block the patient's vision to prevent lip reading.) Then ask the patient to repeat the phrase or series of numbers. To test hearing at both high and low frequencies, repeat the test in a normal speaking voice. (As an alternative, you can hold a ticking watch to the patient's ear.)

If you identify a hearing loss, further testing is necessary to determine if the loss is conductive or sensorineural. A conductive loss can result from

faulty bone conduction (inability of the eighth cranial nerve to respond to sound waves traveling through the skull) or faulty air conduction (impaired transmission of sound through ear structures to the auditory nerve and, ultimately, the temporal lobe of the brain).

Sensorineural hearing loss results from damage to the cochlear or vestibulocochlear nerve, which can result from aging and prolonged exposure to high-frequency or loud noises.

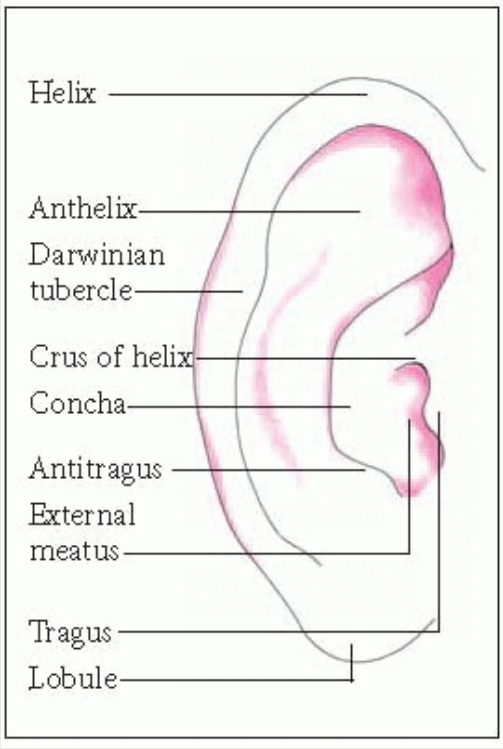
The following tests assess bone and air conduction:

- Impedance audiometry detects middle ear pathology, precisely determining the degree of tympanic membrane and middle ear mobility. One end of the impedance audiometer, a probe with three small tubes, is inserted into the external canal; the other end is attached to an oscillator. One tube delivers a low tone of variable intensity, the second contains a microphone, and the third, an air pump. A mobile tympanic membrane reflects minimal sound waves and produces a low-voltage curve on the graph. A tympanic membrane with decreased mobility reflects maximal sound waves and produces a high-voltage curve.
- Pure tone audiometry uses an audiometer to produce a series of pure tones of calibrated decibels (dB) of loudness at different frequencies (125 to 8,000 Hz). These test tones are conveyed to the patient's ears through headphones or a bone conduction (sound) vibrator. Speech threshold represents the loudness at which a person with normal hearing can perceive the tone. Both air conduction and bone conduction are measured for each ear, and the results are plotted on a graph. If hearing is normal, the line is plotted at 0 dB. In adults, normal hearing may range from 0 to 25 dB.
- In the Rinne test, the base of a lightly vibrating tuning fork is placed on the mastoid process (bone conduction). Then the fork is moved to the front of the meatus, where the patient should continue to hear the vibrations (air conduction). The patient must determine which sounds are louder. In a positive Rinne test, air conduction is greater than bone conduction, which may suggest sensorineural hearing loss. In a negative Rinne test, bone conduction is greater than air conduction, which may suggest a conductive loss.

- Speech audiometry uses the same technique as pure tone audiometry, but with speech, instead of pure tones, transmitted through the headset. (A person with normal hearing can hear and repeat 88% to 100% of transmitted words.)
- Tympanometry, using the impedance audiometer, measures tympanic membrane compliance with air pressure variations in the external canal and determines the degree of negative pressure in the middle ear.
- In Weber's test (used for testing unilateral hearing loss), the handle of a lightly vibrating tuning fork is placed on the midline of the forehead. Normally, the patient should hear sounds equally in both ears. With conductive hearing loss, sound lateralizes (localizes) to the ear with the poorest hearing. With sensorineural loss, sound lateralizes to the better functioning ear.

STRUCTURES OF THE EXTERNAL EAR

The structures of the external ear are depicted below.



The nose

As air travels between the septum and the turbinates, it touches sensory hairs (cilia) in the mucosal surface, which then add, retain, or remove moisture and particles in the air to ensure delivery of humid, bacteria-free air to the pharynx and lungs. In addition, when air touches the mucosal

cilia, the resultant stimulation of the first cranial nerve sends nerve impulses to the olfactory area of the frontal cortex, providing the sense of smell.

Assessment

Check the external nose for redness, edema, masses, or poor alignment. Marked septal cartilage depression may indicate saddle deformity due to septal destruction from trauma or congenital syphilis; extreme lateral deviation may result from injury. Red nostrils may indicate frequent nose blowing caused by allergies or infectious rhinitis. Dilated, engorged blood vessels may suggest alcoholism or constant exposure to the elements. A bulbous, discolored nose may be a sign of rosacea.

With a nasal speculum and adequate lighting, check nasal mucosa for pallor and edema or redness and inflammation, dried mucous plugs, furuncles, and polyps. Also, look for abnormal appearance of the capillaries and a deviated or perforated septum. Check for nasal discharge (assess color, consistency, and odor) and blood. Profuse, thin, watery discharge may indicate allergy or cold; excessive, thin, purulent discharge may indicate cold or chronic sinus infection.

Check for sinus inflammation by applying pressure to the nostrils, orbital rims, and cheeks. Pain after pressure applied above the upper orbital rims indicates frontal sinus irritation; pain after pressure applied to the cheeks, maxillary sinus irritation.

The throat

Parts of the throat include the pharynx, epiglottis, and larynx. The pharynx is the passageway for food to the esophagus and air to the larynx. The epiglottis (the lid of the larynx) diverts material away from the glottis during swallowing. The larynx produces sounds by vibrating expired air through the vocal cords. Changes in vocal cord length and air

pressure affect pitch and voice intensity. The larynx also stimulates the vital cough reflex when a foreign body touches its sensitive mucosa.

Assessment

Using a bright light and a tongue blade, inspect the patient's mouth and throat. Look for inflammation or white patches, and any irregularities on the tongue or throat. Make sure the patient's airway isn't compromised and also assess vital signs. Watch for and immediately report signs of respiratory distress (dyspnea, tachycardia, tachypnea, inspiratory stridor, restlessness, and nasal flaring) and changes in voice or in skin color, such as circumoral or nail bed cyanosis. Assess symmetry of the tongue as well as function of the soft palate. The main diagnostic test used in throat assessment is a culture to identify the infective organism.

EXTERNAL EAR

Otitis externa

Otitis externa, inflammation of the skin of the external ear canal and auricle, may be acute or chronic. Also known as *external otitis* and *swimmer's ear,* it's most common in the summer. With treatment, acute otitis externa usually subsides within 7 days— although it may become chronic—and tends to recur.

Causes and incidence

Otitis externa usually results from bacteria, such as *Pseudomonas, Proteus vulgaris, Staphylococcus aureus,* and streptococci and, sometimes, from fungi, such as *Aspergillus niger* and *Candida albicans* (fungal otitis externa is most common in tropical regions). Occasionally, chronic otitis externa results from dermatologic conditions, such as seborrhea or psoriasis. Allergic reactions stemming from nickel or chromium earrings, chemicals in hair spray, cosmetics, hearing aids, and medications (such as sulfonamide and neomycin, which is commonly used to treat otitis externa) can also cause otitis externa.

Predisposing factors include:

- swimming in contaminated water (Cerumen creates a culture medium for the waterborne organism.)

- cleaning the ear canal with a cotton swab, bobby pin, finger, or other foreign object (This irritates the ear canal and, possibly, introduces the infecting microorganism.)
- exposure to dust or hair-care products (such as hair spray or other irritants), which causes the patient to scratch his ear, excoriating the auricle and canal
- regular use of earphones, earplugs, or earmuffs, which trap moisture in the ear canal, creating a culture medium for infection (especially if earplugs don't fit properly)
- chronic drainage from a perforated tympanic membrane
- perfumes or self-administered eardrops.

Complications

- Complete closure of the ear canal
- Significant hearing loss
- Otitis media
- Cellulitis
- Abscesses
- Stenosis

Signs and symptoms

Acute otitis externa characteristically produces moderate to severe pain that's exacerbated by manipulating the auricle or tragus, clenching the teeth, opening the mouth, or chewing. Its other clinical effects may include fever, foul-smelling discharge, crusting in the external ear, regional cellulitis, partial hearing loss, and itching. It's usually difficult to view the tympanic membrane because of pain in the external canal. Hearing acuity is normal unless complete occlusion has occurred.

Fungal otitis externa may be asymptomatic, although *A. niger* produces a black or gray, blotting, paperlike growth in the ear canal. In chronic otitis externa, pruritus replaces pain, and scratching may lead to scaling and skin thickening. Aural discharge may also occur.

Diagnosis

CONFIRMING DIAGNOSIS

Physical examination confirms otitis externa. In acute otitis externa, otoscopy reveals a swollen external ear canal (sometimes to the point of complete closure), periauricular lymphadenopathy (tender nodes anterior to the tragus, posterior to the ear, or in the upper neck) and, occasionally, regional cellulitis.

In fungal otitis externa, removal of the growth reveals thick red epithelium. Microscopic examination or culture and sensitivity tests can identify the causative organism and determine antibiotic treatment. Pain on palpation of the tragus or auricle distinguishes acute otitis externa from acute otitis media. (See *Differentiating acute otitis externa from acute otitis media*, page 694.)

In chronic otitis externa, physical examination reveals thick red epithelium in the ear canal. Severe chronic otitis externa may reflect underlying diabetes mellitus, hypothyroidism, or nephritis. Microscopic examination or culture and sensitivity tests can identify the causative organism and help in the determination of antibiotic treatment.

Treatment

To relieve the pain of acute otitis externa, treatment includes heat therapy to the periauricular region (heat lamp; hot, damp compresses; or a heating pad), aspirin or acetaminophen, and codeine. Instillation of antibiotic eardrops (with or without hydrocortisone) follows cleaning of the ear and removal of debris. However, a corticosteroid helps reduce the inflammatory response. If fever persists or regional cellulitis or tender postauricular adenopathy develops, a systemic antibiotic is necessary.

If the ear canal is too edematous for the instillation of eardrops, an ear wick may be used for the first few days.

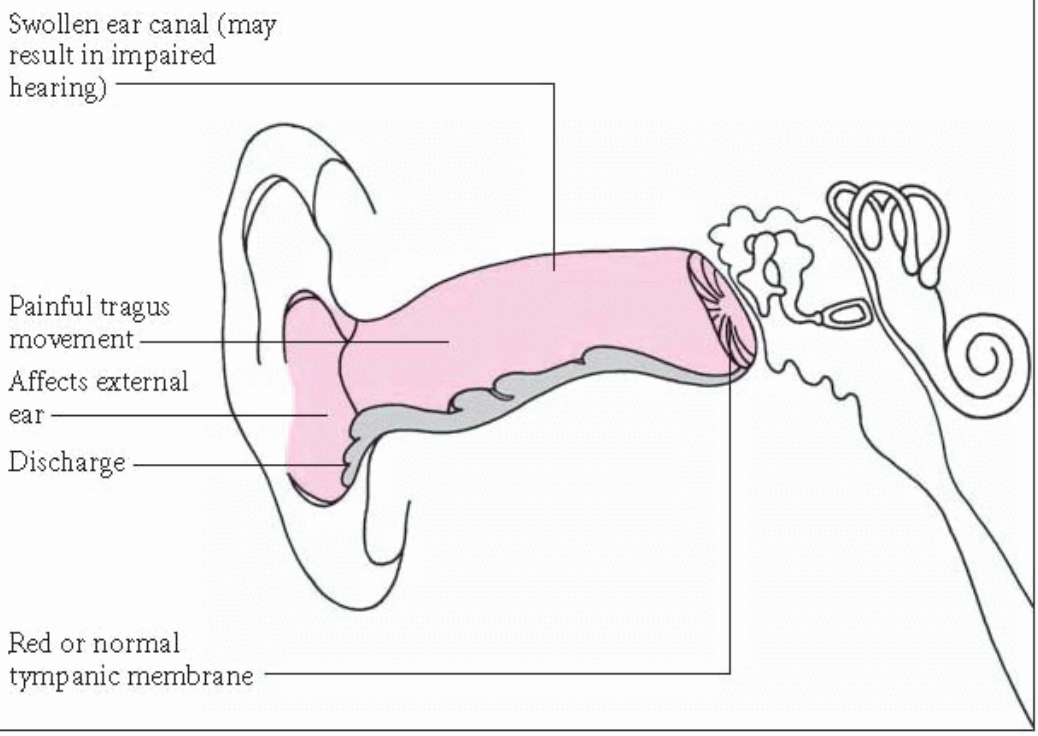
Topical treatment is generally required for otitis externa, as systemic antibiotics alone aren't sufficient. Analgesics, such as acetaminophen or ibuprofen, may be required temporarily.

As with other forms of this disorder, fungal otitis externa necessitates careful cleaning of the ear. Application of a keratolytic or 2% salicylic acid in cream containing nystatin may help treat otitis externa resulting from candidal organisms. Instillation of slightly acidic eardrops creates an unfavorable environment in the ear canal for most fungi as well as *Pseudomonas*. No specific treatment exists for otitis externa caused by *A. niger,* except repeated cleaning of the ear canal with baby oil.

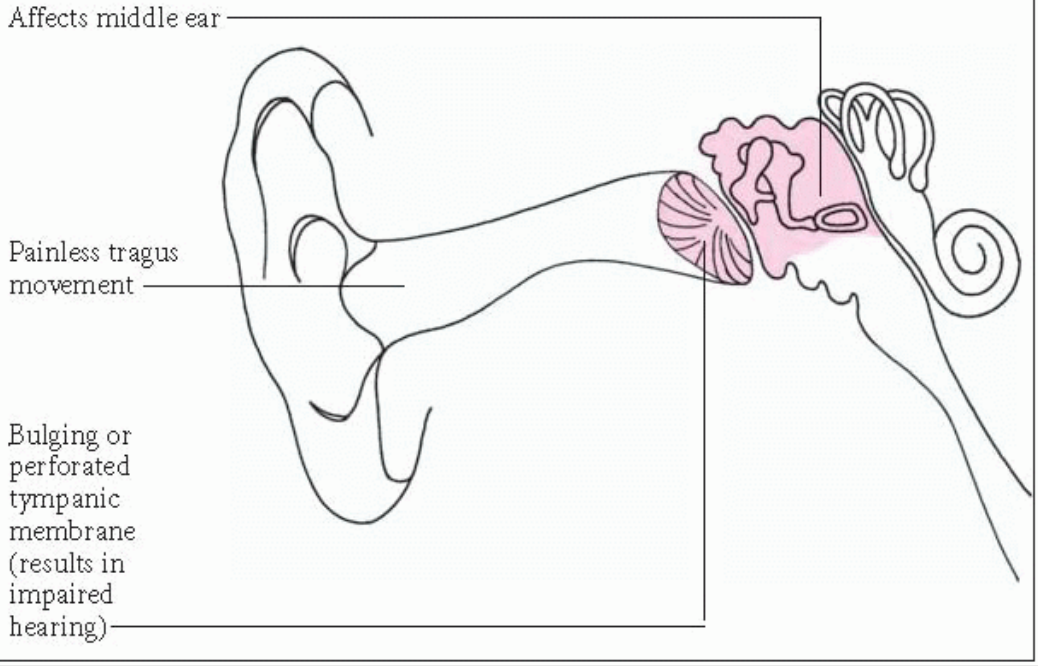
DIFFERENTIATING ACUTE OTITIS EXTERNA FROM ACUTE OTITIS MEDIA

Use the assessment findings shown below to help differentiate acute otitis externa from acute otitis media.

