Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 3: Middle Ear and Mastoid

Chapter 25: Diseases of the Tympanic Membrane

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The tympanic membrane separates the delicate structures of the middle and inner ear from the external environment. As the demarcating structure between the external ear canal and middle ear, the tympanic membrane is involved in diseases of these structures, the mastoid complex and the eustachian tube. The tympanic membrane serves as a window into the middle ear, providing an avenue for the diagnosis of middle ear disease. A thorough history, combined with careful otoscopic examination, will help achieve an accurate diagnosis leading to proper treatment of diseases of the tympanic membrane and associated structures.

Examination

The tympanic membrane is conically shaped, with the apex of the cone at the umbo. Although it has been generally accepted that the vertical axis of the tympanic membrane is longer than the horizontal axis, recent information (Wajnberg, 1987) reveals that the horizontal axis of the tympanic membrane is 9 to 10 mm long and the vertical axis 8 to 9 mm long, from a series of 28 temporal bones. The tympanic membrane is situated obliquely at the medial end of the external ear canal, with the anterior angle between the tympanic membrane and the external ear canal being much more acute in infants and small children. The tympanic membrane develops embryologically from an area of contact between the ectodermally derived meatal plug and the endodermally derived tubo-tympanic recess, which gives rise to the middle ear cavity. Thus, the tympanic membrane consists of three layers, an outer ectodermal layer of squamous epithelium, an intermediate mesodermal fibrous layer, and an inner endodermal layer of mucosa. The thickness of the tympanic membrane is about 130 microns; the lateral epithelial layer is about 30 microns, the lamina propria about 100 microns, and the medial mucosal layer is very thin (Govaerts et al, 1988). The malleal prominence (short process of the malleus) separates the pars tensa inferiorly from the pars flaccida (Shrapnell's membrane) superiorly. The blood supply of the tympanic membrane derives from the anterior tympanic artery, which is a branch of the mandibular portion of the internal maxillary artery. Branches of the anterior tympanic artery supply the ossicles, the mucosa on the medial side of the tympanic membrane, and a descending branch of the malleal artery provides the radial arterial supply to the lateral tympanic membrane. The venous supply closely parallels the arterial supply. The nerve supply of the lateral tympanic membrane is from the auriculotemporal branch of the trigeminal nerve and from Arnold's nerve, the auricular branch of the vagus nerve. Jacobson's nerve, a branch of cranial nerve IX, supplies sensory fibers to the mesotympanum and medial tympanic membrane.

After a careful history, a thorough examination of the tympanic membrane is necessary to accurately identify disease of the tympanic membrane or middle ear. The tympanic membrane can be evaluated with a hand-held otoscope (a halogen light source is preferable)

or a binocular microscope and otologic speculum. The ear canal must be straightened to properly visualize the entire tympanic membrane. After gently pulling the auricle superiorly and posteriorly, a correctly fitting speculum is used. The speculum should be large enough to fill the ear canal, but not so large as to cause patient discomfort. Attachments for pneumatic otoscopy may be used with both the hand-held otoscope and binocular microscope.

Knowledge of the normal tympanic membrane and its anatomy is important to contrast the physical findings in disease states. A careful examination notes the angle and position of the tympanic membrane in relation to the ear canal. The pars tensa, with its normal conical shape, and the pars flaccida are carefully evaluated. The entire tympanic membrane and tympanic ring must be visualized or the examination is incomplete. Middle ear structures seen through a normal translucent tympanic membrane include the long and short process of the malleus, lenticular process of the incus and stapes, chorda tympani nerve, and round window niche. The normal vascular pattern of the tympanic membrane is that of a small, almost indistinguishable malleal artery, and extremely fine, radially distributed tympanic membrane vessels. In inflammation these vessels become more prominent, particularly the radiating vessels at the periphery of the tympanic membrane.

Radial migration of the tympanic membrane squamous epithelium has been recognized for over a century. The epithelium migrates in a spiral pattern from the umbo in most normal subjects. The rate of movement is from 0.016 to 0.05 mm per day (Boxall et al, 1988). Ultrastructural study of the tympanic membrane epithelium has not revealed any special features associated with migrating epithelial cells. Acute perforation or ventilation tube insertion of the tympanic membrane does not seem to disturb the general pattern of migration. Likewise, middle ear effusion does not seem to influence migration characteristics. On the other hand, retraction pockets display marked disturbance of the normal migratory pattern. Labeling of the tympanic membrane in these areas reveals stasis and delayed epithelial migration (O'Donoghue, 1984). There are several theories, all unproven, as to the exact cause of tympanic membrane epithelial migration. The purpose of the epithelial migration is most likely to preserve a thin tympanic membrane for its important function of sound transmission. Accumulation of epithelium on the tympanic membrane would result in a thickened structure with diminished sound transmission.

Abnormalities of the tympanic membrane noted on physical examination are often diagnostic of pathologic conditions. An abnormally patent eustachian tube produces movement of the tympanic membrane with respiration. This is more pronounced if the patient closes his mouth and one nostril and breathes rapidly through the nostril on the same side as the ear being examined. Inflammation of the tympanic membrane causes the radiating blood vessels to become more prominent. This is often the first sign of acute otitis media and can be seen in chronic otitis media as well. Discoloration of the tympanic membrane is a sign of disease. Middle ear effusion may cause the drum to appear whitish to yellow to orange in color. Chronic effusions may cause the tympanic membrane to appear bluish in color. Blood in the middle ear may also cause the tympanic membrane to appear reddish blue to purple in color. A totally opaque white tympanic membrane may be the result of tympanosclerosis, cholesteatoma, or a healed perforation. Fungal infection of the ear canal and tympanic membrane will give the epithelial surface a silky black or green appearance.

