Otosclerosis is a primary disease of the otic capsule and the ossicles. It is not known to occur outside the human temporal bone. It is characterized by a progressive hearing loss that may be conductive, sensorineural, or mixed. Histologically, it is seen as abnormal resorption and then deposition of bone in the labyrinthine capsule and middle ear. Otosclerosis is an important cause of auditory and, to a lesser extent, vestibular symptoms, and both can be treated effectively in most cases.

History

Valsalva was the first to describe stapes fixation as a cause of hearing loss in 1704 when he performed an autopsy of a known deaf patient. In 1894, Politzer termed the ankylosis “otosclerosis.” Many varied procedures were attempted over the years with Lempert being the first to consistently improve hearing in otosclerosis with a fenestration of the horizontal canal in the late 1930s. This was the treatment of choice for 20 years but patients were left with an average residual 20-25 dB CHL even in the best hands. Rosen began to mobilize the stapes in 1953 with complete closure of the air-bone gap in many of his patients. Shea is considered the father of modern stapes surgery when he proposed and performed the first stapes removal with placement of an artificial nylon stapes in 1955. Since then many techniques have been performed and perfected by prominent otologists with the most recent addition being a laser stapedotomy in the 1980s.

Histopathology

Otosclerosis is a pleomorphic bone dyscrasia. It may fix the stapes by a tiny bridge of abnormal bone or may totally obliterate it. It may spread to the cochlea to produce either a sensorineural loss of varying degree or deafness. Lesions may be sclerotic, spongiotic, or fibrous but usually compose all three histologic types. This diversity of size and anatomic distribution coincides with the variable types of hearing loss, from a mild conductive deficit to a total loss.

The smallest otosclerotic lesions usually appear adjacent to the fissula ante fenestram, just anterior to the oval window. Temporal bone studies reveal that 80-90% of patients with otosclerosis are affected in this area. The foci have two phases, and early, spongiotic phase and a later, sclerotic phase. The early phase is a highly vascular, loose, spongy bone that is responsible for the Schwartzze’s sign that is occasionally seen on otoscopic exam. Osteocytes begin resorbing bone in perivascular spaces, which is replaced by cellular, fibrous connective tissue. Within this connective tissue, reticular cells and fibroblasts become osteoblasts, which lay down immature bone with a woven, disordered pattern of collagen fibers. It stains blue with H & E, thus forming “blue mantles of Manasse.”
During the late, or sclerotic phase, the new bone is resorbed and replaced with osseous tissue containing many collagen fibers and little ground substance. The osseous tissue stains red with H & E. The large areas of vascular, cellular connective tissue are replaced with new bone, and the lesion becomes quiescent.

Within each temporal bone containing otosclerosis, lesions can be found in each of these phases although the overall histologic status seems to be fairly uniform. The lesions begin in endochondral bone, but the endosteal and periosteal layers eventually become involved as well.

Foci may be found anywhere in the temporal bone. 80% of affected bones have involvement anterior to the oval window, these foci are more likely to spread along the inferior rim than the superior rim. The round window is involved in 30% of bones but seldom is there complete obliteration. The stapes footplate is involved in 12% of cases and the anteroinferior cochlea in 14%. The wide variation in placement of lesions accounts for the varying presentations.

**Epidemiology**

Otosclerosis has a hereditary basis in 70% of cases. In these patients it is usually transmitted as an autosomal dominant trait with incomplete penetrance (approximately 40%). In Caucasians, the disease is found in 7% of male and 10% of female temporal bones. The stapes appears ankylosed in 12% of these cases, accounting for clinical symptoms of a conductive hearing loss. This represents one percent of the white population. Other races have a lesser incidence of otosclerosis with blacks having a rate one-percent that of Caucasians. In all races, when one ear is affected, the contralateral ear is affected 80-90% of the time.

**Etiology**

The etiology of otosclerosis remains unknown as of yet. Recent work has postulated infection by the measles virus as the initial insult and eventual cause of otosclerosis. McKenna et al were able to isolate measles virus RNA using PCR in 80% of the temporal bones studied with otosclerosis. None of their control temporal bones had the virus isolated. Arnold et al concurred this and also found that measles virus specific antibody was present in perilymph samples of all patients with otosclerosis in their study. There was a much lesser incidence of these antibodies in control patients and their results were statistically significant. In fact, they found that none of their otosclerotic patients had been previously immunized against the measles virus. There are other unproven but logical supports for this etiology as the incidence of otosclerosis has markedly decreased as immunization practices have improved. Also women seem to be susceptible to more severe and even fatal bouts with the measles virus and they have an increased incidence of otosclerosis as well.