ACUTE OTITIS EXTERNA (OCCURS PRIMARILY IN SUMMER)



ACUTE OTITIS MEDIA (OCCURS PRIMARILY IN WINTER)



PREVENTION

PREVENTING OTITIS EXTERNA

Any patient who has experienced otitis externa should be taught to prevent a recurrence by avoiding irritants, such as hair-care products and earrings, and by avoiding cleaning the ears with cottontipped applicators or other objects. Encourage him to keep water out of his ears when showering or shampooing by using lamb's wool earplugs, coated with petroleum jelly. Also, parents of young children should be told that modeling clay makes a tight seal to prevent water from getting into the external ear canal.

In addition, when the patient goes swimming he should keep his head above water or wear earplugs. Following swimming, he should instill one or two drops of a mixture that is one-half 70% alcohol and one-half white vinegar to toughen the skin of the external ear canal.

In chronic otitis externa, primary treatment consists of cleaning the ear and removing debris. Supplemental therapy includes instillation of antibiotic eardrops or application of antibiotic ointment or cream (neomycin, bacitracin, or polymyxin B, possibly combined with hydrocortisone). Another ointment contains phenol, salicylic acid, precipitated sulfur, and petroleum jelly and produces exfoliative and antipruritic effects.

For mild chronic otitis externa, treatment may include instillation of antibiotic eardrops once or twice weekly and wearing of specially fitted earplugs while the patient is showering, shampooing, or swimming.

Special considerations

If the patient has acute otitis externa:

- The patient shouldn't participate in any swimming activity.
- Have the patient return to the clinic in 1 week for evaluation of the tympanic membrane to make sure it's intact.

- Monitor vital signs, particularly temperature. Watch for and record the type and amount of aural drainage.
- Remove debris and gently clean the ear canal with mild Burow's solution (aluminum acetate). Place a wisp of cotton soaked with solution into the ear, and apply a saturated compress directly to the auricle. Afterward, dry the ear gently but thoroughly. (In severe otitis externa, such cleaning may be delayed until after initial treatment with antibiotic eardrops.)
- To instill eardrops in an adult, grasp the helix and pull upward and backward to straighten the canal.

PEDIATRIC TIP

To instill eardrops in a child, pull the earlobe downward and backward. To ensure that the drops reach the epithelium, insert a wisp of cotton moistened with eardrops.

- Tell the patient to notify the physician if he develops an allergic reaction to the antibiotic drops or ointment, which may be indicated by increased swelling and discomfort of the area and worsening of other symptoms.

  If the patient has chronic otitis externa, clean the ear thoroughly. Use wet soaks intermittently on oozing or infected skin. If the patient has a chronic fungal infection, clean the ear canal well, then apply an exfoliative ointment.
- Urge prompt treatment for otitis media to prevent perforation of the tympanic membrane. (See *Preventing otitis externa*.)

ELDER TIP

If the patient is an elderly person or has diabetes, evaluate him for malignant otitis externa.

PEDIATRIC TIP

Children who have an intact tympanic membrane but are predisposed to otitis externa from swimming should instill

two to three drops of a 1:1 solution of white vinegar and 70% ethyl alcohol into their ears before and after swimming.

Benign tumors of the ear canal

Benign tumors may develop anywhere in the ear canal. Common types include keloids, osteomas, and sebaceous cysts; their causes vary. (See *Causes and characteristics of benign ear tumors*.) These tumors seldom become malignant; with proper treatment, the prognosis is excellent.

Signs and symptoms

A benign ear tumor is usually asymptomatic, unless it becomes infected, in which case pain, fever, or inflammation may result. (Pain is usually a sign of a malignant tumor.) If the tumor grows large enough to obstruct the ear canal by itself or through accumulated cerumen and debris, it may cause hearing loss and the sensation of pressure.

CAUSES AND CHARACTERISTICS OF BENIGN EAR TUMORS

Tumor	Causes and incidence	Characteristics
Keloid	▪ Surgery or trauma such as ear piercing ▪ Most common in blacks	▪ Hypertrophy and fibrosis of scar tissue ▪ Commonly recurs
Osteoma	▪ Idiopathic growth ▪ Predisposing factor: swimming in cold water ▪ Three times more common in males than in females ▪ Seldom occurs before adolescence	▪ Bony outgrowth from wall of external auditory meatus ▪ Usually bilateral and multiple (exostoses) ▪ May be circumscribed or diffuse, nondisplaceable, nontender
Sebaceous cyst	▪ Obstruction of a sebaceous gland	▪ Painless, circumscribed, round mass of variable size filled with oily, fatty, glandular secretions

	▪ May occur on external ear and outer third of external auditory canal

Diagnosis

CONFIRMING DIAGNOSIS

Clinical features and patient history suggest a benign tumor of the ear canal; otoscopy confirms it. To rule out cancer, a biopsy may be necessary.

Treatment

Generally, a benign tumor requires surgical excision if it obstructs the ear canal, is cosmetically undesirable, or becomes malignant.

Treatment for keloids may include surgery followed by repeated injections of long-acting steroids into the suture line. Excision must be complete, but even this may not prevent recurrence.

Surgical excision of an osteoma consists of elevating the skin from the surface of the bony growth and shaving the osteoma with a mechanical burr or drill.

Before surgery, a sebaceous cyst requires preliminary treatment with antibiotics, to reduce inflammation. To prevent recurrence, excision must be complete, including the sac or capsule of the cyst.

Special considerations

Because treatment for benign ear tumors generally doesn't require hospitalization, focus care on emotional support and providing appropriate patient education so that the patient follows his therapeutic plan properly when he's at home.

- Thoroughly explain diagnostic procedures and treatment to the patient and his family. Reassure them and answer any questions they may have.
- After surgery, instruct the patient in good aural hygiene. Until his ear is completely healed, advise him not to insert anything into his ear or

allow water to get into it. Suggest that he cover his ears with a cap when showering.

- Teach the patient how to recognize signs of infection, such as pain, fever, localized redness, and swelling. If he detects any of these signs, instruct him to report them immediately.

MIDDLE EAR

Otitis media

Otitis media, inflammation of the middle ear, may be suppurative or secretory, acute, persistent, unresponsive, or chronic. With prompt treatment, the prognosis for acute otitis media is excellent; however, prolonged accumulation of fluid within the middle ear cavity causes chronic otitis media and, possibly, perforation of the tympanic membrane. (See *Site of otitis media*, page 698.)

Chronic suppurative otitis media may lead to scarring, adhesions, and severe structural or functional ear damage. Chronic secretory otitis media, with its persistent inflammation and pressure, may cause conductive hearing loss.

Recurrent otitis media is defined as three near-acute otitis media episodes within 6 months or four episodes of acute otitis media within 1 year.

Otitis media with complications involves damage to middle ear structures (such as adhesions, retraction, pockets, cholesteatoma, and intratemporal and intracranial complications).

Causes and incidence

Otitis media results from disruption of eustachian tube patency. In the suppurative form, respiratory tract infection, allergic reaction, nasotracheal intubation, or positional changes allow nasopharyngeal flora to reflux through the eustachian tube and colonize the middle ear. Suppurative otitis media usually results from bacterial infection with pneumococcus, *Haemophilus influenzae* (the most common cause in children younger than age 6), *Moraxella catarrhalis,* beta-hemolytic streptococci, staphylococci (most common cause in children age 6 or

older), or gram-negative bacteria. Predisposing factors include the normally wider, shorter, more horizontal eustachian tubes and increased lymphoid tissue in children, as well as anatomic anomalies. Chronic suppurative otitis media results from inadequate treatment for acute otitis episodes or from infection by resistant strains of bacteria or, rarely, tuberculosis.

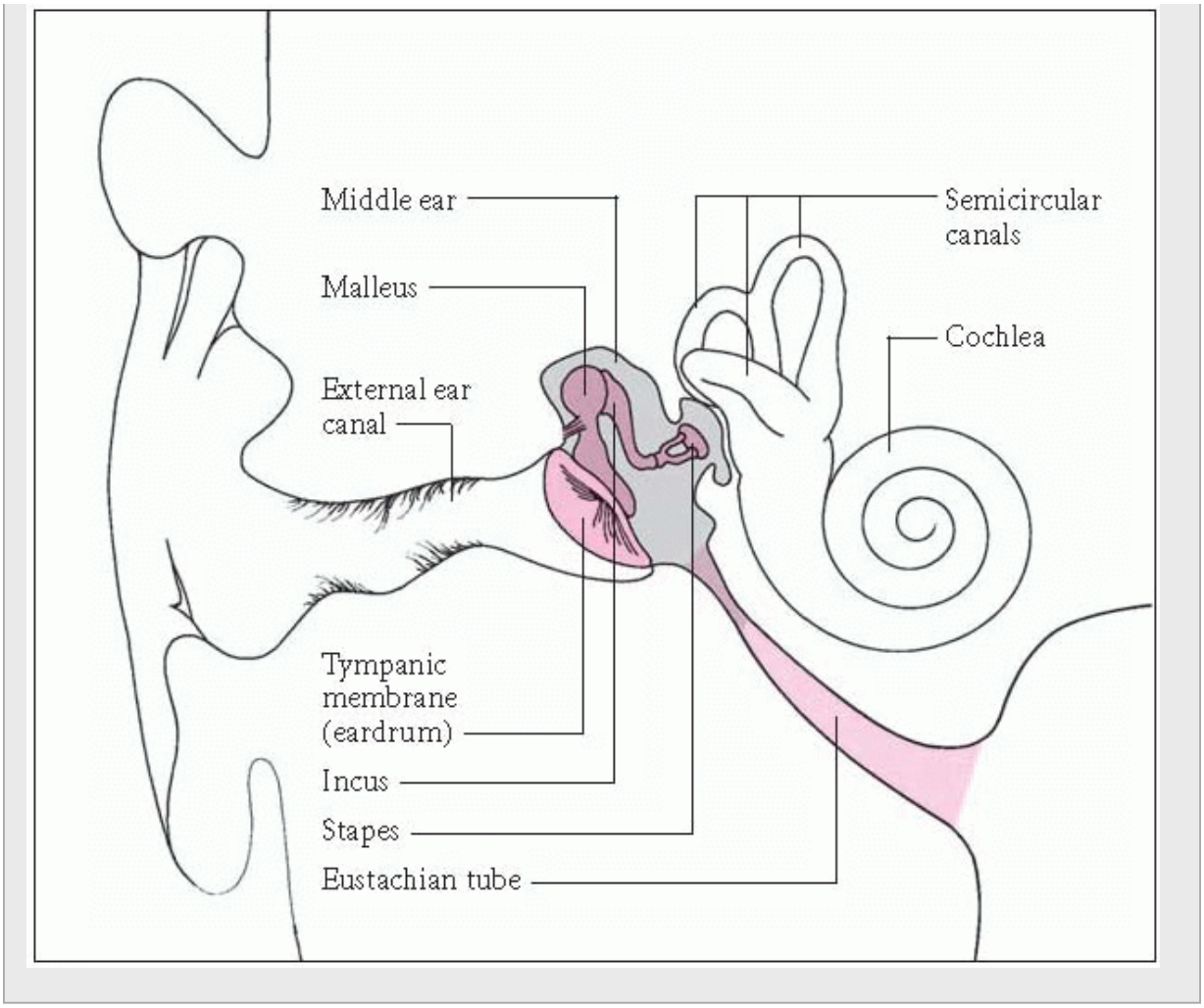
Secretory otitis media results from obstruction of the eustachian tube. This causes a buildup of negative pressure in the middle ear that promotes transudation of sterile serous fluid from blood vessels in the membrane of the middle ear. Such effusion may be secondary to eustachian tube dysfunction from viral infection or allergy. It may also follow barotrauma (pressure injury caused by the inability to equalize pressures between the environment and the middle ear), as occurs during rapid aircraft descent in a person with an upper respiratory tract infection or during rapid underwater ascent in scuba diving (barotitis media).

Chronic secretory otitis media follows persistent eustachian tube dysfunction from mechanical obstruction (adenoidal tissue overgrowth or tumors), edema (allergic rhinitis or chronic sinus infection),

or inadequate treatment for acute suppurative otitis media.

SITE OF OTITIS MEDIA

The common site of otitis media is shown below.



Acute otitis media is common in children; its incidence rises during the winter months, paralleling the seasonal rise in nonbacterial respiratory tract infections. Chronic secretory otitis media most commonly occurs in children with tympanostomy tubes or those with a perforated tympanic membrane.

Complications

- Spontaneous rupure of the tympanic membrane
- Persistent perforation
- Chronic otitis media
- Mastoiditis
- Abscesses

- Vertigo
- Permanent hearing loss

Signs and symptoms

Clinical features of acute suppurative otitis media include severe, deep, throbbing pain (from pressure behind the tympanic membrane); signs of upper respiratory tract infection (sneezing or coughing); mild to very high fever; hearing loss (usually mild and conductive); tinnitus; dizziness; nausea; and vomiting. Other possible effects include bulging of the tympanic membrane, with concomitant erythema, and purulent drainage in the ear canal from tympanic membrane rupture. However, many patients are asymptomatic.

Acute secretory otitis media produces a severe conductive hearing loss—which varies from 15 to 35 dB, depending on the thickness and amount of fluid in the middle ear cavity—and, possibly, a sensation of fullness in the ear and popping, crackling,

or clicking sounds on swallowing or with jaw movement. Accumulation of fluid may also cause the patient to hear an echo when he speaks and to experience a vague feeling of top-heaviness.

The cumulative effects of chronic otitis media include thickening and scarring of the tympanic membrane, decreased or absent tympanic membrane mobility, cholesteatoma (a cystlike mass in the middle ear) and, in chronic suppurative otitis media, a painless, purulent discharge. The extent of associated conductive hearing loss varies with the size and type of tympanic membrane perforation and ossicular destruction.

If the tympanic membrane has ruptured, the patient may state that the pain has suddenly stopped. Complications may include abscesses (brain, subperiosteal, and epidural), sigmoid sinus or jugular vein thrombosis, septicemia, meningitis, suppurative labyrinthitis, facial paralysis, and otitis externa.

PEDIATRIC TIP

The following factors increase a child's risk of developing otitis media:

- *acute otitis media in the first year after birth (recurrent otitis media)*
- *day care*
- *family history of middle ear disease*
- *formula feeding*
- *male gender*
- *sibling history of otitis media*
- *smoking in the household.*

Acute otitis media may not produce any symptoms in the first few months of life; irritability may be the only indication of earache.

Diagnosis

In acute suppurative otitis media, otoscopy reveals obscured or distorted bony landmarks of the tympanic membrane. Pneumatoscopy can show decreased tympanic membrane mobility, but this procedure is painful with an obviously bulging, erythematous tympanic membrane. The pain pattern is diagnostically significant: For example, in acute suppurative otitis media, pulling the auricle *doesn't* exacerbate the pain. A culture of the ear drainage identifies the causative organism.

In acute secretory otitis media, otoscopic examination reveals tympanic membrane retraction, which causes the bony landmarks to appear more prominent.

Examination also detects clear or amber fluid behind the tympanic membrane. If hemorrhage into the middle ear has occurred, as in barotrauma, the tympanic membrane appears blue-black.

In chronic otitis media, patient history discloses recurrent or unresolved otitis media. Otoscopy shows thickening, sometimes scarring, and decreased mobility of the tympanic membrane; pneumatoscopy shows decreased or absent tympanic membrane movement. A history of recent air travel or scuba diving suggests barotitis media.

Tympanocentesis for microbiologic diagnosis is recommended for treatment failures and may be followed by myringotomy. Tympanometry, acoustic reflex measurement, or acoustic reflexometry may be needed

to document the presence of fluid in the middle ear. White blood cell count is higher in bacterial otitis media than in sterile otitis media. Mastoid X-rays or computed tomography scan of the head or mastoids may show the spreading of the infection beyond the middle ear.