Retraction of the tympanic membrane is an important sign of middle ear and tympanic membrane disease. A thorough examination for retraction encompasses both the pars tensa and the pars flaccida. Retraction may be generalized, as an atelectatic tympanic membrane, or a localized pocket, which is identified as an isolated scooped-out area in the tympanic membrane. This occurs most often in the pars flaccida. Binocular microscopic examination with pneumatic otoscopy is extremely valuable in distinguishing a retraction pocket from a perforation. Pneumatic otoscopy may cause a retraction pocket to move under pressure. In some cases the retraction pocket is adhesed to the middle ear mucosa and does not move with pneumatic otoscopy. Careful assessment of the entire periphery of a suspected perforation may reveal a thin, dimeric tympanic membrane retracted into the middle ear space.

Bulging of the tympanic membrane is usually seen in acute inflammatory conditions. Acute otitis media causes a red, inflamed, bulging tympanic membrane with loss of the usual conical shape and landmarks. Bullous myringitis causes single or multiple raised blebs on the tympanic membrane. A bulging tympanic membrane that is bluish red to purple in color may represent a jugular vein dehiscence or middle ear vascular tumor. A glomus tumor should be particularly suspected if the inferior portion of the tympanic membrane or the hypotympanum is involved (Pulec, 1972).

Tympanic membrane perforations are associated with acute and chronic disease processes and can be difficult to recognize. In acute otitis media with perforation of the tympanic membrane there will be active drainage from the ear canal. There may be a tiny button of granulation tissue at the area of perforation. In more chronic disease states, tympanic membrane perforation may or may not be associated with drainage into the external ear canal. With careful ear canal cleaning under the microscope, one can evaluate carefully the entire rim of the perforation. Perforations should be categorized as to whether they involve the pars tens or the pars flaccida. It is important to also categorize a perforation in which a rim of tympanic membrane can be seen completely around the perforation or whether it is a marginal perforation. Careful microscopic examination will reveal areas where squamous epithelium is in the middle ear space or attached to the ossicles.

Immittance audiometry can provide useful objective information as to the status of the tympanic membrane, ossicular chain, and middle ear. These tests are discussed elsewhere and are indicated to confirm the presence or absence of the various diagnoses of tympanic membrane or middle ear disease. On rare occasions patients will complain of fluttering of an ear with certain sound stimuli, swallowing, or eye blink. On reproduction of the stimulus and utilization of immittance audiometry, myoclonus of the tensor tympani muscle can usually be diagnosed.

Otitis Media

Shambaugh (1967) states that there are five distinct types of inflammatory reactions that may involve the middle ear space, all of which are reflected in reactions of the tympanic membrane. These five clinical entities are (1) acute suppurative otitis media, (2) acute viral otitis media and bullous myringitis, (3) acute necrotizing otitis media, (4) tuberculous chronic otitis media, and (5) nontuberculous chronic otitis media.

Acute Suppurative Otitis Media

Acute suppurative otitis media is the term used for bacterial inflammation of the middle ear. It usually runs a specific clinical and pathological course. It may be terminated at any stage, depending on the virulence of the organism, host resistance, and the interjection of appropriate therapy, such as myringotomy and/or administration of antibiotics. The subject is covered elsewhere and is discussed here only in reference to the effect on the tympanic membrane.

The first stage is hyperemia. This phase is characterized by fever, pain, and fullness in the ear, but the hearing is normal. Congestion of the vessels usually starts along the malleus handle and progresses to involve the periphery of the entire membrane. There is no middle ear effusion in this stage.

The second stage is that of exudation. This stage is characterized by increased pain and fever. There is an outpouring from the capillaries of serum, fibrin, red cells, and polymorphonuclear leukocytes. With time the exudate may fill the entire middle ear space. The tympanic membrane is seen to be reddened and thickened and may be bulging. The usual landmarks of the light reflex, umbo, manubrium of malleus, and short process may not be visible. In the presence of severe pain, fever, and bulging of the membrane, a myringotomy may be performed to relieve the pain.

The third stage is suppuration. This follow myringotomy or spontaneous rupture of the membrane. The patient usually becomes less toxic, with a decrease in pain and fever. A small perforation is seen in the pars tensa. At first the discharge may be quite bloody, but shortly it becomes mucopurulent. As opposed to acute necrotizing otitis media, the acute suppurative form causes a small perforation that rarely enlarges. The infection in some cases may progress beyond the stage of suppuration to involve other structures such as the mastoid, lateral sinus, and tegmen.

The stage of resolution is an important stage to recognize clinically. The otologist is often asked to see a patient thought to have had an episode of acute otitis media within the previous few days. If examination does not reveal signs of a resolving acute otitis media, the physician should look elsewhere for a source of the symptoms referable to the ear. The perforation usually closes spontaneously. Desquamation of the epithelial layer of the outer portion of the drum is seen. Characteristically, the desquamation takes place in rather uniform lines radiating toward the umbo. With time the hyperemia decreases and the tympanic membrane becomes less opaque. Frequently there is a residual accumulation of fluid in the middle ear following an episode of acute suppurative otitis media. The fluid is usually mucoid but may be serous or purulent in nature. Spontaneous resolution occurs with time. Ninety per cent of children will have resolved the middle ear effusion 3 months after an episode of acute otitis media.

Acute Viral Otitis Media

Viral infections of the middle ear are of two basic types. The first is that associated with viral upper respiratory infections and is associated with histopathological changes in the lining of the eustachian tube and middle ear. Other than the tympanic membrane findings of

negative middle ear pressure, there is rarely involvement of the tympanic membrane itself that would be of diagnostic value. The second type of middle ear infection is bullous myringitis.

Bullous Myringitis

A form of viral involvement often confined to the tympanic membrane is called bullous myringitis. The condition often occurs in epidemics and primarily involves younger children. There may be severe pain without fever and hearing loss. The inflammation involves the drum and adjacent canal wall and is seen as multiple blebs that are reddened and inflamed. Unless there is a secondary bacterial invasion, the middle ear is not involved.

The condition is self-limiting and usually resolves in 2 to 3 days. Treatment is supportive for pain. Antibiotics, both systemic and topical, are of value to prevent secondary bacterial infection. In the presence of severe pain, the tympanic membrane can be anesthetized with 4 per cent Xylocaine and Adrenalin, using iontophoresis, and the blebs may be opened with a myringotomy knife, although incision does not change the rate of recovery.