**History & Physical Examination**

The patient with otosclerosis typically presents with a history of slowly progressive hearing loss that is often bilateral and asymmetric. The hearing loss usually presents between the ages of 15 and 45. It occurs approximately twice as often in women than in men. In 70% of patients there is a family history of deafness. Younger patients often have a more rapid progression of the hearing loss. It is often rapidly progressive during pregnancy and in women on estrogen therapy.

These patients often speak with a low-volume because they hear their own voices by bone conduction. Tinnitus is a common presenting complaint in patients with otosclerosis and occurs in approximately 75% of patients. Many patients (approximately 25-30%) have some type of associated vestibular symptoms. These are attributed to lesions of the horizontal canal and are usually not severe. These symptoms must be carefully differentiated from those of Meniere’s disease as hydrops is a contraindication to stapes surgery.

Physical examination of the ear is usually normal in patients with otosclerosis but it is important in ruling out other diagnoses that may present with similar symptoms. Occasionally, a reddish blush may be seen over the promontory and in the oval window niche area. This is due to the rich vascular supply of an immature otospongiotic focus of bone and is termed Schwartze’s sign. It is only identified in approximately 10% of patients with otosclerosis. A middle ear effusion may present with symptoms identical to those of otosclerosis. The presence of tympanosclerosis in the tympanic membrane can point to
Otoscopic ossicular fixation. A plaque just anterior to the neck of the malleus can result in malleus fixation. In addition, chronic adhesive otitis media, tympanic membrane perforation, and cholesteatoma can be readily identified by otomicroscopy.

Tuning forks with the Rinne and Weber tests are an important part of the physical exam and should utilize the 256, 512, and 1024 Hz frequencies. A negative Rinne test is first noted with the 256 Hz fork and progresses up the frequency spectrum with disease progression. The Weber test can help identify patients with a more advanced conductive hearing loss and can determine which ear has suffered the greatest insult.

Patients can present with no measurable hearing or with an air conduction threshold of 95 to 100 dB. It is important to consider otosclerosis in these patients as they can obtain serviceable aided hearing after corrective surgery. Sheehy pointed out clues to this diagnosis as: a positive family history with gradual progression of hearing loss starting in early life; paracusis noted in an earlier stage of hearing loss; present or previous wearing of a bone conduction hearing aid; and a previous audiogram demonstrating a measurable air-bone gap.

Your differential diagnosis of the conductive hearing loss with a relatively normal tympanic membrane should include tympanosclerosis, middle ear effusion, incus or malleus fixation, ossicular discontinuity, congenital footplate fixation, and cholesteatoma. Systemic diseases that can masquerade as otosclerosis include Paget's disease and osteogenesis imperfecta. Paget's disease may fix the stapes but more often, it crowds the ossicles in the epitympanum, partially fixing the ossicular chain. Cochlear involvement is a late finding. Elevation of the serum alkaline phosphatase level and involvement of other skeletal bones are often seen. Osteogenesis imperfecta (van der Hoeve syndrome) should be suspected in the presence of blue sclera, progressive conductive hearing loss, and multiple fractures. Their hearing loss is secondary to stapes fixation and these patients can have stapes surgery with results similar to those with otosclerosis.

Imaging

The advent of imaging tests for otosclerosis is fairly recent, coinciding with the introduction of multidirectional tomography in the 1960s. Today, this technique has been replaced with computed tomography, which is the study of choice at present if radiologic testing is used. Proponents claim that CT can visualize the extent of the pathology of the oval window and footplate and can be used when the clinical diagnosis of otosclerosis is in doubt. The radiographic appearance of otosclerosis of the oval window depends on the degree of maturation and the extent of the pathologic process. In reality, most do not use imaging in the routine diagnosis of otosclerosis. Some authors propose obtaining a CT prior to revision surgery to check the position of the prosthesis and rule out other middle ear problems. MRI has been reported to identify lesions in earlier stages with increased enhancement of the capsule.