Treatment

In acute suppurative otitis media, antibiotic therapy includes amoxicillin. In areas with a high incidence of beta-lactamase-producing *H. influenzae* and in patients who aren't responding to ampicillin or amoxicillin, amoxicillin/clavulanate potassium may be used. For those who are allergic to penicillin derivatives, therapy may include cefaclor or co-trimoxazole. Severe, painful bulging of the tympanic membrane usually necessitates myringotomy. Broad-spectrum antibiotics can help prevent acute suppurative otitis media in high-risk patients. A single dose of ceftriaxone 50 mg/kg is effective against major pathogens but is expensive and is reserved for very sick infants. In the patient with recurring otitis media, antibiotics must be used with discretion to prevent development of resistant strains of bacteria.

PREVENTION

PREVENTING OTITIS MEDIA

For a patient recovering from otitis media at home, teach these guidelines to help prevent a recurrence.

Instruct the patient how to recognize upper respiratory infections, and encourage early treatment. Encourage the patient to get a pneumococcal vaccine to prevent infections that can cause respiratory and aural infections.

Tell parents to wash children's toys and promote frequent hand washing. For infants, tell parents to avoid the use of pacifiers and encourage breast-feeding for at least the first six months of the child's life. It has been shown that breast milk contains antibodies that protect the infant from ear infections. If the child is bottle-fed, instruct the parents not to feed the infant in a supine position and not

to put him to bed with a bottle. Explain that doing so could cause reflux of nasopharyngeal flora. Also, teach the parent to keep the child away from secondhand smoke.

To promote eustachian tube patency, instruct the patient to perform Valsalva's maneuver several times a day, especially during airplane travel. Also, explain adverse reactions to the prescribed medications, emphasizing those that require immediate medical attention.

In acute secretory otitis media, inflation of the eustachian tube using Valsalva's maneuver several times a day may be the only treatment required. Otherwise, nasopharyngeal decongestant therapy may be helpful. It should continue for at least 2 weeks and, sometimes, indefinitely, with periodic evaluation. If decongestant therapy fails, myringotomy and aspiration of middle ear fluid are necessary, followed by insertion of a polyethylene tube into the tympanic membrane, for immediate and prolonged equalization of pressure. The tube falls out spontaneously after 9 to 12 months. Concomitant treatment for the underlying cause (such as elimination of allergens, or adenoidectomy for hypertrophied adenoids) may also be helpful in correcting this disorder.

Treatment for chronic otitis media includes broad-spectrum antibiotics, such as amoxicillin/clavulanate potassium or cefuroxime, for exacerbations of acute otitis media; elimination of eustachian tube obstruction; treatment for otitis externa; myringoplasty and tympanoplasty to reconstruct middle ear structures when thickening and scarring are present and, possibly, mastoidectomy. Cholesteatoma requires excision.

Special considerations

- Explain all diagnostic tests and procedures. After myringotomy, maintain drainage flow. Don't place cotton or plugs deeply into the ear canal; however, sterile cotton may be placed loosely in the external ear to absorb drainage. To prevent infection, change the cotton whenever it gets damp, and wash hands before and after giving ear

care. Watch for and report headache, fever, severe pain, or disorientation.

- After tympanoplasty, reinforce dressings, and observe for excessive bleeding from the ear canal. Administer analgesics as needed. Warn the patient against blowing his nose or getting the ear wet when bathing.
- Encourage the patient to complete the prescribed course of antibiotic treatment. If nasopharyngeal decongestants are ordered, teach correct instillation.
- Suggest application of heat to the ear to relieve pain. (See *Preventing otitis media.*)
- Advise the patient with acute secretory otitis media to watch for and immediately

report pain and fever—signs of secondary infection.

- Identify and treat allergies.

Mastoiditis

Mastoiditis is a bacterial infection and inflammation of the air cells of the mastoid antrum. Although the prognosis is good with early treatment, possible complications include meningitis, facial paralysis, brain abscess, and suppurative labyrinthitis.

Causes and incidence

Bacteria that cause mastoiditis include pneumococci, *Haemophilus influenzae, Moraxella catarrhalis,* beta-hemolytic streptococci, staphylococci, and gram-negative organisms. Mastoiditis is usually a complication of chronic otitis media; less frequently, it develops after acute otitis media. An accumulation of pus under pressure in the middle ear cavity results in necrosis of adjacent tissue and extension of the infection into the mastoid cells. Chronic systemic diseases or immunosuppression may also lead to mastoiditis. Anaerobic organisms play a role in chronic mastoiditis.

PEDIATRIC TIP

Acute otitis media increases a child's risk of developing mastoiditis. If mastoiditis does occur in infants younger than age 1, the swelling occurs superior to the ear and pushes the pinna downward instead of outward. I.V. antibiotic treatment choice includes ampicillin or cefuroxime. Before antibiotics, mastoiditis was one of the leading causes of death in children; now, it's uncommon and less dangerous.

Complications

- Destruction of the mastoid bone
- Facial paralysis
- Meningitis
- Partial or complete hearing loss

Signs and symptoms

Primary clinical features include a dull ache and tenderness in the area of the mastoid process, low-grade fever, headache, and a thick, purulent discharge that gradually becomes more profuse, possibly leading to otitis externa. Postauricular erythema and edema may push the auricle out from the head; pressure within the edematous mastoid antrum may produce swelling and obstruction of the external ear canal, causing conductive hearing loss.

Diagnosis

X-rays or computed tomography scan of the mastoid area reveal hazy mastoid air cells; the bony walls between the cells appear decalcified. Audiometric testing may reveal a conductive hearing loss. Physical examination shows a dull, thickened, and edematous tympanic membrane, if the membrane isn't concealed by obstruction. During examination, the external ear canal is cleaned; persistent oozing into the canal indicates perforation of the tympanic membrane.

Treatment

Treatment for mastoiditis consists of intense parenteral antibiotic therapy. Reasonable initial antibiotic choices include ceftriaxone with nafcillin or clindamycin. If bone damage is minimal, myringotomy or tympanocentesis drains purulent fluid and provides a specimen of discharge for culture and sensitivity testing. Recurrent or persistent infection or signs of intracranial complications necessitate simple mastoidectomy. This procedure involves removal of the diseased bone and cleaning of the affected area, after which a drain is inserted.

A chronically inflamed mastoid requires radical mastoidectomy (excision of the posterior wall of the ear canal, remnants of the tympanic membrane, and the malleus and incus, although these bones are usually destroyed by infection before surgery). The stapes and facial nerve remain intact. Radical mastoidectomy, which is seldom necessary because of antibiotic therapy, doesn't drastically affect the patient's hearing because significant hearing loss precedes surgery. With either surgical procedure, the patient continues oral antibiotic therapy for several weeks after surgery and facility

discharge. The prognosis is good if treatment is started early.

Indications for immediate surgical intervention include meningitis, brain abscess, cavernous sinus thrombosis, acute suppurative labyrinthitis, and facial palsy.

Special considerations

- After simple mastoidectomy, give pain medication as needed. Check wound drainage and reinforce dressings (the surgeon usually changes the dressing daily and removes the drain in 72 hours). Check the patient's hearing, and watch for signs of complications, especially infection (either localized or extending to the brain); facial nerve paralysis, with unilateral facial drooping; bleeding; and vertigo, especially when the patient stands.
- After radical mastoidectomy, the wound is packed with petroleum gauze or gauze treated with an antibiotic ointment. Give pain

medication before the packing is removed, on the fourth or fifth postoperative day.

- Because of stimulation to the inner ear during surgery, the patient may feel dizzy and nauseated for several days afterward. Keep the side rails up, and assist the patient with ambulation. Also, give antiemetics as needed.
- Before discharge, teach the patient and his family how to change and care for the dressing. Urge compliance with the prescribed antibiotic treatment, and promote regular follow-up care.
- If the patient is an elderly person or diabetic, evaluate him for malignant otitis externa.

ELDER TIP

Encourage the patient to seek early treatment for ear infections.

Otosclerosis

The most common cause of chronic, progressive conductive hearing loss, otosclerosis is the slow formation of spongy bone in the otic capsule, particularly at the oval window. With surgery, the prognosis is good.

Causes and incidence

Otosclerosis appears to result from a genetic factor transmitted as an autosomal dominant trait; many patients report family histories of hearing loss (excluding presbycusis). Pregnancy may trigger onset of this condition.

Otosclerosis occurs in at least 10% of the U.S. population. It's three times more prevalent in females than in males, usually affecting people between ages 15 and 30. Whites are most susceptible.

Complications

- Bilateral conductive hearing loss
- Taste disturbance

Signs and symptoms

Spongy bone in the otic capsule immobilizes the footplate of the normally mobile stapes, disrupting the conduction of vibrations from the tympanic membrane to the cochlea. This causes progressive unilateral hearing loss, which may advance to bilateral deafness. Other symptoms include tinnitus and paracusis of Willis (hearing conversation better in a noisy environment than in a quiet one).

Diagnosis

Early diagnosis is based on a Rinne test that shows bone conduction lasting longer than air conduction (normally, the reverse is true). As otosclerosis progresses, bone conduction also deteriorates. Audiometric testing reveals hearing loss ranging from 60 dB in early stages to total loss. Weber's test detects sound lateralizing to the more affected ear. Physical examination reveals a normal tympanic membrane. Head computed tomography scan and X-ray help distinguish otosclerosis from other causes of hearing loss.

Treatment

Treatment consists of stapedectomy (removal of the stapes) and insertion of a prosthesis to restore partial or total hearing. This procedure is performed on only one ear at a time, beginning with the ear that has suffered greater damage. Alternative surgery includes stapedotomy (creation

of a small hole in the stapes' footplate), through which a wire and piston are inserted. (See *Types of stapedectomy.*) Recent procedural innovations involve laser surgery. Postoperatively, treatment includes antibiotics to prevent infection. If surgery isn't possible, a hearing aid (air conduction aid with molded ear insert receiver) enables the patient to hear conversation in normal surroundings, although this therapy isn't as effective as stapedectomy.

Special considerations

- During the first 24 hours after surgery, keep the patient supine, with the affected ear facing upward (to maintain the position of the graft). Enforce bed rest with bathroom privileges for 48 hours. Because the patient may be dizzy, keep the side rails up, and assist him with ambulation. Assess for pain and vertigo, which may be relieved with repositioning or prescribed medication.

ALERT

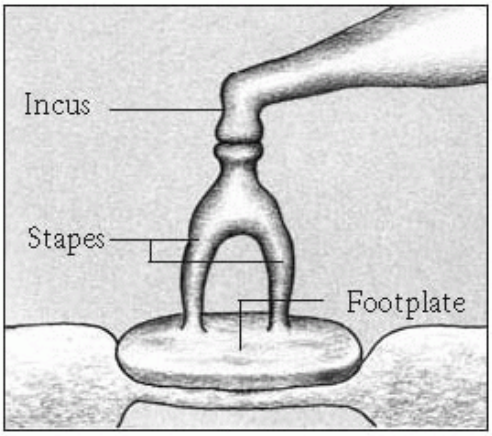
Watch for and report postoperative facial drooping, which may indicate swelling of or around the facial nerve.

- Tell the patient that his hearing won't return until edema subsides and packing is removed.
- Before discharge, instruct the patient to avoid loud noises and sudden pressure changes (such as those that occur while diving or flying) until healing is complete (usually 6 months). Advise the patient not to blow his nose for at least 1 week to prevent contaminated air and bacteria from entering the eustachian tube.
- Stress the importance of protecting the ears against cold; avoiding any activities that provoke dizziness, such as straining, bending, or heavy lifting and, if possible, avoiding contact with anyone who has an upper respiratory tract infection. Teach the patient and his family how to change the external ear dressing (eye or gauze pad) and care for the incision. Emphasize the need to complete the prescribed antibiotic regimen and to return for scheduled follow-up care.

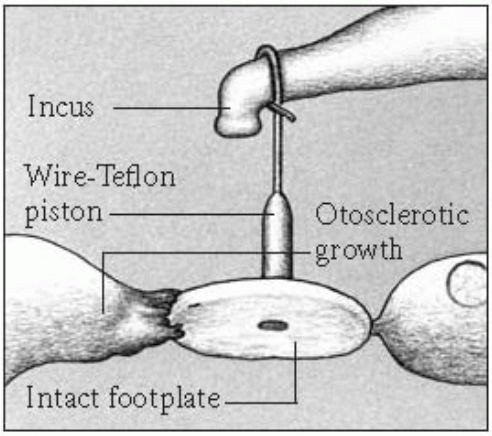
TYPES OF STAPEDECTOMY

Surgery may remove part or all of the stapes, depending on the extent of otosclerotic growth. It may be performed using various techniques. Two techniques used to implant prostheses are depicted below.

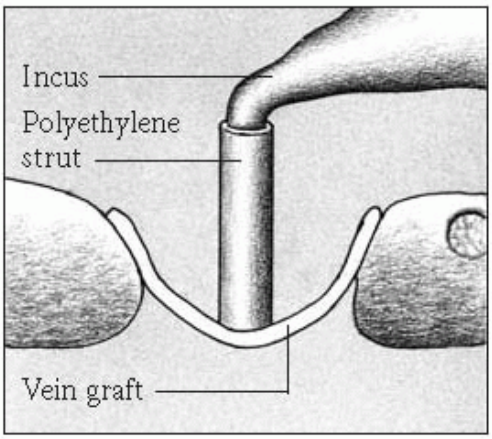
NORMAL MIDDLE EAR



PARTIAL STAPEDECTOMY
Wire-Teflon prosthesis



TOTAL STAPEDECTOMY
Vein graft and strut prosthesis



Infectious myringitis

Acute infectious myringitis is characterized by inflammation, hemorrhage, and effusion of fluid into the tissue at the end of the external ear canal and the tympanic membrane. This self-limiting disorder (resolving spontaneously within 3 days to 2 weeks) commonly follows acute otitis media or upper respiratory tract infection.

Chronic granular myringitis, a rare inflammation of the squamous layer of the tympanic membrane, causes gradual hearing loss. Without specific treatment, this condition can lead to stenosis of the ear canal, as granulation extends from the tympanic membrane to the external ear.

Causes and incidence

Acute infectious myringitis usually follows viral infection but may also result from infection with bacteria (pneumococcus, *Haemophilus influenzae,* beta-hemolytic streptococci, staphylococci) or any other organism that can cause acute otitis media. Myringitis is a rare sequela of atypical pneumonia caused by *Mycoplasma pneumoniae*. The cause of chronic granular myringitis is unknown.

Acute infectious myringitis frequently occurs epidemically in children.

Complications

- Gradual hearing loss
- Stenosis of the ear canal

Signs and symptoms

Acute infectious myringitis begins with severe ear pain, commonly accompanied by tenderness over the mastoid process. Small, reddened, inflamed blebs form in the canal, on the tympanic membrane and, with bacterial invasion, in the middle ear. Fever and hearing loss are rare unless fluid accumulates in the middle ear, or a large bleb totally obstructs the external auditory meatus. Spontaneous rupture of these blebs may cause bloody discharge. Chronic granular myringitis produces pruritus, purulent discharge, and gradual hearing loss.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis of acute infectious myringitis is based on physical examination showing characteristic blebs and a typical patient history. Culture and sensitivity testing of exudate identifies secondary infection. In chronic granular myringitis, physical examination may reveal granulation extending from the tympanic membrane to the external ear.

Treatment

Hospitalization usually isn't required for acute infectious myringitis. Treatment consists of measures to relieve pain: analgesics, such as aspirin or acetaminophen, and application of heat to the external ear are usually sufficient, but severe pain may necessitate use of codeine.

ALERT

Aspirin and combination aspirin products aren't recommended for people younger than age 19 during episodes of fever-causing illnesses because the use of aspirin has been linked to Reye's syndrome.

Systemic or topical antibiotics prevent or treat secondary infection. Incision of blebs and evacuation of serum and blood may relieve pressure and help drain exudate but don't speed recovery.

Treatment for chronic granular myringitis consists of systemic antibiotics or local anti-inflammatory/antibiotic combination eardrops, and surgical excision and cautery. If stenosis is present, surgical reconstruction is necessary.

Special considerations

- Stress the importance of completing the prescribed antibiotic therapy.

- Teach the patient how to instill topical antibiotics (eardrops). When necessary, explain incision of blebs.