Acute Necrotizing Otitis Media

Acute necrotizing otitis media is a more virulent form of bacterial otitis media and has features that are quite distinctive. This disease occurs in infants and young children who are acutely ill from scarlet fever, measles, pneumonia, or influenza.

The pathologic process is a true necrosis of the tympanic membrane and middle ear structures. Beta-hemolytic streptococcus is usually the offending organism. The areas with the poorest blood supply are involved most, and a kidney-shaped perforation of the center of the pars tensa occurs. The process may not extend beyond this kidney-shaped perforation in mild cases. The presence of this large perforation distinguishes this entity from acute suppurative otitis media, in which the perforation is always small. The discharge is devoid of mucus and is purulent and foul-smelling.

Acute necrotizing otitis media may proceed beyond involvement of the tympanic membrane to cause necrosis of ossicular tissue, the scutum of the bony annulus, and mastoid. With resolution of the infection there is usually a residual tympanic membrane perforation or, at best, a thin, healed, two-layer membrane, with absence of the fibrous layer. If the perforation persists, there is often recurrent or continuous drainage from the middle ear.

Cultures should be taken to identify the organism, and treatment consists of antibiotics effective against streptococcus. Surgical therapy is not indicated acutely unless other complications occur. Tympanoplastic reconstructive procedures may be indicated after resolution of infection (Stiernberg et al, 1986).

Tuberculous Otitis Media

Tuberculosis of the ear as a primary source of infection is extremely rare (Kohlmoss and Methany, 1966; Phillips, 1966). Tuberculosis involving the tympanic membrane is usually secondary to pulmonary tuberculosis and is spread through the eustachian tubes, most often by the forceful expulsion of hemoptysis and infected blood into the tympanum. The condition

usually begins as an apparent serous otitis media. When a myringotomy is done, a cloudy exudate is found and the ear continues to drain.

Pain is rarely noticed, and the perforations seem to enlarge in the face of ordinary medical therapy such as aural hygiene and instillation of topical medications. The tympanic membrane becomes thickened and pale and sometimes bulges. There may be multiple perforations in the early stages, but they coalesce into a total tympanic membrane perforation accompanied by pale granulation tissue. Loss of ossicular mass often produces a greater conductive hearing loss than expected from the perforation alone. Diagnosis is confirmed by cultures. Treatment is accomplished by using appropriate drug therapy, similar to that for pulmonary infections. Tympanoplasty may be carried out successfully after all signs of active infection have subsided.

Nontuberculous Chronic Otitis Media

Excluding serous otitis media, which is discussed elsewhere, chronic otitis media can be a result of acute otitis media with persistent perforation, bone destruction, persistent inflammation, cholesterol granuloma, and/or cholesteatoma formation. Localized allergy or the effect of a generalized dermatitis may involve the middle ear. Whenever the clinical course, response to treatment, and otoscopic examination are not entirely typical of chronic suppurative otitis media, the otolaryngologist must consider chloroma, lupus erythematosus, granulomatous diseases such as Wegener's histiocytosis X, or malignant otitis externa (Schwartzman et al, 1972).

Tympanosclerosis

Tympanosclerosis is a common finding in many tympanic membranes, consisting of irregular white patches centrally located in the drum head. Zollner (1956) introduced the term tympanosclerosis and gave a description of the condition. The pathologic lesion consists of thickening of the lamina propria of the tympanic membrane. This central layer contains abnormal hyalinized collagen. Cartilaginous or calcified deposits may also be seen. The central fibrous layer of the tympanic membrane is thinned or absent, and the small remnant may be calcified. Tympanosclerosis usually involves the tympanic membrane alone but may involve the middle ear and ossicular chain. Tympanosclerotic lesions may involve the epitympanum, causing ossicular chain fixation. Involvement of the stapes and tensor tympani tendon may also be seen. Tympanosclerosis is a benign condition and is generally considered to be the inactive end-product of recurrent or chronic otitis media. It is usually not an invasive or progressive process and is not associated with cholesteatoma. Removal of tympanosclerosis is not usually associated with recurrence.

The etiology of tympanosclerosis is not completely understood. Although it is generally accepted that tympanosclerosis follows recurrent acute otitis media or other middle ear inflammatory disease, there are many theories as to the precise mechanism of development of tympanosclerosis. Schiff and colleagues (1980) hypothesized that the disease is caused by an immunologic reaction to exposure of the lamina propria of the tympanic membrane during inflammation. In the presence of middle ear infection, the mucosa of the medial surface of the tympanic membrane becomes permeable and exposes the lamina propria to the immune system. If sensitization occurs, the body may react immunologically against the lamina

propria, which may result in tympanosclerosis. This theory was supported by an elegant experiment in which guinea pig tympanic membrane was removed and an antigen of the lamina propria was prepared. This material was injected into rabbits, and antibodies were formed against the guinea pig tympanic membrane lamina propria. Injected into guinea pigs, these antibodies bound to the tympanic membrane after trauma, infection, or cautery.

It has been noted that tympanosclerosis seems to occur more commonly in tympanic membranes in which grommets have been inserted (Slack et al, 1984). These authors believed that many factors could be involved in the genesis of tympanosclerosis in this instance, including alteration in the blood supply to the tympanic membrane, myringotomy incision, intratympanic membrane hemorrhage following myringotomy, or as a result of the action of mass of the grommet on the microstructure of the tympanic membrane. Wielinga and associates (1988) hypothesized that tympanosclerosis was an end result of mechanical deformation of the pars tensa. They performed an animal experiment in which otitis media was evoked by obstruction of the eustachian tube Wistar rats, which over time resulted in tympanosclerosis. It is more likely that the tympanosclerosis in this instance was generated by the otitis media with effusion, which was caused by the obstruction of the eustachian tube. In summary, the etiology of tympanosclerosis is most likely that of an immune reaction to injury of the lamina propria of the tympanic membrane as a result of recurrent infection. This process might be exacerbated by placement of tympanostomy tubes, which further exposes the lamina propria to the immune system.