Audiology

The key test in the diagnosis of otosclerosis remains the audiogram. The audiological manifestations of otosclerosis are as distinctive as they are varied. Characteristic abnormalities appear on tympanometry, acoustic reflexes, and the pure-tone audiogram. The tympanogram is a graphic representation of the change in acceptance of sound energy through the middle ear as a function of air pressure. The most typical case is the presence of a normal, type A tympanogram. As the disease progresses the height of the peak decreases and you can eventually obtain a type As tympanogram.

Acoustic reflex morphology is a very sensitive indicator of otosclerosis. In contrast to the normal configuration in which middle ear compliance is reduced for the duration of a stimulus, in otosclerosis there is a characteristic diphasic response or “on-off” phenomenon. With this response there is an increase in compliance at both the onset and termination (“offset”) of the sound stimulus. Forty percent of the normal population can have a brief compliance increase at the onset of a stimulus but the offset increase (at the termination of the signal) is virtually pathognomonic of early stapedial fixation. It is often seen in the first 5 years of disease progression but is rarely seen in otosclerosis of 10 years or longer. This “on-off” response can often be seen prior to the development of a detectable air-bone gap. Advancing stapedial fixation affects both the ipsilateral and contralateral acoustic reflexes.
Pure tone audiometry is always a part of the assessment and a gradually progressive low-frequency conductive hearing loss is first seen. As the sclerosis worsens and the footplate becomes fixed, a mass effect is added to the audiogram. This results in a stabilization of low-frequency thresholds, with worsening in the high frequencies and a gradual widening of the air-bone gap. The configuration changes from upward sloping to flat. Complete stapes fixation will give a maximum conductive hearing loss of 60 to 65 dB HL as long as there is not progression of the disease to involve the cochlea. Usually though, there is development of cochlear otosclerosis, and the loss becomes mixed or sensorineural. High frequencies are more severely affected at this point.

The audiometric hallmark of stapes fixation is the Carhart notch, characterized by elevation of bone conduction thresholds of approximately 5 dB at 500 Hz, 10 dB at 1,000 Hz, 15 dB at 2,000 Hz, and 5 dB at 4,000 Hz. It is important to realize that the Carhart notch is a mechanical artifact, not a true representation of cochlear reserve. Commonly, one can obtain “overclosure” of the air-bone gap after stapedectomy with postoperative air-conduction thresholds lower than preoperative bone-conduction thresholds.

Management

Hearing aids
All patients with otosclerosis should be offered the option of amplification during preoperative counseling. Patients refusing surgery or who cannot tolerate surgery should be considered for amplification. Patients with stapedial otosclerosis usually have excellent discrimination and hearing aids may provide effective treatment. Patients with advanced otosclerosis and mixed hearing losses in the severe range can obtain serviceable hearing with hearing aids after stapedectomy.

Medical treatment
Medical treatment is controversial and consists of fluoride therapy, vitamin D, and calcium supplements. This should be considered for any poor surgical candidates, patients who opt against surgery, those who are suspected of having cochlear otosclerosis, or patients with vestibular symptoms due to otosclerosis. Several well conducted studies have been performed which verify the efficacy of fluoride therapy. It is believed that the fluoride ion replaces the hydroxyl ion forming a more stable fluorapatite complex that resists breakdown by osteoclasts. Shambaugh recommends 60 mg of sodium fluoride per day to obtain the maximum bone calcifying effect. The drug is well tolerated with minimal side effects. Evaluation of efficacy can be based on the disappearance of Schwartze’s sign, stabilization or improvement in hearing, and improvement in the CT appearance of the otic capsule. In addition, sodium fluoride can be used after successful stapedectomy to prevent progression of sensorineural hearing loss.

Stapedectomy
Stapedectomy is the mainstay of treatment for otosclerosis. It was the first successful microsurgical operation and played an important part in the development and advancement of otology. More than 35 years after the introduction of stapedectomy, the surgical technique continues to evolve. Modifications are being introduced to better the hearing result with fewer complications. Polyethylene struts are no longer used owing to the frequency of incus erosion and the occasional slippage into the vestibule. Gelfoam has disappeared from use as an oval window seal because of the risk of granuloma formation. It has progressed to the point that H.P. House feels it has reached a “stage of perfection.”