PREVENTION

Advise early treatment for acute otitis media.

INNER EAR

Ménière's disease

Ménière's disease, a labyrinthine dysfunction also known as *endolymphatic hydrops,* produces severe vertigo, sensorineural hearing loss, and tinnitus. After multiple attacks over several years, this disorder leads to residual tinnitus and hearing loss. Usually, only one ear is involved.

Causes and incidence

The exact cause of Ménière's disease is unknown. It may result from overproduction or decreased absorption of endolymph, which causes endolymphatic hydrops or endolymphatic hypertension, with consequent degeneration of the vestibular and cochlear hair cells. This condition may also stem from autonomic nervous system dysfunction that produces a temporary constriction of blood vessels supplying the inner ear. In some cases, Ménière's disease may be related to otitis media, syphilis, or head injury. Risk factors include recent viral illness, respiratory infection, stress, fatigue, use of prescription or nonprescription drugs (such as aspirin), and a history of allergies, smoking, and alcohol use. There also may be genetic risk factors: In some women, premenstrual edema may precipitate attacks of Ménière's disease.

In the United States, about 100,000 people per year develop Ménière's disease.

Complications

- Tinnitus
- Partial to total hearing loss

- Permanent balance disability

Signs and symptoms

Ménière's disease produces three characteristic effects: severe episodic vertigo, tinnitus, and sensorineural hearing loss. A feeling of fullness or blockage in the ear is also common. Violent paroxysmal attacks last from 10 minutes to several hours. During an acute attack, other symptoms include severe nausea, vomiting, sweating, giddiness, and nystagmus. Vertigo may cause loss of balance and falling to the affected side. Symptoms tend to wax and wane as the endolymphatic pressure rises and falls. To lessen these symptoms, the patient may assume a characteristic posture—lying on the side of the unaffected ear and looking in the direction of the affected ear.

Initially, the patient may be asymptomatic between attacks, except for residual tinnitus that worsens during an attack. Such attacks may occur several times a year, or remissions may last as long as several years. These attacks become less frequent as hearing loss progresses (usually unilaterally); they may cease when hearing loss is total. All symptoms are aggravated by motion.

Diagnosis

Presence of all three typical symptoms suggests Ménière's disease. Audiometric studies indicate a sensorineural hearing loss and loss of discrimination and recruitment. Selected studies such as electronystagmography, electrocochleography, computed tomography scan, magnetic resonance imaging, or X-rays of the internal meatus may be necessary for differential diagnosis.

Laboratory studies, including thyroid and lipid studies, may be performed to rule out other conditions such as *Treponema pallidum*.

Caloric testing may reveal loss or impairment of thermally induced nystagmus on the involved side. However, it's important not to overlook an acoustic tumor, which produces an identical clinical picture.

Treatment

Treatment with atropine may stop an attack in 20 to 30 minutes. Epinephrine or diphenhydramine may be necessary in a severe attack; dimenhydrinate, meclizine, diphenhydramine, or diazepam may be effective in a milder attack.

Long-term management includes use of a diuretic or vasodilator and restricted sodium intake (less than 2 g/day). A typical diuretic regime is hydrochlorothiazide 500 to 100 mg daily. Prophylactic antihistamines or mild sedatives (phenobarbital, diazepam) may also be helpful. If Ménière's

disease persists after 2 years of treatment, produces incapacitating vertigo, or resists medical management, surgery may be necessary. Destruction of the affected labyrinth permanently relieves symptoms but results in irreversible hearing loss. Systemic streptomycin is reserved for the patient with bilateral disease for whom no other treatment can be considered. If a patient fails medical therapy and remains disabled by his vertigo, surgical decompression of the endolymphatic sac may bring relief.

Special considerations

If the patient is in the hospital during an attack of Ménière's disease:

- Advise him against reading and exposure to glaring lights, to reduce dizziness.
- Keep the side rails of the patient's bed up to prevent falls. Tell him not to get out of bed or walk without assistance.
- Instruct the patient to avoid sudden position changes and any tasks that vertigo makes hazardous because an attack can begin quite rapidly. Hazardous activities, such as driving and climbing, should be avoided until one week after symptoms disappear.
- Before surgery, if the patient is vomiting, record fluid intake and output and characteristics of vomitus. Administer antiemetics as needed, and give small amounts of fluid frequently.
- After surgery, record intake and output carefully. Tell the patient to expect dizziness and nausea for 1 or 2 days after surgery. Give prophylactic antibiotics and antiemetics, as ordered.

Labyrinthitis

Labyrinthitis, an inflammation of the labyrinth of the inner ear, frequently incapacitates the patient by producing severe vertigo that lasts for 3 to 5 days; symptoms gradually subside over a 3- to 6-week period. Viral labyrinthitis is commonly associated with upper respiratory tract infections.

Causes

Labyrinthitis is usually caused by viral infection. It may be a primary infection, the result of trauma, or a complication of influenza, otitis media, or meningitis. In chronic otitis media, cholesteatoma formation erodes the bone of the labyrinth, allowing bacteria to enter from the middle ear. Toxic drug ingestion is another possible cause of labyrinthitis and neuronitis.

Complications

- Meningitis
- Permanent hearing loss
- Permanent balance disability

Signs and symptoms

Because the inner ear controls both hearing and balance, this infection typically produces severe vertigo (with any movement of the head) and sensorineural hearing loss. Vertigo begins gradually but peaks within 48 hours, causing loss of balance and falling in the direction of the affected ear. Other associated signs and symptoms include spontaneous nystagmus, with jerking movements of the eyes toward the unaffected ear, and nausea, vomiting, and giddiness. With cholesteatoma, signs of middle ear disease may appear. With severe bacterial infection, purulent drainage, increased salivation, generalized malaise, and perspiration can occur. To minimize symptoms such as giddiness and nystagmus, the patient may assume a characteristic posture —lying on the side of the unaffected ear and looking in the direction of the affected ear.

Diagnosis

A typical clinical picture and a history of upper respiratory tract infection suggest labyrinthitis. Typical diagnostic measures include culture and sensitivity testing to identify the infecting organism, if purulent drainage is present, and audiometric testing. When an infectious etiology can't be found, additional testing must be done to rule out a brain lesion or Ménière's disease.

Differentiation from other causes of dizziness or vertigo may include head computed tomography (CT) scan or magnetic resonance imaging, audiology or audiometry testing, caloric stimulation tests, electronystagmography, EEG, and evoked auditory potential studies.

Treatment

Symptomatic treatment includes bed rest, with the head immobilized between pillows, and antibiotics to combat diffuse purulent labyrinthitis. Oral fluids can prevent dehydration caused by vomiting. For severe nausea and vomiting, I.V. fluids may be necessary. Medications that help reduce symptoms include antihistamines, anticholinergics, sedative-hypnotics, and antiemetics; benzodiazepines help control vertigo.

When conservative management fails, treatment necessitates surgical excision of the cholesteatoma and drainage of the infected areas of the middle and inner ear. Prevention is possible by early and vigorous treatment for predisposing conditions, such as otitis media and any local or systemic infection.

Special considerations

- Keep the side rails up to prevent falls. Tell the patient to keep still and rest during attacks and to avoid sudden position changes.
- If vomiting is severe, administer antiemetics as ordered. Record intake and output, and give I.V. fluids as ordered.
- During an attack, dim the lighting and tell the patient to avoid reading.

- Tell the patient that recovery may take as long as 6 weeks. During this time, he should limit activities that vertigo may make hazardous. Hazardous activities, such as driving and climbing, should be avoided until one week after symptoms disappear.
- If recovery doesn't occur within 4 to 6 weeks, a CT scan should be performed to rule out an intracranial lesion.

Hearing loss

Hearing loss results from a mechanical or nervous impediment to the transmission of sound waves. The major forms of hearing loss are classified as *conductive loss* (interrupted passage of sound from the external ear to the junction of the stapes and oval window), *sensorineural loss* (impaired cochlea or acoustic [eighth cranial] nerve dysfunction, causing failure of transmission of sound impulses within the inner ear or brain), or *mixed loss* (combined dysfunction of conduction and sensorineural transmission). Hearing loss may be partial or total and is calculated from this American Medical Association formula: Hearing is 1.5% impaired for every decibel that the pure tone average exceeds 25 dB.

Causes and incidence

Congenital hearing loss may be transmitted as a dominant, autosomal dominant, autosomal recessive, or sex-linked recessive trait. Hearing loss in neonates may also result from trauma, toxicity, or infection during pregnancy or delivery. Predisposing factors include a family history of hearing loss or known hereditary disorders (otosclerosis, for example), maternal exposure to rubella or syphilis during pregnancy, use of ototoxic drugs during pregnancy, prolonged fetal anoxia during delivery, and congenital abnormalities of the ears, nose, or throat. Premature or low-birth-weight neonates are most likely to have structural or functional hearing impairment; those with serum bilirubin levels above 20 mg/dl also risk hearing impairment from the toxic effect of high serum bilirubin levels on the brain. In addition, trauma during delivery may cause intracranial hemorrhage and may damage the cochlea or the acoustic nerve.

Sudden deafness refers to sudden hearing loss in a person with no prior hearing impairment. This condition is considered a medical emergency because prompt treatment may restore full hearing. Its causes and predisposing factors may include:

- acute infections, especially mumps (most common cause of unilateral sensorineural hearing loss in children), and other bacterial and viral infections, such as rubella, rubeola, influenza, herpes zoster, and infectious mononucleosis; and mycoplasma infections
- blood dyscrasias (leukemia, hypercoagulation)
- head trauma or brain tumors
- metabolic disorders (diabetes mellitus, hypothyroidism, hyperlipoproteinemia)
- neurologic disorders (multiple sclerosis, neurosyphilis)
- ototoxic drugs (tobramycin, streptomycin, quinine, gentamicin, furosemide, ethacrynic acid)
- vascular disorders (hypertension, arteriosclerosis).

Noise-induced hearing loss, which may be transient or permanent, may follow prolonged exposure to loud noise (85 to 90 dB) or brief exposure to extremely loud noise (greater than 90 dB). Such hearing loss is common in workers subjected to constant industrial noise and in military personnel, hunters, and rock musicians.

Presbycusis, an otologic effect of aging, results from a loss of hair cells in the organ of Corti. This disorder causes progressive, symmetrical, bilateral sensorineural hearing loss, usually of high-frequency tones.

Minor decreases in hearing are common after age 20. Some deafness due to nerve damage occurs in one of every five people by age 55.

Complications

- Tympanic membrane perforation
- Cholesteatoma
- Permanent hearing loss

Signs and symptoms

PEDIATRIC TIP

Although congenital hearing loss may produce no obvious signs of hearing impairment at birth, a deficient response to auditory stimuli generally becomes apparent within 2 to 3 days. As the child grows older, hearing loss impairs speech development.

Sudden deafness may be conductive, sensorineural, or mixed, depending on etiology. Associated clinical features depend on the underlying cause.

Noise-induced hearing loss causes sensorineural damage, the extent of which depends on the duration and intensity of the noise. Initially, the patient loses perception of certain frequencies (around 4,000 Hz) but, with continued exposure, eventually loses perception of all frequencies.

ELDER TIP

Presbycusis usually produces tinnitus and the inability to understand the spoken word.

PEDIATRIC TIP

The behavior of an infant who's deaf may appear normal and mislead the parents as well as the professional, especially if the infant has autosomal recessive deafness and is the first child of carrier parents.

Diagnosis

CONFIRMING DIAGNOSIS

Patient, family, and occupational histories and a complete audiologic examination usually provide ample evidence of hearing loss and suggest possible causes or predisposing factors.

The Weber, Rinne, and specialized audiologic tests differentiate between conductive and sensorineural hearing loss.

Treatment

After the underlying cause is identified, therapy for congenital hearing loss refractory to surgery consists of developing the patient's ability to communicate through sign language, speech reading, or other effective means. Measures to prevent congenital hearing loss include aggressively immunizing children against rubella to reduce the risk of maternal exposure during pregnancy; educating pregnant women about the dangers of exposure to drugs, chemicals, or infection; and careful monitoring during labor and delivery to prevent fetal anoxia.

Treatment for sudden deafness requires prompt identification of the underlying cause. Prevention necessitates educating patients and health care professionals about the many causes of sudden deafness and the ways to recognize and treat them.

Hyperbilirubinemia can be controlled by phototherapy and exchange transfusions. Children need the appropriate immunizations. Medications that may be ototoxic should be used judiciously in children and monitored closely. Reduction of exposure to loud noises generally prevents high-frequency hearing loss.

In people with noise-induced hearing loss, overnight rest usually restores normal hearing in those who have been exposed to noise levels greater than 90 dB for several hours; but not in those who have been exposed to such noise repeatedly. As hearing deteriorates, treatment must include speech and hearing rehabilitation, because hearing aids are seldom helpful. Prevention

of noise-induced hearing loss requires public recognition of the dangers of noise exposure and insistence on the use, as mandated by law, of protective devices such as earplugs during occupational exposure to noise.

Amplifying sound, as with a hearing aid, helps some patients with presbycusis, but many patients have an intolerance to loud noise and wouldn't be helped by a hearing aid.

Special considerations

- When speaking to a patient with hearing loss who can read lips, stand directly in front of him, with the light on your face, and speak slowly and distinctly. If possible, speak to him at eye level. Approach the patient within his visual range, and elicit his attention by raising your arm or waving; touching him may be unnecessarily startling.
- Make other staff members and facility personnel aware of the patient's disability and his established method of communication. Carefully explain diagnostic tests and facility procedures in a way the patient understands.
- Make sure the patient with a hearing loss is in an area where he can observe unit activities and people approaching because such a patient depends totally on visual clues.
- When addressing an older patient, speak slowly and distinctly in a low tone; avoid shouting.
- Provide emotional support and encouragement to the patient learning to use a hearing aid. Teach him how the aid works and how to maintain it.
- Refer children with suspected hearing loss to an audiologist or otolaryngologist for further evaluation. Any child who fails a language screening examination should be referred to a speech pathologist for language evaluation. The child with a mild language delay may be involved with a home language-enrichment program.

PREVENTION

Watch for signs of hearing impairment in the patient receiving ototoxic drugs. Emphasize the danger of excessive exposure to noise; stress the danger to pregnant women of exposure to drugs, chemicals, and infection (especially rubella); and encourage the use of protective devices in a noisy environment.

Motion sickness

Motion sickness is characterized by loss of equilibrium associated with nausea and vomiting that results from irregular or rhythmic movements or from the sensation of motion. Removal of the stimulus restores

normal equilibrium. Motion sickness also can be induced when patterns of motion differ from what the patient has previously experienced.

Causes and incidence

Motion sickness may result from excessive stimulation of the labyrinthine receptors of the inner ear by certain motions, such as those experienced in a car, boat, plane, or swing. The disorder may also be caused by confusion in the cerebellum from conflicting sensory input—the visual stimulus (a moving horizon) conflicts with labyrinthine perception. Predisposing factors include tension or fear, offensive odors, or sights and sounds associated with a previous attack. Motion sickness from cars, elevators, trains, and swings is most common in children; from boats and airplanes in adults. People who suffer from one kind of motion sickness aren't necessarily susceptible to other types.

Signs and symptoms

Typically, motion sickness induces nausea, vomiting, headache, dizziness, fatigue, diaphoresis and, occasionally, difficulty in breathing, leading to a sensation of suffocation. These symptoms usually subside when the precipitating stimulus is removed, but they may persist for several hours or days.

Treatment

The best way to treat the disorder is to stop the motion that's causing it. If this isn't possible, the patient will benefit from lying down, closing his eyes, and trying to sleep. Antiemetics, such as dimenhydrinate, cyclizine, meclizine, and scopolamine (transdermal

patch), may prevent or relieve motion sickness.

Special considerations

- Tell the patient to avoid exposure to precipitating motion whenever possible.

PEDIATRIC TIP

An elevated car seat may help prevent motion sickness in a child by allowing him to see out the front window.