Tympanosclerotic plaques are a common clinical finding and are rarely of significance. Hearing loss is uncommon unless a majority of the tympanic membrane or middle ear cleft is involved. Treatment is not indicated in cases in which hearing loss is not evident. In patients undergoing tympanoplasty, tympanosclerotic plaques are associated with poor vascular supply to the affected areas of the tympanic membrane. This may be associated with graft failure, and it is generally recommended to remove large tympanosclerotic plaques in order to improve the success rate of grafting. Small residual tympanosclerotic plaques, if left intact, do not usually affect hearing results.

Tympanosclerosis involving the middle ear is generally handled by removal of the tympanosclerotic lesions. Lesions with extensive involvement of the ossicles or stapes must be handled with tympanoplastic reconstructive techniques. Removal of the incus or malleus may be necessary, with reconstruction to the stapes. Tympanosclerosis involving the stapes is handled by removal of the tympanosclerotic plaques and mobilization of the stapes when possible. Stapedectomy is not done initially. If the hearing remains poor or improves and then decreases, a stapedectomy may be done. Technical details of reconstruction of the ossicular chain in these instances is discussed elsewhere (Chapter 32).

Atelectatic Otitis Media

Atelectatic otitis media is characterized by collapse of the tympanic membrane into the middle ear space. This is a difficult problem in middle ear surgery and is generally regarded as a sequela of long-term eustachian tube dysfunction and otitis media with effusion. As a result of long-standing infection and negative pressure in the middle ear space, the lamina propria of the tympanic membrane becomes deficient and atrophic. This results in loss of the normal lateral position of the tympanic membrane and collapse into the middle ear

space. The tympanic membrane is usually thin, atrophic, and draped over the lenticular process of the incus, the stapes, and the promontory (collapse of Wilde). The tympanic membrane may adhese to the mucosa of the middle ear structures and promontory, particularly in long-standing cases. Erosion of the ossicles, especially the lenticular process of the incus, is not uncommon. It is felt that atelectasis may be a precursor to cholesteatoma development, particularly with posterior retraction pockets.

It is important to determine whether the atrophic tympanic membrane is adherent to the middle ear mucosa. On examination, using a Valsalva maneuver or with pneumatic otoscopy, the tympanic membrane may be seen to reflect off the promontory. In cases in which the tympanic membrane does not lift from the middle ear mucosa, adhesion has probably taken place. In some older patients, eustachian tube function may be normal, but because of atrophy of the lamina propria the tympanic membrane is unable to maintain its normal lateral position.

In most cases, the initial treatment of atelectatic otitis media is placement of a ventilation tube. The drum will tighten and resume its normal position, often in a very short period of time. Long-term follow-up of these patients is necessary, as extrusion of the ventilation tube may result in recurrence of retraction. In these cases it must be elucidated whether the cause is continued eustachian tube dysfunction or a weak, atrophic drum head that cannot support itself. The status of the opposite ear is often helpful in making this decision.

Patients with atelectasis and adhesion to the promontory (adhesive otitis media) or with recurrent atelectasis with an atrophic tympanic membrane that cannot support itself should be considered for surgical reconstruction of the tympanic membrane. Occasionally, a patient with adhesive otitis media can be treated by elevation of the adherent tympanic membrane from the promontory by the use of nitrous oxide anesthesia and placement of a ventilation tube. This technique was reported by Graham and Knight (1981), but the long-term results of this therapy are unclear. Paparella and Jung (1981) described tympanoplasty for atelectasis and adhesive otitis media. The tympanic membrane is repaired by first elevating a large posterior tympanomeatal flap. The very thin epithelium that has collapsed on the promontory is very carefully elevated, and the mobility and continuity of the ossicular chain is examined. Particular attention is paid to ossicular fixation and discontinuity of the ossicles. The tensor tympani tendon is severed to allow the malleus to be lateralized and to widen the middle ear space. The tympanic membrane is reinforced with a medial graft of temporalis fascia, and the middle ear space is packed with Gelfoam.

Paparella recommends the use of silicone rubber sheeting (Silastic) as a middle ear stent. Recent experience indicates that in the middle ear Silastic may be extruded and can be a potential nidus for infection, especially when chronic eustachian tube dysfunction exists. The use of Silastic for reconstruction may help maintain the middle ear space but may also cause long-term problems, requiring removal.

To finish the procedure, a ventilation tube is inserted in the anterior tympanic membrane to ensure middle ear space ventilation. Another method of reconstruction of the atelectatic tympanic membrane is the use of homograft tympanic membrane. With the advent of AIDS, the use of homografts is declining.

Tympanic Membrane Perforations

Traumatic Perforations

Perforation of the tympanic membrane is a frequent manifestation of injury and may be separated into four general categories: Compression, instrumentation, burn-slag, and blast injury. Compression injuries tend to be the most common and are usually the result of a blow to the ear that simultaneously strikes and occludes the external ear canal. Keller (1958) stated that a pressure of 25 pounds per square inch is required to rupture the ear drum. There is usually slight bleeding and pain, and there may be hearing loss and an abnormal feeling of fullness in the ear. These perforations usually involve the pars tensa and are either linear or stellate in appearance.

Instrumentation injuries are the second largest category. These are often caused by Q-Tips or bobby pins, and are usually inadvertent, occurring when a distraction causes accidental motion of the hand holding the object, driving it through the tympanic membrane. Children may occasionally injure the tympanic membrane while playing. Water is also a cause of instrumentation injury; these injuries may occur while swimming, diving, or water skiing. Rupture of the tympanic membrane is more frequent with water skiing, which involves high-speed contact with water. These injuries may have a higher incidence of marked hearing loss due to ossicular dislocation or inner ear trauma. With water injuries, contamination of the middle ear is frequent, and infection with resultant otorrhea is a therapeutic consideration.

Slag burns are frequently seen in industry from welding or caustic agents. These injuries are caused by hot metal from machines or a welder's torch contacting the tympanic membrane and usually passing into the middle ear space. There is intense pain but usually no bleeding. These perforations have a low incidence of spontaneous healing and a higher incidence of infection and otorrhea.