The objectives of stapedectomy are: (1) to open the oval window for sound entry into the labyrinth; (2) to reconstruct a conductive bridge between the incus and the labyrinth; and (3) to accomplish these goals as efficiently and physiologically as possible for long-term hearing without complications.

When performing stapes surgery there is always a fenestration made into the vestibule, the variable is the size. The techniques vary from total stapedectomy to partial stapedectomy to stapedotomy. In addition, various surgeons report success with using instruments, drills, and lasers for their procedure. Recently there has been a shift to stapedotomy, as one can gain reportedly equal or improved hearing with fewer complications. Because of this recent shift, the procedural steps of stapedotomy will be discussed in this review. Other techniques include stapes mobilization, total stapedectomy, partial stapedectomy, and anterior crurotomy with posterior crus preservation. Most otologists can perform each of these procedures with great results and it is ultimately their decision as to which method to use. In addition, I have found in
my readings that many prominent otologists feel strongly that their preferred method is the optimal procedure. As stapes surgery has become refined, regardless of which procedure is used, there are very similar gains in hearing with a low incidence of complications. With larger fenestrations of the footplate there is a larger conductive advantage gained in hearing but the vestibule is more prone to trauma. This can translate to worsened postoperative vertigo or dysequilibrium which usually resolves shortly but is more uncomfortable for both the patient and surgeon. It is important to note that the stapes surgeon should be able to perform each of these different techniques as anatomic variations and intraoperative findings can prohibit certain techniques.

Both local and general anesthesia can be used for stapes surgery. The canal is injected using 1% lidocaine with 1:100,000 epinephrine to fully vasoconstrict the tympanomeatal flap. The flap is elevated between the 6 and 12 o’clock positions to allow for 5 to 6 mm of flap lateral to the annulus. The posterior canal wall bone is curretted with the chorda tympani being preserved whenever possible. The ossicles are inspected and palpated to establish the precise diagnosis of the conductive hearing loss. The distance between the undersurface of the incus and the stapes footplate is measured to determine prosthesis length. It is then customary to add 0.25 to 0.5 mm to this length to help prevent mobilization of the prosthesis. At this point in the operation the surgeon must decide which procedure will be performed as the initial approach is similar for all. In a carbon dioxide laser stapedotomy, the stapedius tendon and posterior crus are vaporized with a carbon dioxide laser. Lesinski has found that the laser will not vaporize the anterior crus so one must fracture it with crura crushers. The incudostapedial joint is separated and the stapes superstructure is removed. A stapedotomy opening is vaporized with the laser in the center of the footplate. A prosthesis is inserted into the stapedotomy opening and then attached to the neck of the incus. The incus is gently palpated to observe the motion of the prosthesis. Clotted blood can be used to seal the oval window and the tympanomeatal flap is returned to its anatomic position.

In addition to the types of fenestrations performed, there are other variabilities. The CO2, argon, and KTP lasers are all used in laser stapedotomies. They have varying characteristics that makes each useful. The KTP and argon lasers have visible wavelengths providing convenience. The CO2 laser needs another optical aiming beam transposed with it but has improved tissue characteristics. Also, some surgeons prefer to make their stapedotomy with a drill. There is also variability in the types of prostheses used and each surgeon has his preference. There has been a move away from wire and beveled prostheses as there was a higher complication rate associated. The issue of an oval window seal also varies. Many who perform stapedotomies feel that only a blood seal is necessary while others use perichondrium, vein graft, temporalis fascia, or fat. Gelfoam seals have been abandoned due to increased morbidity.

In the immediate postoperative period, the patient is asked to avoid lifting and straining for about 6 weeks. Nose blowing should be discouraged and one should sneeze with the mouth open if necessary. Water is kept out of the ear until the tympanomeatal flap is healed. A postoperative audiogram is obtained about 4 to 6 weeks after surgery.

**Intraoperative problems**

*Tympanic membrane perforation*

Tympanic membrane perforation usually happens as the tympanic membrane is being elevated from the posterior sulcus. Injury to the TM occurs 1.9% of the time and can be repaired by the underlay technique. Persistent perforations should be repaired within 4 to 6 weeks after surgery to prevent any problems with transcanal contamination and otitis media.