- Instruct the patient to avoid eating or drinking for at least 4 hours before traveling and to take an antiemetic 30 to 60 minutes before traveling, or to apply a transdermal scopolamine patch at least 4 hours before traveling. Tell the patient with prostate enlargement or glaucoma to consult a physician or pharmacist before taking antiemetics.

PREVENTION

The traveler can minimize motion sickness by sitting where motion is least apparent (near the wing section in an aircraft, in the center of a boat, or in the front seat of an automobile). Instruct him to keep his head still and his eyes closed or focused on a distant and stationary object.

NOSE

Epistaxis

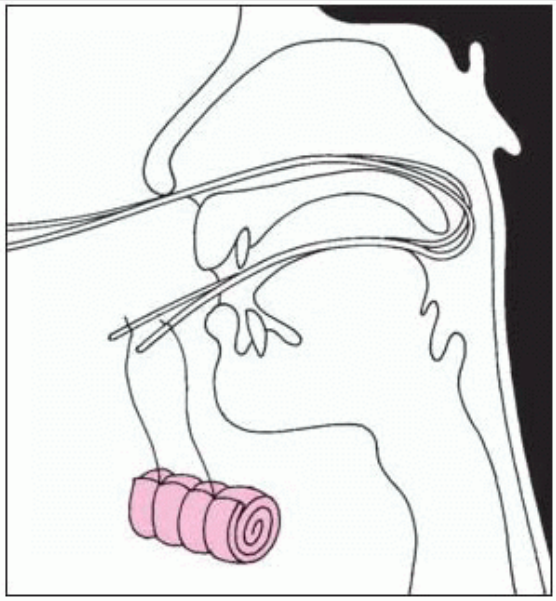
Epistaxis, commonly known as a *nosebleed*, may be a primary disorder or may occur secondary to another condition. Such bleeding in children generally originates in the anterior nasal septum and tends to be mild. In adults, such bleeding is most likely to originate in the posterior septum and can be severe enough to warrent nasal packing. (See *Inserting an anterior-posterior nasal pack*.) Epistaxis is twice as common in children as in adults.

Causes

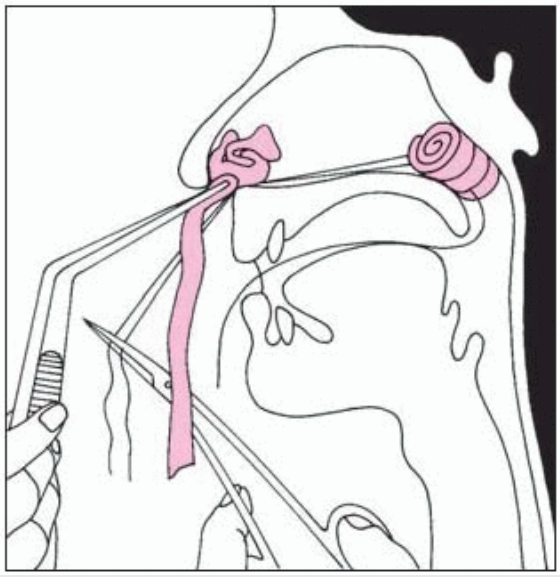
Epistaxis usually follows trauma from external or internal causes: a blow to the nose, nose picking, or insertion of a foreign body; low humidity; or allergies, colds, or sinusitis. Less commonly, it follows polyps; acute or chronic infections such as sinusitis or rhinitis, which cause congestion

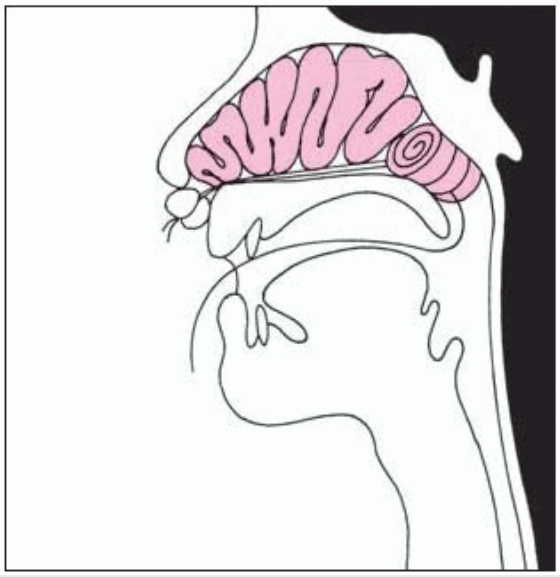
and eventual bleeding of the capillary blood vessels; or inhalation of chemicals that irritate the nasal mucosa.

INSERTING AN ANTERIOR-POSTERIOR NASAL PACK



The first step in the insertion of an anterior-posterior nasal pack is the insertion of catheters into the nostrils. After the catheters are drawn through the mouth, a suture from the pack is tied to each (as shown above).





This positions the pack in place as the catheters are drawn back through the nostrils. While the sutures are held tightly, packing is inserted into the anterior nose (as shown above).

The sutures are then secured around a dental roll; the middle suture extends from the mouth (as shown above) and is taped to the cheek.

Predisposing factors include anticoagulant therapy, hypertension, long-term use of aspirin, high altitudes and dry climates, sclerotic vessel disease, Hodgkin's disease, hereditary hemorrhagic telangiectasia, neoplastic disorders (such as juvenile nasopharyngeal angiofibromas), scurvy, vitamin K deficiency, rheumatic fever, and blood dyscrasias (hemophilia, purpura, leukemia, and anemias).

Complications

- Aspiration
- Shock

Signs and symptoms

Blood oozing from the nostrils usually originates in the anterior nose and is bright red. Blood from the back of the throat originates in the posterior area and may be dark or bright red (commonly mistaken for hemoptysis due to expectoration).

Epistaxis is generally unilateral, except when it's due to dyscrasia or severe trauma. In severe epistaxis, blood may seep behind the nasal septum; it may also appear in the middle ear and in the corners of the eyes.

Associated clinical effects depend on the severity of bleeding. Moderate blood loss may produce light-headedness, dizziness, and slight respiratory difficulty; severe hemorrhage causes hypotension, rapid and bounding pulse, dyspnea, and pallor. Bleeding is considered severe if it persists longer than 10 minutes after pressure is applied and causes

blood loss as great as 1 L/hour in adults. Exsanguination (bleeding to death) from epistaxis is rare.

Diagnosis

CONFIRMING DIAGNOSIS

Although simple observation confirms epistaxis, inspection with a bright light and a nasal speculum is necessary to locate the site of bleeding.

Relevant laboratory values include:

- gradual reduction in hemoglobin levels and hematocrit (HCT; usually inaccurate immediately following epistaxis because of hemoconcentration)
- decreased platelet count in the patient with blood dyscrasia
- prothrombin time and partial thromboplastin time showing a coagulation time twice the control, because of a bleeding disorder or anticoagulant therapy.

Diagnosis must rule out underlying systemic causes of epistaxis, especially disseminated intravascular coagulation and rheumatic fever. Bruises or concomitant bleeding elsewhere probably indicates a hematologic disorder.

PEDIATRIC TIP

Bleeding tests are indicated if any of the following are present:

- *family history of a bleeding disorder*
- *medical history of easy bleeding*
- *spontaneous bleeding at other sites*
- *onset before age 2 or a drop in HCT due to epistaxis.*
- *bleeding that won't clot with direct pressure by the physician*

- *bleeding that lasts longer than 30 minutes.*

Treatment

Mild nosebleeds that occur spontaneously may be treated by gently squeezing the soft portion of the nose between the thumb and finger for 5 to 10 minutes while the patient leans forward slightly (to avoid swallowing the blood) and breathes through his mouth.

For anterior bleeding, treatment consists of application to the bleeding site of a cotton ball saturated with epinephrine, and external pressure, followed by cauterization with electrocautery or a silver nitrate stick. If these measures don't control the bleeding, petroleum gauze nasal packing may be needed.

For posterior bleeding, therapy includes gauze packing inserted through the nose, or postnasal packing inserted through the mouth, depending on the bleeding site. (Gauze packing generally remains in place for 24 to 48 hours; postnasal packing, 3 to 5 days.) An alternate method, the nasal balloon catheter, also controls bleeding effectively. Antibiotics may be appropriate if packing must remain in place for longer than 24 hours. If local measures fail to control bleeding, additional treatment may include supplemental vitamin K and, for severe bleeding, blood transfusions and surgical ligation or embolization of a bleeding artery.

Special considerations

To control epistaxis:

- Elevate the patient's head 45 degrees.
- Continuously compress the soft portion of the nares against the septum for 5 to 10 minutes. Apply an ice collar or cold, wet compresses to the nose. If bleeding continues after 10 minutes of pressure, notify the physician.
- Administer oxygen as needed, and monitor saturation levels.
- Monitor vital signs and skin color; record blood loss.
- Tell the patient to breathe through his mouth and not to swallow blood, talk, or blow his nose.

- Keep vasoconstrictors, such as phenylephrine, handy.
- Reassure the patient and his family that epistaxis usually looks worse than it is.

PREVENTION

- *Instruct the patient not to pick his nose or insert foreign objects into it, and to avoid bending or lifting. Emphasize the need for follow-up examinations and periodic blood studies after an episode of epistaxis. Advise prompt treatment for nasal infection or irritation.*
- *Suggest humidifiers for people who live in dry climates or at high elevations, or whose homes are heated with circulating hot air.*

Septal perforation and deviation

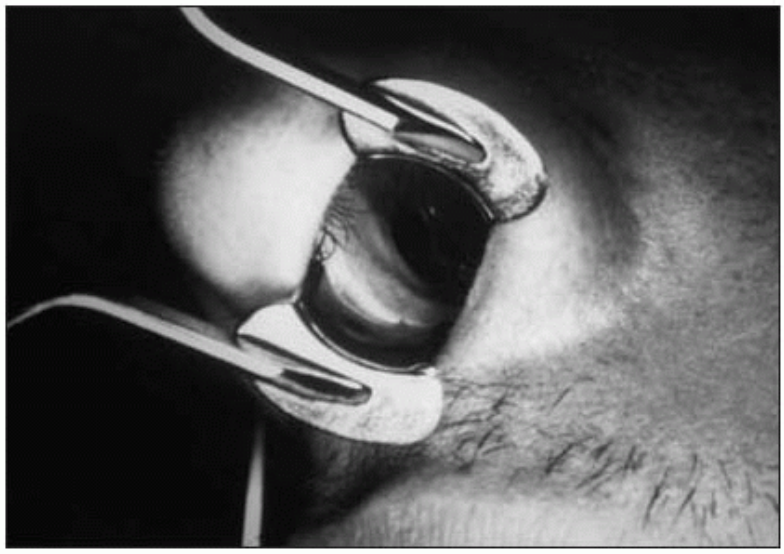
Perforated septum, a hole in the nasal septum between the two air passages, usually occurs in the anterior cartilaginous septum but may occur in the bony septum. Deviated septum, a shift from the midline, is common in most adults. This condition may be severe enough to obstruct the passage of air through the nostrils. With surgical correction, the prognosis for either perforated or deviated septum is good. (See *Septal perforation*.)

Causes and incidence

Generally, perforated septum is caused by traumatic irritation, most commonly resulting from excessive nose picking; less frequently, it results from repeated cauterization for epistaxis or from penetrating septal injury. It may also result from perichondritis, an infection that gradually erodes the perichondrial layer and cartilage, finally forming an ulcer that perforates the septum. Other causes of septal perforation include syphilis, tuberculosis, untreated septal hematoma, inhalation of irritating chemicals, cocaine snorting, chronic nasal infections, nasal carcinoma, granuloma, and chronic sinusitis.

Deviated septum commonly develops during normal growth, as the septum shifts from one side to the other. Consequently, few adults have perfectly straight septa. Nasal trauma resulting from a fall, a blow to the nose, or surgery further exaggerates the deviation. Congenital deviated septum is rare.

SEPTAL PERFORATION



In the photograph at right, the nasal septum shows obvious perforation of the cartilage between the two air passages.

Complications

- Hemorrhage
- Infections
- Deformity

Signs and symptoms

A small septal perforation is usually asymptomatic but may produce a whistle on inspiration. A large perforation causes rhinitis, epistaxis, nasal crusting, and watery discharge.

The patient with a deviated septum may develop a crooked nose, as the midline deflects to one side. The predominant symptom of severe deflection, however, is nasal obstruction. Other manifestations include a sensation of fullness in the face, shortness of breath, stertor (snoring or laborious breathing), nasal discharge, recurring epistaxis, infection, sinusitis, and headache.

Diagnosis

Although clinical features suggest septal perforation or deviation, confirmation requires inspection of the nasal mucosa with a bright light and a nasal speculum.

Treatment

Symptomatic treatment for perforated septum includes decongestants to reduce nasal congestion by local vasoconstriction, local application of lanolin or petroleum jelly to prevent ulceration and crusting, and antibiotics to combat infection. Surgery may be necessary to graft part of the perichondrial layer over the perforation. Also, a plastic or Silastic "button" prosthesis may be used to close the perforation.

Symptomatic treatment for deviated septum usually includes analgesics to relieve headache, decongestants to minimize secretions and, as necessary, vasoconstrictors, nasal packing, or cautery to control hemorrhage. Manipulation of the nasal septum at birth can correct congenital deviated septum.

Corrective surgical procedures include:

- reconstruction of the nasal septum by submucous resection to reposition the nasal septal cartilage and relieve nasal obstruction.
- rhinoplasty to correct nasal structure deformity by intranasal incisions.
- septoplasty to relieve nasal obstruction and enhance cosmetic appearance.

Special considerations

- In the patient with perforated septum, use a cotton applicator to apply petroleum jelly to the nasal mucosa to minimize crusting and ulceration.
- Warn the patient with perforation or severe deviation against blowing his nose. To relieve nasal congestion, instill saline nosedrops and suggest use of a humidifier. Give decongestants as ordered.
- To treat epistaxis, have the patient sit upright, provide an emesis basin, and instruct the patient to expectorate any blood. Compress the outer portion of the nose against the septum for 10 to 15 minutes, and apply ice packs. If bleeding persists, notify the physician.
- If corrective surgery is scheduled, prepare the patient to expect postoperative facial edema, periorbital bruising, and nasal packing, which remains in place for 12 to 24 hours. The patient must breathe through his mouth. After surgery for deviated septum, the patient may also have a splint on his nose.
- To reduce or prevent edema and promote drainage, place the patient in semi-Fowler's position, and use a cool-mist vaporizer to liquefy secretions and facilitate normal breathing. To lessen facial edema and pain, place crushed ice in a rubber glove or a small ice bag, and apply the glove or ice bag intermittently over the eyes and nose for 24 hours.
- Because the patient is breathing through his mouth, provide frequent mouth care.
- Change the mustache dressing or drip pad as needed. Record the color, consistency, and amount of drainage. While nasal packing is in place, expect slight, bright red drainage, with clots. After packing is removed, watch for purulent discharge, an indication of infection.
- Watch for and report excessive swallowing, hematoma, or a falling or flapping septum (depressed, or soft and unstable septum). Intranasal examination is necessary to detect hematoma formation. Any of these complications requires surgical correction.
- Administer sedatives and analgesics as needed. Because of its anticoagulant properties, aspirin is contraindicated after surgery for septal deviation or perforation.

- Nose blowing may cause bruising and swelling even after nasal packing is removed. After surgery, the patient must limit physical activity for 2 or 3 days, and if he's a smoker, he must stop smoking for at least 2 days.
- Instruct the patient to sneeze with his mouth open and to avoid bending over at the waist. (Advise him to stoop to pick up fallen objects.)

Sinusitis

Sinusitis—inflammation of the paranasal sinuses—may be acute, subacute, chronic, allergic, or hyperplastic. Acute sinusitis usually results from the common cold and lingers in subacute form in only about 10% of patients. Chronic sinusitis follows persistent bacterial infection; allergic sinusitis accompanies allergic rhinitis; hyperplastic sinusitis is a combination of purulent acute sinusitis and allergic sinusitis or rhinitis. The prognosis is good for all types.