Blast injuries are usually seen during war and are believed to be the result of air compression of the tympanic membrane. Keller (1958) calculated that a sound level of 199 dB is necessary to cause traumatic perforation of the tympanic membrane. This level of sound would also be accompanied by an inner ear injury and probable severe to profound sensorineural hearing loss.

The vast majority of traumatic tympanic membrane perforations heal spontaneously. The healing process begins very soon after injury. One day after traumatic perforation, edema of the lamina propria and hemorrhage of any of the three layers of the tympanic membrane is seen. At 36 to 48 hours, epithelial cells begin to proliferate, followed by cells of the lamina propria and mucosal layer. Usually the outer epithelial layer migrates over the perforation, followed by mucosal and young propria cells. It is believed by some that occasionally the lamina propria may bridge the perforation. Causes of failure of healing of a perforation include (1) a defect too large for the epithelium to bridge, (2) failure of the epithelial or lamina propria cells to proliferate due to damage from the trauma, (3) infection, and (4) failure to remove the cause of the perforation, such as an indwelling ventilation tube (Govaerts et al, 1988).

Although most tympanic membranes heal with all three layers intact, some healed perforations consist of a thin, transparent sheet that is thinner than the normal tympanic membrane (dimeric membrane). Govaerts and co-workers (1988) found that this was due to thinning of the lamina propria from its normal thickness of 100 microns to approximately 2 to 3 microns. No fibroblasts were found in the lamina propria on electron microscopy, but collagen fibers were present. It seems that in the dimeric tympanic membrane, normal fibroblasts are lacking in the healing process. The most reasonable explanation is that the normal proliferation of fibroblasts in the lamina propria is inhibited, causing thinning of the lamina propria and of the tympanic membrane. The exact cause of this inhibition of fibroblastic growth is unclear.

There are a number of treatment plans for traumatic perforations. These include observation only, paper patch repair, and immediate myringoplasty. As most series show that about 90 per cent of traumatic perforations heal spontaneously, operative intervention must be measured against this standard. Reports of paper patch repair indicate a slightly higher success rate; however, because of the small numbers in these series, statistical reliability has not been achieved. Because of the high spontaneous healing rate of traumatic perforations, immediate myringoplasty is not the usual standard of care.

In general, treatment consists of complete examination of the injury using the operating microscope. The ear canal is cleaned and any foreign body is removed. The patient is instructed to keep water out of the ear and return in 4 to 8 weeks for a follow-up examination, or if otorrhea occurs. Infected perforations are treated with oral antimicrobials and possibly short-term otic drops. (The use of these drops is somewhat controversial because of the potential for ototoxicity.) Audiometric studies should be performed, and a careful assessment for middle and inner ear injury is made. In patients with associated vertigo or sensorineural hearing loss, one must suspect injury to the stapedial footplate and vestibule. In such instances, middle ear exploration is advised.

Patients who have early paper patch closure of the tympanic membrane may have slightly higher closure rates and faster healing. The ear is anesthetized with an injection of 1 per cent Xylocaine with epinephrine; occasionally no anesthesia is necessary. The ear is carefully examined for associated injury, and the edges of the perforation are evaginated. A small section of paper tape or a paper patch is positioned over the perforation, and occasionally wetting or trichloroacetic acid is used to adhese the patch to the epithelium. Gelfoam is usually not used in the middle ear. Antibiotics are not indicated unless a contaminated water injury or slag burn has occurred. These patients are usually not candidates for immediate paper patch repair (Camnitz and Bost, 1985; Lindeman et al, 1987). If a traumatic perforation has not healed after 3 to 6 months, some may respond to having the edges of the perforation freshened and a paper patch applied. If this is not successful, myringoplasty should be considered.

Tympanostomy Tubes

Tympanostomy tubes for the treatment of otits media with effusion were re-introduced in 1952 by Armstrong. Since then, tympanostomy tube surgery has become one of the most common operations performed in the USA, with over 1 million children treated annually. Effects of tympanostomy tubes on the tympanic membrane include scarring, atrophy with

dimeric membrane formation, persistent perforation, and rarely cholesteatoma formation. The most frequent complication of tympanostomy tube placement is tympanosclerosis, which has been described previously. The incidence of atrophy and dimeric membrane formation is unknown. There is controversy about other structural changes in the tympanic membrane with tympanostomy tube placement.

Meyerhoff and Shea (1984) studied 16 chinchillas in which tympanostomy tubes were inserted in the tympanic membrane. They observed granulation tissue found around the tympanostomy tubes by 7 days. This was replaced in time by a mature fibrous ring, which explained the white tissue band at the area of the tympanostomy tube that was observed with the operating microscope. Expulsion of the tympanostomy tube was accompanied by healing, with a thickened lamina propria. There were no other significant changes in the tympanic membranes.

In contrast, Soderberg and colleagues (1986) found that in a rat model repeated tympanostomy tube insertion caused a dramatically thickened pars tensa, due to thickening of the lamina propria with sclerotic plaques and occasional bonelike formations. These structural changes were found not only in the quadrant in which the tympanostomy tube is inserted but also in other areas of the tympanic membrane. These changes are consistent with tympanosclerosis formation.

Long-term perforation of the tympanic membrane following tympanostomy tube placement is probably related to the diameter of the tube used. The reported incidence of long-term perforation with small-bore tubes is between 0.5 and 3.4 per cent (Chevretton et al, 1987). The incidence with larger-bore tubes is 25 per cent with Per-Lee tubes, and 8.5 per cent with Paparella No. 2 tubes. There are several methods of treatment of persistent perforation caused by tympanostomy tubes. Chevretton and co-workers (1987) found that freshening the edges of a perforation increased the healing rate. No correlation seems to exist between the incidence of perforation and the age, sex, duration of intubation of the tympanic membrane, or presence of tympanosclerosis.