*Chorda injury*

Damage to the chorda tympani nerve may occur in up to 30% of cases and symptoms include dry mouth, tongue soreness, and a metallic taste that usually resolves in 3 to 4 months. Most authors state that less severe symptoms are reported with complete sectioning of the nerve than with stretching or partial tearing. To prevent injury, the bone overlying the chorda can be outfractured allowing mobilization of the nerve during the procedure.

*Malleus fixation*

Malleus fixation must always be kept in mind during stapedectomy and necessitates palpation of the manubrium during surgery. It can occur as a primary disorder or secondary to tympanosclerosis. The incidence of malleus fixation occurring with stapedial otosclerosis has been reported between 1 to 10% of
cases. Several clues can help differentiate primary malleus head fixation from otosclerosis preoperatively. Malleus fixation occurs later in life and the air-bone gap is seldom more than 30 dB and is more commonly 10-15 dB. The simplest method of management is to break the ossified ligaments in the epitympanum but this is associated with a high rate of refixation. Alternatively, an incus bypass procedure can be performed.

Incus problems
These include congenital abnormalities that cause problems with placement of the prosthesis. Incus necrosis may occur due to chronic otitis media or secondary to a previously placed prosthesis with erosion. Also, intraoperative dislocation or subluxation occurs in 0.2% of cases. Incus problems as a group constitute the most common indication for an incus bypass procedure. A surgeon who performs stapedectomy must be capable of performing the bypass technique because of these instances.

Tympanosclerosis
Tympanosclerosis will be the most common process masquerading as otosclerosis with a conductive hearing loss. Clues include a history of repeated bouts of otitis media and the presence of myringosclerosis. In addition, the conductive hearing loss is not as severe. Gidding and House found that the air-bone gap was less than 20 dB in 72% of patients and less than 30 dB in 90% of patients. If the tympanosclerosis progresses to involve the footplate, stapes surgery can be performed with good results although not as profound as in otosclerosis.

Solid or obliterated footplate
Extensive involvement of the oval window and footplate ranges from 7 to 11%. A “biscuit” (or solid) footplate is a more common finding than the completely obliterated footplate. Most authors state that a “drill-out” procedure or at least thinning to the footplate is required but this carries a significantly higher risk of SNHL (about 4%). Partial or complete reclosure of the oval window following primary stapedectomy may occur. This is considered to be a cause of early failure (within one year) and some authors advocate the use of sodium fluoride postoperatively in these patients. Use of CT before revision procedures when there was initial obliterative otosclerosis may detect this regrowth. Risks of reoperation and deafness must be weighed against the use of hearing aids without reoperation.

Floating or fractured footplate
Once a footplate has become mobilized in a stapedectomy, it is very difficult to remove without pushing it into the vestibule. In order to prevent this, it is recommended that control holes be made in the footplate prior to removing the stapes superstructure. These can be made with hand-held instruments or the laser. If the footplate has already been mobilized prior to making the control holes, the laser can be used at this point. With the control hole, one can get under the footplate withhooks or needles for removal.

Cerebrospinal fluid (Perilymph gusher)
A “gusher” or rapid, profuse flow of fluid immediately upon entering the vestibule occasionally is encountered. The fluid may completely fill the middle ear and enter the external canal. The perilymph volume is on the scale of microliters so this abundant flow represents CSF. Two sources are postulated: either a widened cochlear aqueduct or a defect in the fundus of the internal auditory canal. Causse and associates describe two clues that may tip the surgeon to the possibility of a gusher before the footplate is opened: an avascular congenital middle ear and an abnormally anterior insertion of the posterior crus to the footplate. It also more commonly seen in congenital footplate fixation and on the left side. Their method of management consists of immediate head elevation, creation of a small-hole stapedotomy, and placement of a large tissue graft prior to prosthesis insertion. Occasionally these patients will require a lumbar drain.

Bleeding
Intraoperative bleeding can be troublesome during the delicate parts of the stapedectomy. The most common cause of bleeding is mucosal trauma. Intraoperative bleeding can significantly hamper surgery during the hyperemic or active phase of otosclerosis. Because of this, some surgeons recommend treatment with sodium fluoride preoperatively in these patients to stabilize the lesion. Rarely, a persistent
stapedial artery may be found, which can cause considerable bleeding if traumatized. It may be coagulated with bipolar cautery or moved to one side with small-hole stapedotomy.