Causes and incidence

Sinusitis usually results from viral or bacterial infection. The bacteria responsible for acute sinusitis are usually pneumococci, other streptococci, *Haemophilus influenzae,* and *Moraxella catarrhalis*. Staphylococci and gram-negative bacteria are more likely to cause sinusitis in chronic cases or in intensive care patients.

Predisposing factors include any condition that interferes with drainage and ventilation of the sinuses, such as chronic nasal edema, deviated septum, viscous mucus, nasal polyps, allergic rhinitis, nasal intubation, or debilitation due to chemotherapy, malnutrition, diabetes, blood dyscrasias, cystic fibrosis, human immunodeficiency virus or other immunodeficiency disorders, or chronic use of steroids. Bacterial invasion commonly occurs as a result of the conditions listed above or after a viral infection. It may also result from swimming in contaminated water.

Other risk factors for developing sinusitis include a history of asthma, overuse of

nasal decongestants, presence of a foreign body in the nose, frequent

swimming or diving, dental work, pregnancy, changes in altitude (flying or climbing), air pollution and smoke, gastroesophageal reflux disease, and having a deviated nasal septum, nasal bone spur, or polyp.

Each year, more than 30 million adults and children get sinusitis.

PEDIATRIC TIP

The incidence of both acute and chronic sinusitis increases in later childhood. Sinusitis may be more prevalent in children who have had tonsils and adenoids removed.

Complications

- Meningitis
- Cavernous and sinus thrombosis
- Bacteremia or septicemia
- Brain abscess
- Osteomyelitis
- Mucocele
- Orbital cellulitis abscess

Signs and symptoms

The primary indication of acute sinusitis is nasal congestion, followed by a gradual buildup of pressure in the affected sinus. For 24 to 48 hours after onset, nasal discharge may be present and later may become purulent. Associated symptoms include malaise, sore throat, headache, and low-grade fever of 99° to 99.5° F [37.2° to 37.5° C]).

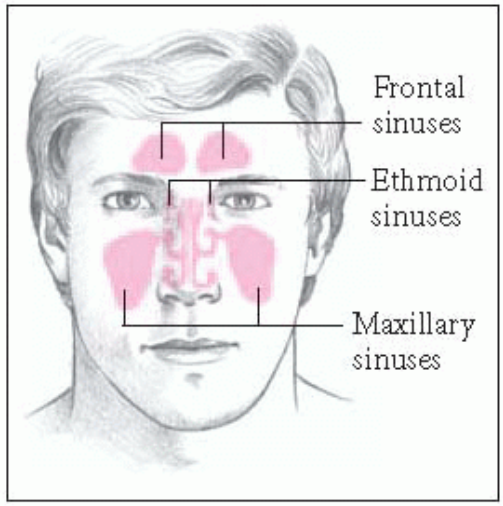
Characteristic pain depends on the affected sinus: maxillary sinusitis causes pain over the cheeks and upper teeth; ethmoid sinusitis, pain over the eyes; frontal sinusitis, pain over the eyebrows; and sphenoid sinusitis (rare), pain behind the eyes. (See *Locating the paranasal sinuses.*)

Purulent nasal drainage that continues for longer than 3 weeks after an acute infection subsides suggests *subacute sinusitis*. Other clinical features of the subacute form include nasal congestion, vague facial discomfort, fatigue, and a nonproductive cough.

The effects of *chronic sinusitis* are similar to those of acute sinusitis, but the chronic form causes continuous mucopurulent discharge.

LOCATING THE PARANASAL SINUSES

The location of a patient's sinusitis pain indicates the affected sinus. For example, an infected maxillary sinus can cause tooth pain. (*Note*: The sphenoid sinus, which lies under the eye and above the soft palate, isn't depicted here.)



The effects of *allergic sinusitis* are the same as those of allergic rhinitis. In both conditions, the prominent symptoms are sneezing, frontal headache, watery nasal discharge, and a stuffy, burning, itchy nose.

In *hyperplastic sinusitis,* bacterial growth on the diseased tissue causes pronounced tissue edema; thickening of the mucosal lining and the development of mucosal polyps combine to produce chronic stuffiness of the nose, in addition to headaches.

Diagnosis

The following measures are useful:

- Antral puncture promotes drainage of purulent material. It may also be used to provide a specimen for culture and sensitivity testing of the infecting organism, but it's seldom performed.
- Nasal examination reveals inflammation and pus.
- Sinus X-rays reveal cloudiness in the affected sinus, air and fluid, and any thickening of the mucosal lining.

- Transillumination is a simple diagnostic tool that involves shining a light into the patient's mouth with his lips closed around it. Infected sinuses look dark and normal sinuses transilluminate.
- Ultrasound, computed tomography scan, magnetic resonance imaging, and X-rays aid in diagnosing suspected complications.

Treatment

Local decongestants usually are tried before systemic decongestants; steam inhalation may also be helpful. Antibiotics are necessary to combat purulent or persistent infection. Amoxicillin and amoxicillin/clavulanate potassium are usually the antibiotics of choice. Other possible therapy includes cefixime for responsive infections or if beta-lactamase-producing bacteria are present. Because sinusitis is a deep-seated infection, antibiotics should be given for 10 days to 2 weeks, with the exception of azithromycin, which is given for 5 days. Local applications of heat may help to relieve pain and congestion. In subacute sinusitis, antibiotics and decongestants may be helpful.

Treatment for allergic sinusitis must include treatment for allergic rhinitis— administration of antihistamines, identification of allergens by skin testing, and desensitization by immunotherapy. Severe allergic symptoms may require treatment with corticosteroids and epinephrine.

In both chronic sinusitis and hyperplastic sinusitis, using antihistamines, antibiotics, and a steroid nasal spray may relieve pain and congestion. If subacute infection persists, the sinuses may be irrigated. If irrigation fails to relieve symptoms, endoscopic sinus surgery may be required to obtain a histologic diagnosis, remove polyps, and provide adequate ventilation of the infected sinuses. Partial or total resection of the

middle turbinate as well as more radical procedures, such as total sphenoethmoidectomy, may be performed.

Special considerations

- Enforce bed rest, and encourage the patient to drink plenty of fluids to promote drainage. Don't elevate the head of the bed more than 30 degrees.
- To relieve pain and promote drainage, apply warm compresses continuously, or four times daily for 2-hour intervals. Also, give analgesics and antihistamines as needed.
- Watch for and report complications, such as vomiting, chills, fever, edema of the forehead or eyelids, blurred or double vision, and personality changes.
- If surgery is necessary, tell the patient what to expect postoperatively: nasal packing will be in place for 12 to 24 hours following surgery; he'll have to breathe through his mouth and won't be able to blow his nose. After surgery, monitor for excessive drainage or bleeding and watch for complications.
- To prevent edema and promote drainage, place the patient in semi-Fowler's position. To relieve edema and pain and to minimize bleeding, apply ice compresses or a rubber glove filled with ice chips over the nose, and iced saline gauze over the eyes. Continue these measures for 24 hours.
- Frequently change the mustache dressing or drip pad, and record the consistency, amount, and color of drainage (expect scant, bright red, and clotty drainage).
- Because the patient will be breathing through his mouth, provide meticulous mouth care.
- Tell the patient that even after the packing is removed, nose blowing may cause bleeding and swelling. If the patient is a smoker, instruct him not to smoke for at least 2 or 3 days after surgery.
- Tell the patient to finish the prescribed antibiotics, even if his symptoms disappear.

Nasal polyps

Benign and edematous growths, nasal polyps are usually multiple, mobile, and bilateral. Nasal polyps may become large and numerous enough to cause nasal distention and enlargement of the bony framework, possibly occluding the airway.

Causes and incidence

Nasal polyps are usually produced by the continuous pressure resulting from a chronic allergy that causes prolonged mucous membrane edema in the nose and sinuses. Other predisposing factors include chronic sinusitis, chronic rhinitis, and recurrent nasal infections.

Nasal polyps are more common in adults than in children and tend to recur. They're also commonly seen in patients with long-term allergic rhinitis. About 1 in 4 people with cystic fibrosis have nasal polyps.

Complication

- Airway obstruction

Signs and symptoms

Nasal obstruction is the primary indication of nasal polyps. Such obstruction causes anosmia, a sensation of fullness in the face, nasal discharge, headache, and shortness of breath. Associated clinical features are usually the same as those of allergic rhinitis.

Diagnosis

Diagnosis of nasal polyps is aided by the following tests.

- Examination with a nasal speculum shows a dry, red surface, with clear or gray growths. Large growths may resemble tumors.
- X-rays of sinuses and nasal passages reveal soft tissue shadows over the affected areas.

PEDIATRIC TIP

Nasal polyps in children require further testing to rule out cystic fibrosis and Peutz-Jeghers syndrome.

Treatment

Generally, treatment consists of corticosteroids (either by direct injection into the polyps or by local spray) to temporarily reduce the polyp. A short course of oral corticosteroids (such as prednisone) may be beneficial. Treatment for the underlying cause may include antihistamines to control allergy, and antibiotic therapy if infection is present. Local application of an astringent shrinks hypertrophied tissue. However, medical management alone is seldom effective.

Consequently, the treatment of choice is polypectomy, which is usually performed under a local anesthetic. The use of surgical lasers is becoming more popular. Continued recurrence may require surgical opening of the ethmoid, sphenoid, and maxillary sinuses and evacuation of diseased tissue.

Special considerations

- Administer antihistamines, as ordered, for the patient with allergies. Prepare the patient for scheduled surgery by telling him what to expect postoperatively, such as nasal packing for 1 to 2 days after surgery.

After surgery:

- Watch for excessive bleeding or other drainage, and promote patient comfort.
- Elevate the head of the bed to facilitate breathing, reduce swelling, and promote adequate drainage. Change the mustache dressing or drip pad, as needed, and record the consistency, amount, and color of nasal drainage.
- Intermittently apply ice compresses over the nostrils to lessen swelling, prevent bleeding, and relieve pain.
- If nasal bleeding occurs—most likely after packing is removed—sit the patient upright, monitor his vital signs, and advise him not to swallow

blood. Compress the outside of his nose against the septum for 10 to 15 minutes. If bleeding persists, nasal packing may be necessary.

PREVENTION

Instruct patients with allergies to avoid exposure to allergens and to take antihistamines at the first sign of an allergic reaction. Also, advise them to avoid overuse of nose drops and sprays.

Nasal papillomas

A papilloma is a benign epithelial tissue overgrowth within the intranasal mucosa. Inverted papillomas grow into the underlying tissue, usually at the junction of the antrum and the maxillary sinus; they generally occur singly but sometimes are associated

with squamous cell cancer. Exophytic papillomas, which also tend to occur singly, arise from epithelial tissue, commonly on the surface of the nasal septum.

Causes and incidence

A papilloma may arise as a benign precursor of a neoplasm or as a response to tissue injury or viral infection, but its cause is unknown. Both types of papillomas are most prevalent in males. Recurrence is common, even after surgical excision.

Complications

- Severe respiratory distress (rare)
- Nasal drainage
- Infection

Signs and symptoms

Both inverted and exophytic papillomas typically produce symptoms related to unilateral nasal obstruction—congestion, postnasal drip, headache, shortness of breath, dyspnea and, rarely, severe respiratory

distress, nasal drainage, and infection. Epistaxis is most likely to occur with exophytic papillomas. Occasionally hemorrhage may be the presenting symptom.

Diagnosis

On examination of the nasal mucosa, inverted papillomas usually appear large, bulky, highly vascular, and edematous; color varies from dark red to gray; and consistency, from firm to friable. Exophytic papillomas are usually raised, firm, and rubbery; pink to gray; and securely attached by a broad or pedunculated base to the mucous membrane.

PEDIATRIC TIP

Juvenile angiofibroma is a benign vascular tumor that arises in the nasopharynx and occurs most commonly in adolescent males. Nasal obstruction and hemorrhage may occur as with nasal papillomas. Any adolescent male who continues to have recurrent episodes of epistaxis should be assessed for juvenile angiofibroma. Medical management involves surgical excision, with preoperative embolization to reduce bleeding.

CONFIRMING DIAGNOSIS

Tissue biopsy followed by histologic examination of excised tissue confirms the diagnosis.

Treatment

The most effective treatment is wide surgical excision or diathermy, with careful inspection of adjacent tissues and sinuses to rule out extension. The use of surgical lasers is becoming more popular. Ibuprofen or acetaminophen and decongestants may relieve symptoms.

Special considerations

- If bleeding occurs, have the patient sit upright, and instruct him to expectorate blood into an emesis basin. Compress both sides of his nose against the septum for 10 to 15 minutes, and apply ice

compresses to the nose. If the bleeding doesn't stop, notify the physician.

ALERT

Check for airway obstruction. Place your hand under the patient's nostrils to assess air exchange, and watch for signs of mild shortness of breath.

- If surgery is scheduled, tell the patient what to expect postoperatively. Instruct him not to blow his nose. (Packing is usually removed 12 to 24 hours after surgery.)
- Postoperatively, monitor vital signs and respiratory status. Use pulse oximetry to monitor oxygen saturation levels. As needed, administer analgesics and facilitate breathing with a cool-mist vaporizer. Provide mouth care.
- Frequently change the mustache dressing or drip pad, to ensure proper absorption of drainage. Record the type and amount of drainage. While the nasal packing is in place, expect scant, usually bright red, clotted drainage. Remember that the amount of drainage typically increases for a few hours after the packing is removed.
- Because papillomas tend to recur, tell the patient to seek medical attention at the first sign of nasal discomfort, discharge, or congestion that doesn't subside with conservative treatment.
- Encourage regular follow-up visits to detect early signs of recurrence.

Adenoid hyperplasia

A fairly common childhood condition, adenoid hyperplasia (also known as *adenoid hypertrophy*) is enlargement of the lymphoid tissue of the nasopharynx. Normally, adenoidal tissue is small at birth (¾" to 1¼" [2 to 3 cm]), grows until the child reaches adolescence, and then begins to slowly atrophy. In adenoid hyperplasia, however, this tissue continues to grow. Enlarged adenoids commonly accompany tonsillitis.

Causes and incidence

The cause of adenoid hyperplasia is unknown, but contributing factors may include heredity, chronic infection, chronic nasal congestion, persistent allergy, insufficient aeration, and inefficient nasal breathing. Inflammation resulting from repeated infection increases the patient's risk of respiratory obstruction.

Complications

- Otitis media
- Conductive hearing loss
- Sinusitis
- Cor pulmonale
- Pulmonary arterial hypertension

Signs and symptoms

Typically, adenoid hyperplasia produces symptoms of respiratory obstruction, especially mouth breathing, snoring at night, and frequent, prolonged nasal congestion. Persistent mouth breathing during the formative years produces voice alteration and distinctive changes in facial features—a slightly elongated face, open mouth, highly arched palate, shortened upper lip, and vacant expression.

PEDIATRIC TIP

Occasionally, the child is incapable of mouth breathing, snores loudly at night, and may eventually show effects of nocturnal respiratory insufficiency (sleep apnea), such as intercostal retractions and nasal flaring.

Diagnosis

CONFIRMING DIAGNOSIS

Nasopharyngoscopy or rhinoscopy confirms adenoid hyperplasia by allowing visualization of abnormal tissue. Lateral pharyngeal X-rays show an obliterated nasopharyngeal air column.

Treatment

Adenoidectomy is the treatment of choice for adenoid hyperplasia and is commonly recommended for the patient with prolonged mouth breathing, nasal speech, adenoid facies, recurrent otitis media, constant nasopharyngitis, and nocturnal respiratory distress. This procedure usually eliminates recurrent nasal infections and ear complications and reverses any secondary hearing loss.

Special considerations

Care requires sympathetic preoperative care and diligent postoperative monitoring.

Before surgery, do the following.

- Describe the facility routine, and arrange for the patient and his parents to tour relevant areas.
- Explain adenoidectomy to the child, using illustrations if necessary, and detail the recovery process. Advise him that he'll probably need to be hospitalized. If facility protocol allows, encourage one parent to stay with the child and participate in his care.

After surgery, take these steps.