It appears that freshening the edge of the perforation, possibly accompanied by a paper patch, is reasonable initial therapy for persistent tympanic membrane perforation following tube extrusion. If this fails, myringoplasty with medial fascial grafting can be considered. The timing of this procedure is important. Patients with eustachian tube dysfunction and concurrent middle ear disease should not undergo myringoplasty. Recurrence of otitis media with effusion can be expected, possibly necessitating treatment with a tympanostomy tube again. A cycle of intubation, perforation, myringoplasty, and recurrence of otitis media with effusion is to be avoided. Myringoplasty should be delayed until eustachian tube function is adequate to provide ventilation of the middle ear space and a good result. The timing of this is difficult. Most patients 5 to 6 years of age have reasonable eustachian tube function and can undergo this surgery if the perforation has not healed. The status of the opposite ear can be very helpful. If the opposite ear has no perforation and has been clear for a year, myringoplasty can be confidently undertaken.

An alternative to grommet insertion in the treatment of otitis media with effusion is thermal myringotomy. This was reviewed by Ruckley and Blair (1988), who studied a battery-powered thermal myringotomy device that produced a small tympanic membrane perforation

in conjunction with adenoidectomy, in the treatment of otitis media with effusion. These authors found that all thermal perforations closed by 42 days, whereas patients with grommets placed had a much longer duration of intubation. Conventional myringotomy and grommet surgery provided significantly better sustained middle ear ventilation. There seems to be little indication for the use of thermal myringotomy in the treatment of otitis media with effusion.

Chronic Granular Myringitis

Chronic inflammation of the tympanic membrane, in which the inflammation is confined to the squamous layer, is among the rarer diseases of the ear (Politzer, 1894; Moffett, 1943; Senturia, 1957). Chronic granular myringitis is of unknown etiology and runs a painless course or is only occasionally accompanied by fleeting, lancinating pain in the ear. The most troublesome symptoms may be itching, otorrhea, and hearing loss. The condition is characterized by replacement of the squamous layer of the surface of the tympanic membrane by proliferating granulation tissue. Over a period of weeks or months, the squamous epithelium melts away from the entire tympanic membrane surface, and the process creeps laterally along the external auditory canal. The mass of granulation builds up over the surface of the tympanic membrane and the medial portion of the external auditory canal, with persistent drainage and moisture over a period of many years. Finally, if untreated over a period of 20 to 30 years, squamous epithelium sometimes grows over the surface of the granulation to give the appearance of a stenotic ear canal, with several millimeters of thick fibrous tissue between the normal fibrous layer of the tympanic membrane and the external skin.

With early recognition, treatment can sometimes reverse the process. Persistent and prolonged regular use of anti-inflammatory antibiotic ear drops (Cortisporin otic suspension), systemic antibiotics, and local excision and cautery with trichloroacetic acid frequently will produce a cure. Sometimes it is necessary, both in the early and late stages of the disease, to excise all granulation tissue surgically lateral to the fibrous layer of the tympanic membrane. A graft of canal skin removed from the opposite ear is placed over the denuded surfaces. Careful follow-up with local treatment is necessary in the postoperative period.

When long-term myringitis has healed spontaneously and the patient has developed stenosis of the ear canal, the mass is surgically excised, the canal is enlarged, and flaps of skin (or skin grafts) are fashioned to reepithelialize the ear canal and tympanic membrane.

Keratosis Obturans

Keratosis obturans is an unusual condition in which a cholesteatoma-like mass of desquamated epithelium forms recurrently within the external auditory canal (Bunting, 1968). The condition involves the skin of the tympanic membrane and auditory canal, which for unknown reasons does not have the property of migration to provide self-cleaning of the ear. As the debris from desquamation collects, a cystic mass enlarges to first obliterate the external auditory canal and then, if not removed, to produce bone absorption of the external auditory canal and destruction of the tympanic membrane. Treatment involves frequent regular physical cleaning of the external auditory canal to remove the accumulation of dead skin.

Chronic Otitis Media

The general topic of chronic otitis media is covered elsewhere in this text. However, a review of the effect of chronic otitis media on the tympanic membrane is discussed here. The tympanic membrane has a strong inherent capability to heal itself and retain its normal function. Many investigators have attempted to create tympanic membrane perforations in order to test various grafting materials, only to be frustrated by the rapid spontaneous healing of the drum.

The ability of the tympanic membrane to heal spontaneously is dependent on the following factors: (1) the status of disease and/or infection of the middle ear mucosa, (2) the function of the eustachian tube, (3) the status of the blood supply of the tympanic membrane, and (4) the extent of destruction of the fibrous or middle layer of the eardrum.

In the normal ear, a spontaneous rupture of the tympanic membrane, such as might occur in a rapid decompression injury or from a myringotomy, often closes within hours. Initial repair of the lamina propria may be accomplished by 2 to 3 days. There is a strong effort by the epithelium of the canal skin to bridge the gap of an incision or perforation. There is an even more rapid migration of the mucosal lining of the middle ear to bridge the gap. This fortunate situation usually prevents squamous epithelium from growing into the tympanum. In most instances, depending on the factors mentioned previously, the perforation will close. If the defect is sufficiently large that the epithelium is unable to bridge the gap, a number of situations may occur.

In most cases the epithelium of the outer surface and the mucosa of the inner surface will fuse into a transitional zone. This is often seen during the deepithelialization phase of a tympanoplasty procedure. This transitional zone will strip freely from the entire margin of the perforation.

The differentiation between central and marginal perforations was once thought to be important. A central perforation is one that is confined to the pars tensa of the tympanic membrane, and its entire circumference is bounded by a rim of intact membrane and an intact annulus. A marginal perforation is one that extends to the periphery of the membrane. Often the annular ring is deficient in the peripheral area.

Before the advent of the operating microscope and current tympanoplasty techniques, it was believed that a central perforation was "safe" because the inner layer of mucosa of the tympanic membrane would prevent ingrowth of squamous epithelium into the middle ear (contact inhibition). However, this is not always the case. It is agreed that the epithelium is much more likely to grow into the middle ear when there is a deficient annular ring, but some perforations with an intact margin will have squamous epithelium growing on the undersurface or mucosal surface of the remnant. The squamous epithelium may grow into the middle ear to involve the ossicles or into the eustachian tube. Histologic studies of temporal bones confirm this clinical observation. It is important, therefore, that the otologist study each patient with chronic otitis media and a perforation carefully to determine the extent of involvement and the surgical procedure required.