Prosthesis-oval window problems

A number of problems can occur including displaced prostheses, short prostheses, oval window fibrosis, and high membrane formation. A displaced prosthesis is the most common cause of failure, requiring revision surgery in the majority of cases. A displaced prosthesis is responsible for failures 30 to 82% of the time. The manner and frequency of complications is directly related to the type of prosthesis used. In many studies, the wire-gelatin sponge, which was the most common prosthesis initially, was responsible for the greatest percentage of problems. A short prosthesis is the cause of failure 8-9% of cases but is the most common cause of failure in cases in which footplate fragmentation occurred initially. Oval window fibrosis with subsequent reclosure can be an early or late problem and is related to mucosal trauma at the time of initial surgery and use of gelatin sponge.

Postoperative Complications

Sensorineural Hearing Loss (SNHL)

Partial or total SNHL is one of the most dreaded complications of stapedectomy. Many series describe the incidence as 0.2% to 10%. Depending on the degree of loss. Some experience this loss during the first few days after surgery while others note it months after the procedure. Causes are varied but surgical trauma is most often cited. Serous labyrinthitis is common and presents as mild unsteadiness, vertigo with head movement, slight decrease in hearing for frequencies above 2 kHz, and reduced speech discrimination. Symptoms usually resolve within a few days to weeks but sometimes takes months to resolve. By definition, serous labyrinthitis eventually resolves and one must consider other more serious complications if they do not or worsen.

Surgical trauma is the most common cause of permanent hearing loss following stapedectomy. Factors include extensive drilling, prior traumatic mobilization, hemorrhage, and surgical instrument trauma. Also, the cochlea becomes more fragile as otosclerosis progresses making the cases which require the most drilling and instrumentation the most prone to damage. Use of a laser has been associated with a lesser incidence of hearing loss.

Causse has also found that increased negative pressure in the middle ear can force the prosthesis into the vestibule and lead to a Meniere’s type response with tinnitus, imbalance or vertigo, and decreased bone conduction thresholds which are seen initially in the low frequencies. He manages these patients with early active aeration of the middle ear and steroids. Patients with some degree of endolymphatic hydrops preoperatively can have a sudden release of perilymphatic pressure. This will lead to tinnitus, vertigo, and a drop in all bone conduction thresholds but especially in the lower frequencies. Some of these patients will develop a total SNHL. It is recommended that any patient who has symptoms of endolymphatic hydrops within the prior 6 months should avoid surgery to prevent this complication.

Perilymph Fistula

Following stapedectomy, the potential for leakage persists until an endosteal membrane forms at the oval window. Also, a secondary fistula can form many years after apparently successful procedure. The symptoms classically consist of hearing loss, tinnitus, and vertigo or dysequilibrium but vary widely. The most common symptom reported is a drop or fluctuation in hearing. Use of a gelfoam seal has been associated with a high rate of perilymph fistulas and its use is discouraged. Fascia grafts have performed superiorly to other seals and are advocated by many authors. With these changes and the discontinuation of beveled and wire prostheses, the incidence of fistulas has decreased.

Reparative granuloma

Postoperative reparative granuloma has long been cited as a cause of sensorineural hearing loss following stapedectomy but its existence is controversial. It is reported to occur in 1-5% of cases. Classically, a patient has an initial improvement in hearing after surgery with either gradual or sudden deterioration 1 to 6 weeks postoperatively. Inspection of the TM demonstrates a reddish discoloration, especially in the posterosuperior quadrant. Bone conduction and speech discrimination worsens. This
problem should be suspected when the commonly seen symptoms of serous labyrinthitis, namely SNHL and dysequilibrium, persist beyond several days after operation or worsen with time. In general, this complication is found when either fat or gelfoam is used as an oval window covering. Immediate recognition and timely removal of the granuloma has been shown to offer excellent results of bone conduction thresholds along with the relief of vertigo and tinnitus.