ALERT

Maintain a patent airway. Position the child on his side, with his head down, to prevent aspiration of draining secretions. Frequently check the throat for bleeding. Be alert for vomiting of old, partially digested blood (coffee-ground vomitus). Closely monitor vital signs, and report excessive bleeding, rise in pulse rate, drop in blood pressure, tachypnea, and restlessness.

- If no bleeding occurs, offer cracked ice or water when the patient is fully awake.
- Tell the parents that their child may temporarily have a nasal voice.

Velopharyngeal insufficiency

Velopharyngeal insufficiency results from failure of the velopharyngeal sphincter to close properly during speech, giving the voice a hypernasal quality and permitting nasal emission (air escape during pronunciation of consonants).

Causes and incidence

Velopharyngeal insufficiency can result from an inherited palate abnormality, or it can be acquired from tonsillectomy, adenoidectomy, or palatal paresis. It commonly occurs in people who undergo cleft palate surgery and those with submucous cleft palates. Middle ear disease and hearing loss frequently accompany this disorder.

Complication

- Airway obstruction

Signs and symptoms

Generally, this condition causes unintelligible speech, marked by hypernasality, nasal emission, poor consonant definition, and a weak voice. The patient experiences dysphagia and, if velopharyngeal insufficiency is severe, he may regurgitate through the nose.

Diagnosis

Fiber-optic nasopharyngoscopy, which permits monitoring of velopharyngeal patency during speech, suggests this diagnosis. Ultrasound scanning, which shows air-tissue overlap, reflects the degree of velopharyngeal sphincter incompetence (an opening greater than 20 mm2 results in unintelligible speech). Videofluoroscopy simultaneously records the movement of the velopharyngeal sphincter and the patient's speech.

Treatment

Treatment consists of corrective surgery, usually at age 6 or 7. The preferred surgical method is the pharyngeal flap procedure, which diverts a tissue flap from the pharynx to the soft palate. Children with velopharyngeal insufficiency shouldn't have adenoidectomy except in cases of life-threatening obstruction.

Other appropriate surgical procedures include:

- augmentation pharyngoplasty, which narrows the velopharyngeal opening by enlarging the pharyngeal wall with a retropharyngeal implant
- palatal push-back, which separates the hard and soft palates to allow insertion of an obturator, thus lengthening the soft palate
- pharyngoplasty, which rotates pharyngeal flaps to lengthen the soft palate and narrow the pharynx
- velopharyngeal sphincter reconstruction, which uses free muscle implantation to reconstruct the sphincter.

Surgery eliminates hypernasality and nasal emission, but speech abnormalities persist and usually necessitate speech therapy. Immediate postoperative therapy includes antibiotics and a clear, liquid diet for the first 3 days, followed by a soft diet for 2 weeks.

Special considerations

- After surgery for velopharyngeal insufficiency, maintain a patent airway (nasopharynx edema may obstruct the airway). Position the patient on his side, and suction the dependent side of his mouth, avoiding the pharynx.
- Control postoperative agitation, which may provoke pharyngeal bleeding, with sedation, as ordered.
- Administer high-humidity oxygen as ordered.
- Monitor vital signs frequently, and report any changes immediately. Observe for bleeding from the mouth or nose. Check intake and output, and watch for signs of dehydration.
- Advise the patient that preoperative and postoperative speech therapy require time and effort on his part, but with persistence and

practice, his speech will improve. Before discharge, emphasize the importance of completing the prescribed antibiotic therapy.

THROAT

Pharyngitis

The most common throat disorder, pharyngitis is an acute or chronic inflammation of the pharynx. It frequently accompanies the common cold.

Causes and incidence

Pharyngitis is usually caused by a virus. The most common bacterial cause is group A beta-hemolytic streptococci. Other common causes include *Mycoplasma* and *Chlamydia*. In up to 30% of cases, no organism is identified.

Pharyngitis is widespread among adults who live or work in dusty or very dry environments, use their voices excessively, habitually use tobacco or alcohol, or suffer from chronic sinusitis, persistent coughs, or allergies.

Complications

- Otitis media
- Sinusitis
- Mastoiditis
- Rheumatic fever
- Nephritis

Signs and symptoms

Pharyngitis produces a sore throat and slight difficulty in swallowing. Swallowing saliva is usually more painful than swallowing food. Pharyngitis may also cause the sensation of a lump in the throat as well as a constant, aggravating urge to swallow. Associated features may

include mild fever, headache, muscle and joint pain, coryza, and rhinorrhea. Uncomplicated pharyngitis usually subsides in 3 to 10 days.

PEDIATRIC TIP

More than 90% of cases of sore throat and fever in children are of viral origin. Associated symptoms usually include runny nose and nonproductive cough.

Diagnosis

Physical examination of the pharynx reveals generalized redness and inflammation of the posterior wall, and red, edematous mucous membranes studded with white or yellow follicles. Exudate is usually confined to the lymphoid areas of the throat, sparing the tonsillar pillars. Bacterial pharyngitis usually produces a large amount of exudate.

A throat culture may be performed to identify bacterial organisms that may be the cause of the inflammation.

Treatment

Treatment for acute viral pharyngitis is usually symptomatic and consists mainly of rest, warm saline gargles, throat lozenges containing a mild anesthetic, plenty of fluids, and analgesics as needed. If the patient can't swallow fluids, I.V. hydration may be required.

Suspected bacterial pharyngitis requires rigorous treatment with penicillin or another broad-spectrum antibiotic because *Streptococcus* is the chief infecting organism. Antibiotic therapy should continue for 48 hours until culture results are back. If the culture (or a rapid strep test) is positive for group A beta-hemolytic streptococci, or if bacterial infection is suspected despite negative culture results, penicillin therapy should be continued for 10 days. This is to prevent the sequelae of acute rheumatic fever.

Chronic pharyngitis requires the same supportive measures as acute pharyngitis but with greater emphasis on eliminating the underlying cause, such as an allergen. Preventive measures include adequate

humidification and avoiding excessive exposure to air conditioning. In addition, the patient should be urged to stop smoking.

Special considerations

- Administer analgesics and warm saline gargles, as ordered and as appropriate.
- Encourage the patient to drink plenty of fluids. Scrupulously monitor intake and output, and watch for signs of dehydration.
- Provide meticulous mouth care to prevent dry lips and oral pyoderma, and maintain a restful environment.
- Obtain throat cultures, and administer antibiotics as needed. If the patient has acute bacterial pharyngitis, emphasize the importance of completing the full course of antibiotic therapy.
- Teach the patient with chronic pharyngitis how to minimize sources of throat irritation in the environment such as by using a bedside humidifier.
- Refer the patient to a self-help group to stop smoking if appropriate.
- Children attending school should receive at least 24 hours of therapy before being allowed to return to school.
- If the patient has exhibited three or more documented bacterial infections within 6 months, consider daily penicillin prophylaxis during the winter months. Also, consider treatment of carriers who live in closed or semiclosed communities.

Tonsillitis

Tonsillitis—inflammation of the tonsils— can be acute or chronic. The uncomplicated acute form usually lasts 4 to 6 days. The presence of proven chronic tonsillitis justifies tonsillectomy, the only effective treatment. Tonsils tend to hypertrophy during childhood and atrophy after puberty.

Causes and incidence

Tonsillitis generally results from infection with group A beta-hemolytic streptococci but can result from other bacteria or viruses or from oral anaerobes. It commonly affects children between ages 5 and 10.

Complications

- Chronic upper airway obstruction
- Sleep apnea
- Cor pulmonale
- Failure to thrive
- Eating or swallowing disorders
- Febrile seizures
- Otitis media
- Cardiac valvular disease
- Peritonsillar abscesses
- Bacterial endocarditis
- Cervical lymph node abscesses

Signs and symptoms

Acute tonsillitis commonly begins with a mild to severe sore throat. A very young child, unable to describe a sore throat, may stop eating. Tonsillitis may also produce dysphagia, fever, swelling and tenderness of the lymph glands in the submandibular area, muscle and joint pain, chills, malaise, headache, and pain (frequently referred to the ears). Excess secretions may elicit the complaint of a constant urge to swallow; the back of the throat may feel constricted. Such discomfort usually subsides after 72 hours.

Chronic tonsillitis produces a recurrent sore throat and purulent drainage in the tonsillar crypts. Frequent attacks of acute tonsillitis may also occur. Complications include obstruction from tonsillar hypertrophy and peritonsillar abscess.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnostic confirmation requires a thorough throat examination that reveals:

- *generalized inflammation of the pharyngeal wall*
- *swollen tonsils that project from between the pillars of the fauces and exude white or yellow follicles*
- *purulent drainage when pressure is applied to the tonsillar pillars*
- *possible edematous and inflamed uvula.*

Culture may determine the infecting organism and indicate appropriate antibiotic therapy. Leukocytosis is also usually present. Differential diagnosis rules out infectious mononucleosis and diphtheria.

Treatment

Treatment for acute tonsillitis requires rest, adequate fluid intake, administration of ibuprofen or acetaminophen and, for bacterial infection, antibiotics. When the causative organism is group A beta-hemolytic streptococcus, penicillin is the drug of choice (another broad-spectrum antibiotic may be substituted). Most oral anaerobes also respond to penicillin. To prevent complications, antibiotic therapy should continue for 10 to 14 days.

Chronic tonsillitis or the development of complications (obstructions from tonsillar hypertrophy, peritonsillar abscess) may require a tonsillectomy, but only after the patient has been free from tonsillar or respiratory tract infections for 3 to 4 weeks.

Special considerations

- Despite dysphagia, urge the patient to drink plenty of fluids, especially if he has a fever. Offer a child ice cream and flavored drinks and ices. Suggest gargling with warm salt water to soothe the throat, unless it exacerbates pain. Make sure the patient and his parents

understand the importance of completing the prescribed course of antibiotic therapy.

- Before tonsillectomy, explain to the adult patient that a local anesthetic prevents pain but allows a sensation of pressure during surgery. Warn the patient to expect considerable throat discomfort and some bleeding postoperatively. Watch for continuous swallowing, a sign of heavy bleeding.

PEDIATRIC TIP

For the pediatric patient, keep your explanation simple and nonthreatening. Show him the operating and recovery areas, and briefly explain the facility routine.Most facilities allow one parent to stay with the child.

- Postoperatively, maintain a patent airway. To prevent aspiration, place the patient on his side. Monitor vital signs frequently, and check for bleeding. Immediately report excessive bleeding, increased pulse rate, or dropping blood pressure. After he's fully alert and the gag reflex has returned, allow him to drink water. Later, urge him to drink plenty of nonirritating fluids, to ambulate, and to take frequent deep breaths to prevent pulmonary complications. Give pain medication as needed.
- Before discharge, provide the patient or his parents with written instructions on home care. Tell them to expect a white scab to form in the throat between 5 and 10 days postoperatively, and to report bleeding, ear discomfort, or a fever that lasts longer than 3 days.

Throat abscesses

Throat abscesses may be peritonsillar (quinsy) or retropharyngeal. Peritonsillar abscesses form in the connective tissue space between the tonsil capsule and the constrictor muscle of the pharynx. Retropharyngeal abscesses, or abscesses of the potential space, form between the posterior pharyngeal wall and the prevertebral fascia. With treatment, the prognosis for both types of abscesses is good.

Causes and incidence

Peritonsillar abscess is a complication of acute tonsillitis, usually after streptococcal or staphylococcal infection. It occurs more commonly in adolescents and young adults than in children.

Acute retropharyngeal abscess results from infection in the retropharyngeal lymph glands, which may follow an upper respiratory tract bacterial infection. Most common pathogens are beta-hemolytic *Streptococcus* and *Staphylococcus aureus*. These lymph glands begin to atrophy after age 2. Acute retropharyngeal abscess most commonly affects infants and children younger than age 2.

Chronic retropharyngeal abscess may result from tuberculosis of the cervical spine (Pott's disease) and may occur at any age.

Complications

- Airway obstruction
- Cellulitis
- Endocarditis
- Pericarditis
- Pleural effusion
- Pneumonia

Signs and symptoms

Key symptoms of peritonsillar abscess include severe throat pain, occasional ear pain on the same side as the abscess, and tenderness of the submandibular gland. Dysphagia causes drooling. Trismus may occur as a result of the spread of edema and infection from the peritonsillar space to the pterygoid muscles. Other effects include fever, chills, malaise, rancid breath, nausea, muffled speech, dehydration, cervical adenopathy, and localized or systemic sepsis.

Clinical features of retropharyngeal abscess include pain, dysphagia, fever and, when the abscess is located in the upper pharynx, nasal obstruction; with a lowpositioned abscess, dyspnea, progressive

inspiratory stridor (from laryngeal obstruction), neck hyperextension and, in

children, drooling and muffled crying occur. Other symptoms in children may include gurgling respirations, dyspnea and dysphagia, respiratory symptoms, and fever. A very large abscess may press on the larynx, causing edema, or may erode into major vessels, causing sudden death from asphyxia or aspiration.

Diagnosis

Diagnosis of peritonsillar abscess usually begins with a patient history of bacterial pharyngitis. Examination of the throat shows swelling of the soft palate on the abscessed side, with displacement of the uvula to the opposite side; red, edematous mucous membranes; and tonsil displacement toward the midline. Culture may reveal streptococcal or staphylococcal infection.

Diagnosis of retropharyngeal abscess is based on patient history of nasopharyngitis or pharyngitis and on physical examination revealing a soft, red bulging of the posterior pharyngeal wall. X-rays show the larynx pushed forward and a widened space between the posterior pharyngeal wall and vertebrae. If neck pain or stiffness occurs, look for extension to the epidural space or the cervical vertebrae. Culture and sensitivity tests isolate the causative organism and reveal the appropriate antibiotic.

Treatment

For early-stage peritonsillar abscess, large doses of penicillin or another broad-spectrum antibiotic are necessary. If the patient is immunocompromised or has been repeatedly hospitalized, antibiotic therapy should include coverage for staphylococci and gram-negative organisms. For late-stage abscess, with cellulitis of the tonsillar space, primary treatment is usually incision and drainage under a local anesthetic, followed by antibiotic therapy for 7 to 10 days. Tonsillectomy, scheduled no sooner than 1 month after healing, prevents recurrence but is recommended only after several episodes.

In acute retropharyngeal abscess, the primary treatment is incision and drainage through the pharyngeal wall. It's considered a surgical emergency. In chronic retropharyngeal abscess, drainage is performed through an external incision behind the sternomastoid muscle. During incision and drainage, strong, continuous mouth suction is necessary to prevent aspiration of pus, and the head should be kept down. Postoperative drug therapy includes I.V. antibiotics (usually penicillin or clindamycin) and analgesics.

Special considerations

ALERT

Be alert for signs of respiratory obstruction (inspiratory stridor, dyspnea, retractions and nasal flaring, increasing restlessness, and cyanosis). Keep emergency airway equipment nearby.

- Explain the drainage procedure to the patient and his parents. Because the procedure is usually done under local anesthesia, the patient may be apprehensive.
- Assist with incision and drainage. To allow easy expectoration and suction of pus and blood, place the patient in a semirecumbent or sitting position.

After incision and drainage:

- Give antibiotics, analgesics, and antipyretics, as ordered. Stress the importance of completing the full course of prescribed antibiotic therapy.
- Monitor vital signs, and report significant changes or bleeding. Assess pain, and treat accordingly.
- If the patient is unable to swallow, ensure adequate hydration with I.V. therapy. Monitor fluid intake and output, and watch for dehydration.
- Provide meticulous mouth care. Apply petroleum jelly to the patient's lips. Promote healing with warm saline gargles or throat irrigations for 24 to 36 hours after incision and drainage. Encourage adequate rest.

PREVENTION

Encourage early treatment of tonsillitis.

Vocal cord paralysis

Vocal cord paralysis results from disease of or injury to the superior or, most commonly, the recurrent laryngeal nerve. It may also be congenital.