Another major area of involvement of the tympanic membrane by chronic otitis media is the pars flaccida. A particular problem is attic retraction cholesteatoma. The first stage of this entity is a small, dimple-like depression of the pars flaccida of the tympanic membrane. This may occur just anterior to the malleus handle (an anterior attic retraction) or just posterior to the short process of the malleus (a posterior attic retraction). It is important to completely examine the attic area whenever examining an ear. A quick glance without careful superior examination may suggest the existence of a normal pars tensa, when there is significant attic retraction. The presence of a small crust of cerumen in the attic area should alert one to the possibility of an attic retraction pocket. These crusts should be gently teased away with the help of an operating microscope.

The second stage of an attic cholesteatoma occurs if the retraction becomes filled with desquamated epithelium. When this happens, the sac begins to enlarge rapidly. The third stage follows closely. As the sac begins to enlarge there is bone destruction. This is usually seen as loss of a portion of the bony posterior superior canal wall. This process then proceeds to destruction of ossicular tissue and other consequences of cholesteatoma formation.

The etiology of attic retraction cholesteatoma was thoroughly discussed by Williams (1969). Attic retraction formation results from "aditus block" or obstruction of passageway from the middle ear to the mastoid, preventing aeration. The negative pressure produced in the mastoid draws in the pars flaccida. Another proposed mechanism is smoldering infection that produces a contraction scar from the attic folds and retraction of the pars flaccida.

Attic retractions must be followed closely by the otologist. If all portions of the sac are visible and can be meticulously cleaned, there is little danger of bone destruction, associated hearing loss, or more significant complications. If the process develops past this point it becomes a surgical problem, and the principles of modern otology should be applied to the surgical treatment of these patients. With strict attention to technique, the disease process can be removed, with retention of normal anatomic structures and restoration of good hearing.

Management of Chronic Otitis Media and Tympanic Membrane Grafting

The presence of a perforation of the tympanic membrane, or injury or malfunction of other portions of the tympanum and ossicular chain, is an indication for surgical repair as an elective procedure. Since Zollner (1951) and Wullstein (1952) opened the way to tympanoplasty, many grafting materials have been used, the most common being ear canal skin (House, 1958) and fascia (Storrs, 1961). Other materials that have been used include perichondrium, vein, split-thickness skin, animal intestinal membranes, heart valves, dura, corneas, and homograft fascia.

The goals of myringoplasty or tympanoplasty are (1) complete removal of disease and squamous epithelium from the middle ear and mastoid; (2) preservation of the posterior bony external auditory canal; (3) aeration of the middle ear and mastoid; (4) preservation or restoration of useful hearing with complete elimination of an air-bone gap; and (5) permanent closure of the tympanic membrane perforation. This produces a dry, trouble-free ear. These goals can be obtained only if the well-trained, experienced otologist follows the principles of good surgical management.

Attention to detail and execution of meticulous surgical technique are requisite for success. It is also of significant importance that the procedures be accomplished with a minimum of morbidity and trouble for the patient. It is convenient to classify the area and amount of involvement of the ear by disease, so that a method of management can be outlined and the patient can be preoperatively apprised of the extent and type of surgery required. Depending on the area and extent of involvement, predetermined surgical procedures can be utilized, either alone or together, as building blocks that can be called on when needed.

Myringoplasty is defined as repair of the tympanic membrane without disturbing the middle ear ossicular chain. Tympanoplasty includes operations on the middle ear and ossicular chain in addition to repair of tympanic membrane perforations. Myringoplasty is one of the more common and important otologic procedures and must be mastered as a basis for all otologic surgery.

Repair of the tympanic membrane is accomplished with grafting, usually autologous fascia, and the most commonly used methods are the overlay and underlay medial grafting techniques. The graft acts as a scaffold for epithelial healing, as the migrating squamous epithelium exactly follows the shape of the graft from the margin of the perforation until healing is complete. Fresh fascia grafts undergo early autolysis and are quickly resorbed (Boedts, 1984). Formaldehyde-preserved tympanic homografts preserve their structure longer; however, homografting techniques are falling into disfavor due to concerns about transmission of human immunodeficiency virus and the Creutzfeldt-Jakob disease agent (Glasscock, 1988).

Success rates of tympanoplasty procedures in most reported series approach 90 per cent. The expectations for success are high, but the 90 per cent success rates reported are usually short-term results. Tos reported an 88 per cent success rate, and Glasscock 93 per cent success rate with short-term follow-up (Tos, 1985; Glasscock, 1982).

Halik and Smyth (1988) reported an "unadjusted" 89 per cent success rate, with as long as 15 years of follow-up. However, due to pitfalls in reporting data, the corrected success rate calculated on survival life table analysis methods revealed a real success rate of 81 per cent for perforation closure at 11 years, and a normally healed intact tympanic membrane in only 74 per cent of the patients at this follow-up interval. They found that patients less than 10 years of age had a higher failure rate, and that revision surgery had a significantly smaller chance of success, with only a 60 per cent chance of closure at each revision procedure. They found that certain factors have no effect on the final outcome, including sex of the patient, presence or absence of purulent drainage from the ear at the time of surgery, and the type of graft material used.

Ophir and colleagues (1987) found that children in the 5- to 8-year-old age group undergoing myringoplasty had a 77 per cent success rate versus an 80 per cent success rate for older children 9 to 12 years of age. They agreed that success in tympanoplasty surgery is not related to the presence or absence of purulent drainage from the ear or to the performing of adenoidectomy before myringoplasty.