Rare complications

Partial or total facial paralysis is a rare complication of stapedectomy. A dehiscent facial nerve may obscure the footplate but usually a small-hole stapedotomy can be performed. During revision surgery a dehiscent nerve can be embedded in fibrous tissue filling the oval window with unavoidable injury. Shea also described a “five-and-one-half day post-op facial palsy after stapedectomy” with an incidence of 0.1%. It is believed to be mediated by the herpes virus. It is associated with an uncomplicated case without trauma to the nerve. Complete recovery within 6 weeks regardless of treatment is the rule. Acute otitis media must be treated rapidly and aggressively to prevent bacterial labyrinthitis with meningitis and subsequent death. Cholesteatoma is also mentioned as an extremely rare complication.

Revision cases

The best chance of success is at the time of the initial procedure, with revision surgery successful in less than 80% of cases. Revision stapedectomy should be approached with extreme caution. Reports from the most experienced surgeons indicate that the risk of severe sensorineural loss (“dead ear”) is from two to ten times higher than with primary stapedectomy. They have shown that the air-bone gap is successfully closed to within 10 dB in less than half of their revision stapedectomy patients. Following revision surgery, 8% to 33% of the patients developed worse hearing. The incidence of significant postoperative SNHL is 3% to 20% in these series.

Hough states that the examination for possible revision stapedectomy should reveal: (1) the condition of the opposite ear and the relative function of the two ears in relation to adequate hearing; (2) the amount of the air-bone gap remaining; and (3) the absolute functional hearing by speech threshold and discrimination scores. If the primary surgery was done elsewhere, the operative reports should be carefully examined as they can give clues to the patient’s problem.

With all the information available, the surgeon must decide whether reoperation would improve the patient’s overall hearing perception significantly. One should seldom risk violating two axioms: “Do not operate on an only hearing ear unless it is useless, even with a powered hearing aid,” and “operate only on the poorer ear.”

Results

Most authors are able to obtain closure of the air-bone gap to within 10 dB of the preoperative bone-conduction level in 90% of their patients. There has been much debate regarding results of total stapedectomy vs. partial stapedectomy vs. stapedotomy. Recent literature has stated that you can get equal or better results with the stapedotomy technique (with fewer complications) and thus there has been a recent shift toward this procedure. Most consider stapedotomy to be technically easier to perform and with less potential damage to the vestibule.

Persson et al compared the techniques and found that partial and total stapedectomies had better improvement in hearing over the first three postoperative years at all frequencies except at 4 kHz. They closed the air-bone gap to within 10 dB in 94.0% of total stapedectomies, 83.9% of partial stapedectomies, and 82.8% of stapedotomies at one year. They found that there were better results at 4 kHz for the stapedotomy patients however. At three years postoperatively they found that the hearing in the stapedotomy group was more stable and concluded that due to this and better results at 4 kHz that the stapedotomy technique was superior. A problem with this study included the fact that the gain at 3 years in the stapedectomy group was still greater than that in the stapedotomy group.

Rizer and Lippy compared the three techniques with the only variable being the size of the fenestration in the oval window. They found that they achieved success (within 10 dB) in 97% of total stapedectomies, 96% of partial stapedectomies, and 87% of stapedotomies. They found that there was no significant difference at 4 kHz in regard to technique. Using these results they recommended the removal of a moderate portion of the footplate for three reasons: (1) a stapedotomy cannot be achieved in all cases;
(2) overclosure is slightly less common with stapedotomy than with stapedectomy; and (3) each technique achieves the same hearing result at 4 kHz.

It is important to note that most of the literature has been compiled by accomplished otologists who trained in an era when stapedectomy was a very common procedure (circa 1960-1980). The key to successful stapes surgery is to perform an adequate number of procedures to attain proficiency. In the 1980s, several studies were published regarding stapes surgery in residency programs. Backous et al were obtaining “success” in only 68% of patients with residents performing an average of 2.9 stapedectomies during their training. Other training programs during this same time period achieved “success” from 62 to 96% of the time with 0.78 to 18 procedures per resident.

In summary, one must decide if they are adequately trained in stapes surgery prior to entering the operating suite. They also must determine which technique is most successful in their hands and which best suits the patient’s disease process. One must discuss the rate of “success” in hearing improvement and the rate of complications in his or her own experience, not that quoted in the literature.

Bibliography