Causes and incidence

Vocal cord paralysis commonly results from the accidental severing of the recurrent laryngeal nerve, or of one of its extralaryngeal branches, during thyroidectomy. Other causes include pressure from a thoracic aortic aneurysm or from an enlarged atrium (in patients with mitral stenosis), bronchial or esophageal carcinoma, hypertrophy of the thyroid gland, trauma (such as neck injuries) and intubation, and neuritis due to infections or metallic poisoning. Vocal cord paralysis can also result from hysteria and, rarely, lesions of the central nervous system.

Complications

- Airway obstruction
- Respiratory failure

Signs and symptoms

Unilateral paralysis, the most common form, may cause vocal weakness and hoarseness. Bilateral paralysis typically produces vocal weakness and incapacitating airway obstruction if the cords become paralyzed in the adducted position.

PEDIATRIC TIP

Children may present with hoarseness, aspiration, and stridor. If the paralysis is unilateral, it typically involves the left recurrent laryngeal nerve. In unilateral paralysis, airway intervention involving intubation and

tracheostomy is rarely indicated; it's usually required if the paralysis is bilateral.

Diagnosis

The patient history and characteristic features suggest vocal cord paralysis.

CONFIRMING DIAGNOSIS

Visualization by indirect laryngoscopy shows one or both cords fixed in an adducted or partially abducted position and confirms the diagnosis.

X-ray or computed tomography scan detect abnormalities in the mediastinum that may be responsible for the injury.

Treatment

Treatment for unilateral vocal cord paralysis consists of injection of Teflon into the paralyzed cord, under direct laryngoscopy. This procedure enlarges the cord and brings it closer to the other cord, which usually strengthens the voice and protects the airway from aspiration. Thyroplasty also serves to reposition the vocal cord, but in this procedure an implant is placed through a neck incision. The ansa cervicalis nerve transfer allows for reinnervation of the muscles of the vocal cord. Bilateral cord paralysis in an adducted position necessitates a tracheostomy.

Alternative treatments for adults include endoscopic arytenoidectomy to open the glottis, and lateral fixation of the arytenoid cartilage through an external neck incision. Excision or fixation of the arytenoid cartilage improves airway patency but produces residual voice impairment.

Treatment for hysterical aphonia may include psychotherapy and hypnosis.

Special considerations

If the patient chooses direct laryngoscopy and Teflon injection, explain these procedures thoroughly. Tell him these measures will improve his

voice but won't restore it to normal. Patients are sometimes placed on voice rest for 24 to 48 hours to reduce stress on the vocal cords, which would increase the edema and might lead to airway obstruction.

Many patients with bilateral cord paralysis prefer to keep a tracheostomy instead of having an arytenoidectomy; voice quality is generally better with a tracheostomy alone than after corrective surgery.

If the patient is scheduled to undergo a tracheostomy:

- Explain the procedure thoroughly, and offer reassurance. Because the procedure is performed under a local anesthetic, the patient may be apprehensive.
- Teach the patient how to suction, clean, and change the tracheostomy tube.
- Reassure the patient that he can still speak by covering the lumen of the tracheostomy tube with his finger or a tracheostomy plug.

If the patient elects to have an arytenoidectomy, explain the procedure thoroughly. Advise the patient that the tracheostomy will remain in place until the edema has subsided and the airway is patent.

Vocal cord nodules and polyps

Vocal cord nodules result from hypertrophy of fibrous tissue and form at the point where the cords come together forcibly. Vocal cord polyps are chronic, subepithelial, edematous masses. Both nodules and polyps have a good prognosis unless continued voice abuse causes recurrence, with subsequent scarring and permanent hoarseness.

Causes and incidence

Vocal cord nodules and polyps usually result from voice abuse, especially in the presence of infection. Consequently, they're most common in teachers, singers, and sports fans, and in energetic children (ages 8 to 12) who continually shout while playing. Polyps are common in adults who smoke, live in dry climates, or have allergies.

PEDIATRIC TIP

In children, papillomas of the larynx (benign warty growths) are the most common laryngeal neoplasm. Suspected causes include human papillomavirus types 6, 11, and 16. The virus may be acquired during birth because many mothers have a history of condylomata acuminata at the time of delivery.

Complication

- Permanent hoarseness

Signs and symptoms

Nodules and polyps inhibit the approximation of vocal cords and produce painless hoarseness. The voice may also develop a breathy or husky quality.

Diagnosis

Persistent hoarseness suggests vocal cord nodules and polyps; visualization by indirect laryngoscopy confirms it. In the patient with vocal cord nodules, laryngoscopy initially shows small red nodes; later, white solid nodes on one or both cords. (See *Vocal cord nodules*.) In the patient with polyps, laryngoscopy reveals unilateral or, occasionally, bilateral, sessile or pedunculated polyps of varying size, anywhere on the vocal cords.

Treatment

Conservative management of small vocal cord nodules and polyps includes humidification, speech therapy (voice rest, training to reduce the intensity and duration of voice production), and treatment for any underlying allergies.

When conservative treatment fails to relieve hoarseness, nodules or polyps require removal under direct laryngoscopy. Microlaryngoscopy may be done for small lesions, to avoid injuring the vocal cord surface. If nodules or polyps are bilateral, excision may be performed in two stages: one cord is allowed to heal before excision of polyps on the

other cord. Two-stage excision prevents laryngeal web, which occurs when epithelial tissue is removed from adjacent cord surfaces, and these surfaces grow together.

PEDIATRIC TIP

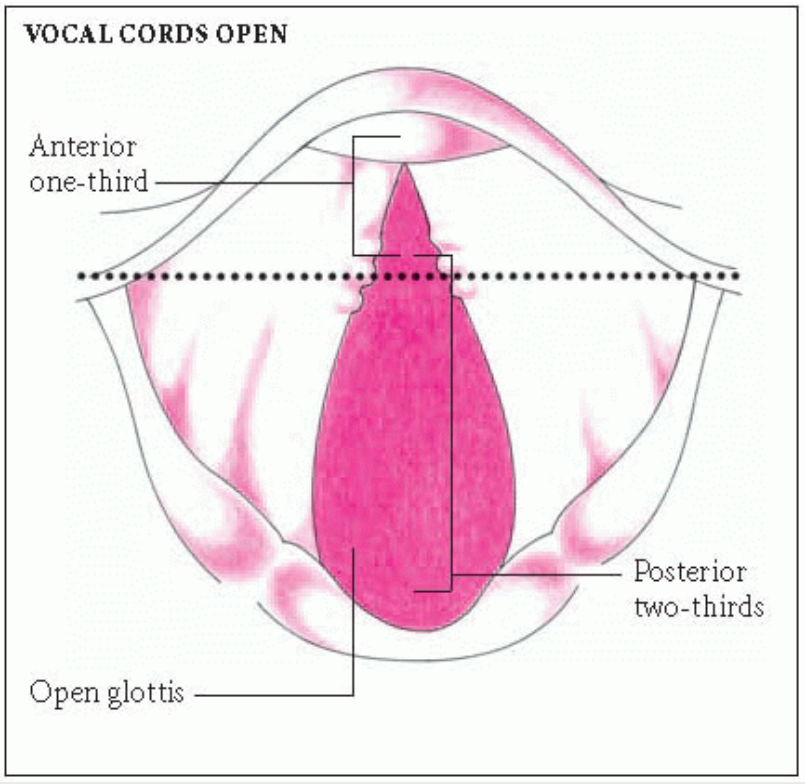
For children, treatment consists of speech therapy. If possible, surgery should be delayed until the child is old enough to benefit from voice training, or until he can understand the need to abstain from voice abuse.

Special considerations

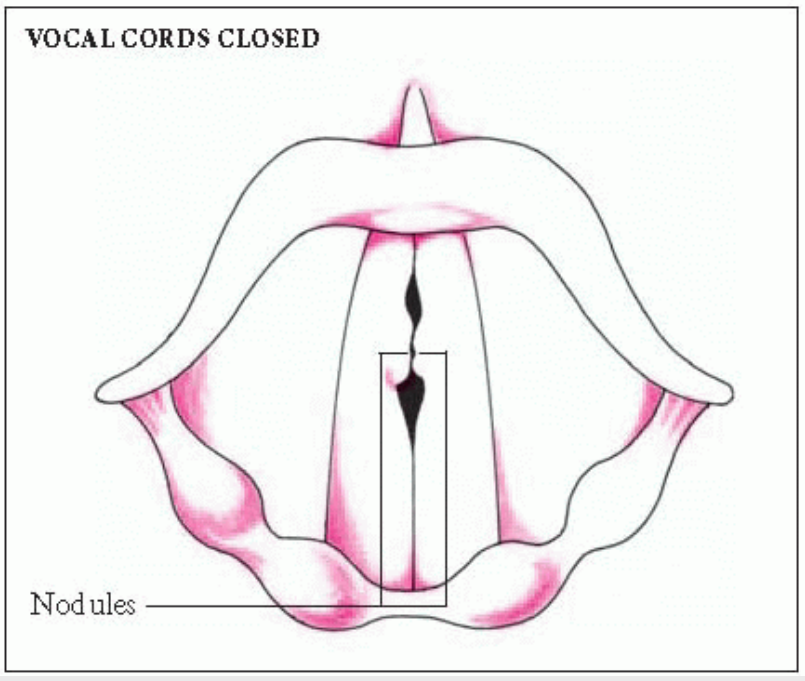
- Postoperatively, stress the importance of resting the voice for 10 to 14 days while the vocal cords heal. Provide an alternative means of communication—Magic Slate, pad and pencil, or alphabet board. Place a sign over the bed to remind visitors that the patient shouldn't talk. Mark the intercom so other facility personnel are aware that the patient can't answer. Minimize the need to speak by trying to anticipate the patient's needs.
- If the patient is a smoker, encourage him to stop smoking entirely or, at the very least, to refrain from smoking during recovery from surgery.
- Use a vaporizer to increase humidity and decrease throat irritation.
- Make sure the patient receives speech therapy after healing if necessary, because continued voice abuse causes recurrence of growths.

VOCAL CORD NODULES

The most common site of vocal cord nodules is the point of maximal vibration and impact (junction of the anterior one-third and the posterior two-thirds of the vocal cord).



Vocal cord nodules affect the voice by inhibiting proper closure of the vocal cords during phonation.



Laryngitis

A common disorder, laryngitis is an acute or chronic inflammation of the vocal cords. Acute laryngitis may occur as an isolated infection or as part of a generalized bacterial or viral upper respiratory tract infection. Repeated attacks of acute laryngitis produce inflammatory changes associated with chronic laryngitis.

ALERT

Several forms of laryngitis occur in children and can lead to significant or fatal respiratory obstruction, such as croup and epiglottiditis.

Causes and incidence

Acute laryngitis usually results from infection (primarily viral) or excessive use of the voice, an occupational hazard in certain vocations (teaching, public speaking, or singing, for example). It may also result from leisure activities (such as cheering at a sports event), inhalation of smoke or fumes, or aspiration of caustic chemicals. Chronic laryngitis may be caused by chronic upper respiratory tract disorders (sinusitis, bronchitis, nasal polyps, or allergy), mouth breathing, smoking, constant exposure to dust or other irritants, and alcohol abuse.

Complications

- Permanent hoarseness
- Airway obstruction in severe laryngitis

Signs and symptoms

Acute laryngitis typically begins with hoarseness, ranging from mild to complete loss of voice. Associated clinical features include pain (especially when swallowing or speaking), a persistent dry cough, fever, laryngeal edema, and malaise. In chronic laryngitis, persistent hoarseness is usually the only symptom.

Diagnosis

CONFIRMING DIAGNOSIS

Indirect laryngoscopy confirms the diagnosis by revealing red, inflamed and, occasionally, hemorrhagic vocal cords, with rounded rather than sharp edges and exudate. Bilateral swelling may be present.

In severe cases or if toxicity is a concern, a culture of the exudate is obtained. Consider 24-hour pH probe testing in chronic laryngitis and gastroesophageal reflux disease (GERD). Also consider biopsy in chronic laryngitis in an adult with a history of smoking or alcohol abuse.

Treatment

Primary treatment consists of resting the voice. For viral infection, symptomatic care includes analgesics and throat lozenges for pain relief. Bacterial infection requires antibiotic therapy. Severe, acute laryngitis may necessitate hospitalization. When laryngeal edema results in airway obstruction, a tracheostomy may be necessary. In chronic laryngitis, effective treatment must eliminate the underlying cause. Antacids or histamine-2 blockers may be used if GERD is the cause. Steam inhalation may also prove beneficial as are smoking cessation, reducing alcohol intake, and job change or modification if warranted.

Special considerations

- Explain to the patient why he shouldn't talk, and place a sign over the bed to remind others of this restriction. Provide a Magic Slate or a pad and pencil for communication. Mark the intercom panel so other facility personnel are aware that the patient can't answer. Minimize the need to talk by trying to anticipate the patient's needs.
- For the patient with a bacterial infection, stress the importance of completing the full course of antibiotic therapy.
- Suggest that the patient maintain adequate humidification by using a vaporizer or humidifier during the winter, by avoiding air conditioning

during the summer (because it dehumidifies), by using medicated throat lozenges, and by not smoking.

- Obtain a detailed patient history to help determine the cause of chronic laryngitis. Encourage the patient to modify predisposing habits, especially to stop smoking.
- Provide the patient with assistance for smoking cessation as well as for modification of other predisposing habits or occupational hazards.

Juvenile angiofibroma

An uncommon disorder, juvenile angiofibroma is a highly vascular, nasopharyngeal tumor made up of masses of fibrous tissue that contain many thin-walled blood vessels. The prognosis is good with treatment.

Causes and incidence

A type of hemangioma, this tumor grows on one side of the posterior nares and may completely fill the nasopharynx, nose, paranasal sinuses and, possibly, the orbit. More commonly sessile than polypoid, juvenile angiofibroma is nonencapsulated; it invades surrounding tissue.

Juvenile angiofibroma is typically found in adolescent males and is extremely rare in females. It's associated with nasal obstruction and epistaxis.

Complication

- Secondary anemia

Signs and symptoms

Juvenile angiofibroma produces unilateral or bilateral nasal obstruction and severe recurrent epistaxis, usually between ages 7 and 21. Recurrent epistaxis eventually causes secondary anemia. Associated effects include purulent rhinorrhea, facial deformity, and nasal speech. Serous otitis media and hearing loss may result from eustachian tube obstruction.

Diagnosis

A nasopharyngeal mirror or nasal speculum permits visualization of the tumor. X-rays show a bowing of the posterior wall of the maxillary sinus. Three-plane magnetic resonance imaging and computed tomography scans determine the extent of the tumors, which are seldom limited to the nasopharynx. Angiography determines the size and location of the tumor and shows the source of vascularization.

ALERT

Tumor biopsy is contraindicated because of the risk of hemorrhage.

Treatment

Surgical procedures range from avulsion to cryosurgical techniques. Surgical excision is preferred after embolization with Teflon or an absorbable gelatin sponge to decrease vascularization. Whichever surgical method is used, this tumor must be removed in its entirety and not in pieces.

Preoperative hormonal therapy may decrease the tumor's size and vascularity. Blood transfusions may be necessary during avulsion. Radiation therapy produces only a temporary regression in an angiofibroma but is the treatment of choice if the tumor has expanded into the cranium or orbit. Because the tumor is multilobular and locally invasive, it recurs in about 30% of patients during the first year after treatment, but rarely after 2 years.

Special considerations

- Explain all diagnostic and surgical procedures. Provide emotional support; severe epistaxis frightens many people to the point of panic. Monitor hemoglobin levels and hematocrit for anemia.
- After surgery, immediately report excessive bleeding. Make sure an adequate supply of typed and crossmatched blood is available for transfusion.

- Monitor for any change in vital signs. Provide good oral hygiene, and use a bedside vaporizer to raise humidity.
- During blood transfusion, watch for transfusion reactions, such as fever, pruritus, chills, or a rash. If any of these reactions occur, discontinue the blood transfusion and notify the physician immediately.
- Teach the patient's family how to apply pressure over the affected area, and instruct them to seek immediate medical attention if bleeding occurs after discharge. Stress the importance of providing adequate humidification at home to keep the nasal mucosa moist.

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