Studies of hearing results in tympanoplasty reveal generally good results. Halik and Smyth (1988) found that 80 per cent of their patients had closure of the air-bone gap to within 10 decibels at 5 years of follow-up. They noted a 3 per cent rate of sensorineural

hearing loss and believed that this was comparable to other series reviewed in their report. Ophir and co-workers (1987), studying the pediatric population, found that in 88 per cent of children the postoperative air-bone gap was closed to within 20 decibels, with a 2.6 per cent incidence of anacoustic ear as a result of surgery. They reported that the rate of closure of younger children and older children was comparable. As such, the success rates of graft "take" and hearing results seem to be reasonably comparable in myringoplasty and tympanoplasty.

It is generally believed that poor eustachian tube function and the presence of otitis media in the pediatric age group are reasons for the less successful outcome of myringoplasty. The optimal age for myringoplasty in the pediatric age group has not been determined. The function of the eustachian tube is felt to have a significant role in the outcome of this surgery. Manning and associates (1987) studied 56 children (63 ears) undergoing tympanoplasty for central perforation. A 78 per cent success rate was reported. They found that the prognostic value of preoperative eustachian tube function studies was low, and that other factors, such as medial or lateral graft placement, were also not associated with outcome. They concluded that eustachian tube function tests as evaluated were predictive of a good outcome if the results were good, but that poor tubal function was not helpful in predicting a poor outcome. As such, there exists as yet no good eustachian tube function tests to predict the long-term function and recovery of eustachian tube function, and with that the success of graft placement.

Myringoplasty can be accomplished by either a lateral grafting technique or an underlay grafting technique. Autologous temporalis fascia is the most common and readily available grafting material. Lateral grafting is accomplished by first injecting Xylocaine with epinephrine through a speculum in the posterior vascular strip of the ear canal. Incisions are made in the ear canal, and all canal skin is removed in continuity with the squamous epithelium of the tympanic membrane remnant. The canal skin is trimmed, and usually the tympanic membrane perforation rim is excised to freshen the edges and remove any squamous epithelium that may have inverted into the middle ear space at the edge of the perforation. The canal skin is replaced over the trimmed perforation; however, in some cases fascia may be placed over the perforation, and then the canal skin is placed lateral to the fascia graft. The ear canal is then filled with Gelfoam.

Alternatively, Glasscock (1973) described the commonly used medial grafting technique. After proper preparation of the ear, vascular strip incisions are made in the ear canal. The inferior incision is extended toward the inferior canal. A postauricular incision is then made, and dissection is carried down the posterior ear canal. The vascular strip, which has been outlined by the previously made ear canal incisions is retracted anterior with the pinna, and the tympanic membrane remnant is exposed. The edges of the perforation are freshened, and any inverted epithelium is removed. The posterior tympanic membrane remnant is then elevated and may be bisected with the flaps reflected superiorly and inferiorly. The middle ear is filled with absorbable gelatin sponge, and a previously harvested autologous fascia graft is trimmed and placed medially to the tympanic membrane remnant. The flaps are reflected back over the fascia graft, and Gelfoam is placed over the graft and remnant. The pinna is then replaced in its normal position and the posterior vascular strip is replaced. The ear canal is then filled with Gelfoam or antibiotic ointment.

Surgical techniques for cholesteatoma and ossicular chain reconstruction are described elsewhere and will not be addressed in this chapter (Vol II, Chap 32).

Ototoxicity

Topical otic drops containing aminoglycoside antibiotics, steroids, and propylene glycol solvent are commonly used in the treatment of external otitis, otorrhea from tympanic membrane perforations, or draining tympanostomy tubes. Recently, interest has been spurred in the ototoxicity of these preparations because of reports of their toxic effects (Meyerhoff et al, 1983; Wright and Meyerhoff, 1984; Wright et al, 1987). These preparations cause severe middle ear inflammatory changes in experimental animals, including mucosal hyperplasia, granulation tissue formation, and osteoneogenesis. In addition, cochlear damage with degeneration of inner and outer hair cells and damage to the stria vascularis have been reported.

Masaki and colleagues (1988) studied the effects of these drops on the tympanic membrane in chinchillas. They found a consistent pattern of structural changes following the administration of this topical preparation into the middle ear cavities of chinchillas. At 2 days after application, the mucosal and epidermal layers of the tympanic membrane were completely destroyed over wide areas of the tympanic membrane. The fibrous lamina propria retained its structure relatively intact. At 4 days, reepithelialization of the lateral surface of the tympanic membrane occurred, with hyperplasia and thickening. At 1 week the epidermis was dramatically hyperplastic. The medial surface of the tympanic membrane remained denuded of mucosa cells at 1 week but returned approximately 2 weeks after administration of the drops. At 3 weeks, animals were observed to have disruption of the fibrous layer of the lamina propria and invasion of keratinizing epithelium through the lamina propria to the medial surface of the tympanic membrane. Perforation was observed in some animals.

The most toxic element of these combination otic drops used may be the solvent propylene glycol. This component is a potent irritant and produces severe inflammation in the middle ear cavity. Vasocidin ophthalmic drops, which contain sulfacetamide sodium and prednisolone sodium phosphate, have been studied. This agent is in common clinical use and was studied to assess its effect in the middle ear in the chinchillas. After administration into the middle ear, thickening of the epidermal layer of the tympanic membrane in the mucosal layer, with increased keratin production on the lateral surface was found at 1 week. Hemorrhage of small blood vessels into the tympanic membrane was also found. Often there was an accumulation of clear fluid between the keratin layer and the epidermal cells on the lateral aspects of the tympanic membrane, and some of these specimens contained crystals characteristics of cholesterol. At 4 weeks, the inflammatory changes were essentially resolved. The tympanic membranes were much more normal in appearance, with only crusts of keratinous debris on the lateral surfaces of the tympanic membranes. Otherwise the examinations were essentially normal. This implies that Vasocidin causes short-term inflammatory process that is quickly reversible (Brown et al, 1988).

The clinical implications of these findings are not clear as to the toxicity in humans of topical agents containing aminoglycosides or propylene glycol. Studies to assess the clinical effects on patients with otorrhea have not been performed. As such, clinical recommendations regarding the use of these topical preparations in patients with tympanic membrane

perforations or tympanostomy tubes cannot be made. Further research to define the toxicity of these drugs in patients and to develop new, nontoxic topical preparations is needed.