Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 4: Inner Ear

Chapter 50: Surgical Treatment of Vertigo

Stephen L. Liston, Rick L. Nissen, Michael M. Paparella, Sady S. Da Costa

Vertigo can be a disabling symptom and the patient so incapacitated that relief is almost demanded. However, before considering any surgical procedure for the treatment of vertigo, there are several important principles to consider.

1. *A diagnosis of the condition causing the vertigo needs to be established.* In some instances - for example, Ménière's disease - the diagnosis depends mainly on the history. Even so, a thorough physical examination and the appropriate laboratory work as well as radiologic examinations should be performed. Electrical testing such as brain-stem-evoked responses and rotational testing may be required to accurately diagnose the cause of the vertigo. In a few cases, a surgical procedure will be part of the diagnostic workup, as for example in the case of a fistula of the round or the oval window.

2. *The majority of patients with disorders in balance are treated successfully with conservative medical treatment and reassurance.* Hence these patients should have an adequate trial of medical therapy before surgical intervention is undertaken. The length of time under medical treatment will vary, depending on the severity of the symptoms and the amount of disability experienced by each patient. Surgical intervention is offered to those patients who do not experience control of dizziness to their satisfaction and to whom the risks of surgery are acceptable. When accurate preoperative evaluation has been performed, surgical results have been gratifying and risks minimal.

3. *The risks of the surgical procedure and the alternatives to that particular procedure should be fully discussed with the patient.* For example, sectioning the vestibular nerve provides excellent results in controlling the vertigo but, even though they are rare, the potential complications are life-threatening. One other point should be emphasized, and that is that Ménière's disease may be bilateral in a high proportion of patients (Paparella and Griebie, 1984). If a destructive procedure has been performed on one ear and then the other ear develops Ménière's disease, the patient and the surgeon are in an unenviable position

Conditions Causing Vertigo

On a pragmatic basis, we can classify these conditions that cause vertigo that may require surgical intervention, according to whether they occur in the middle ear, inner ear, or internal acoustic meatus. There is also another group of conditions, such as vertebrobasilar insufficiency

and the subclavian steal syndrome, in which surgical correction of the vascular obstruction may relieve vertigo. Similarly, the vertiginous aura of epilepsy can sometimes be relieved by excising the irritative focus on the cerebral cortex.

Vertiginous Conditions Arising in the Middle Ear

Serous Labyrinthitis

Schuknecht (1974) defined this disease process as an irritation of the labyrinth caused by otitic or meningitic infection without bacterial invasion of the inner ear. It is presumed that toxic substances cross the round window and the oval window to affect the inner ear. Chronic otitis media may be associated with endolymphatic hydrops, presumably because of toxic substances crossing the round window membrane. Histologic findings in serous otitis media show endolymphatic hydrops and increased protein content, as evidenced by eosinophilic staining fluid or by serofibrinous bands (Sugiura and Paparella, 1967).

If vertigo occurs in association with a bulging, inflamed tympanic membrane, then a wide myringotomy should be performed. The contents of the middle ear should be cultured and tested for antibiotic sensitivity. In theory, serous labyrinthitis, in contrast to suppurative labyrinthitis, is due to irritation of the labyrinth and so the direction of the nystagmus should be toward the affected ear. In practice, the direction of the nystagmus is often not so clear-cut and the diagnosis of serous otitis media is established by the fact that some function is preserved in the inner ear after the disease has been treated.

Suppurative Labyrinthitis

In this condition, bacteria from the middle ear or meninges actually invade the inner ear. At first the inner ear contains bacteria and leukocytes, but the process may progress to fibroblastic proliferation and even to the formation of new bone in the labyrinth (Sugiura and Paparella, 1967). The patient becomes violently vertiginous and deaf in the involved ear. Theoretically the nystagmus is paralytic and therefore away from the involved ear but, again, in practice this may not be so clear-cut. High doses of antibiotics should be used and a wide myringotomy should be performed. In the preantibiotic era, the inner ear was opened widely to drain the pus and prevent meningitis, but such procedures are only rarely required now.

Cholesteatoma

Cholesteatoma can cause vertigo by eroding away the bony capsule of the inner ear and producing fistulas. The lateral semicircular canal is the most common site for such fistulas, but other areas of the labyrinth may also be involved. The presence of cholesteatoma is diagnosed on finding a positive fistula sign on otoscopy. A fistula makes the patient prone to vertigo from such activities as straining, sneezing, stooping, and sudden movements of the head. The Tullio phenomenon characteristic of a third window into the middle ear consists of vertigo produced by loud noises. The cholesteatomatous fistula may also be the site of entrance of bacteria to produce

serous or suppurative labyrinthitis. Granulation tissue in association with the cholesteatoma may also invade the inner ear (as may granulation tissue without cholesteatoma), and this may produce vertigo and hearing loss.

The techniques and surgical principles involved in the treatment of cholesteatoma are discussed in the chapter on chronic otitis media (Chap 29, this vol). Suffice it to say that vertigo in association with a cholesteatoma demands surgical management. A good deal of judgment is required in deciding the best surgical procedure, and each case should be decided on its own particular merits. Gacek (1974) listed four factors to be considered by the surgeon in the management of such fistulas: (1) the surgeon's ability and experience, (2) the location of the fistula, (3) the function available in the ear with the fistula and the function of the opposite ear, and (4) the mechanism of the erosion of bone by the cholesteatoma. Dealing with a cholesteatoma in an only hearing ear is a very perplexing problem.

The surgeon must decide whether to use an intact canal wall procedure versus a procedure that exteriorizes the matrix. Another decision is whether to remove the matrix of the cholesteatoma or to leave it in place. If the cholesteatomatous matrix is to be removed, the surgeon then decides whether this should be done initially or the procedure should be staged. In general, a cholesteatomatous matrix can safely be removed from a small fistula in the lateral, but in the case of a large fistula into the cochlea it may be safer to leave the matrix in place and exteriorize it. Removing the matrix, in any case, can cause a sensorineural hearing loss (Sheehy and Brackmann, 1979).

Perilymphatic Fistula: Ruptures of the Round and the Oval Window

Rupture of the round or oval window may occur due to changes in pressure (Goodhill et al, 1973). These changes may be implosive (from diving or flying) or explosive (associated with straining or sneezing) (Schuknecht and Witt, 1985). Rupture can also happen after head trauma (Lehrer et al, 1984). The patient experiences the sudden onset of hearing loss and vertigo. It should be noted that not all cases of sudden hearing loss are due to perilymphatic fistula; however, in patients in whom the history is suggestive of this etiology or in whom the symptom persist, consideration should be given to an exploratory tympanotomy. If a fistula is identified, it can be repaired with a graft of connective tissue.

Subluxation of the Stapes

Traumatic perforations of the tympanic membrane can be caused by accidents involving such objects as cotton-tipped swabs, hairpins, and twigs that have been poked into the external ear canal. In a small percentage of such injuries, the ossicular chain can be involved, and the stapes can be subluxed into the vestibule (Arragg and Paparella, 1964). An emergency stapedectomy should be performed and the stapes gently replaced in its normal position and supported with a graft of connective tissue, or the stapes can be removed and a prosthesis inserted. The tympanic membrane is repaired at the same time.

Otosclerosis

There are a variety of conditions associated with otosclerosis that can produce vertigo.

Post-Stapedectomy Granuloma

Granulomas were associated with the use of Gelfoam and other grafts to close the oval window. The treatment involved removing the granuloma and replacing it with connective tissue. Post-stapedectomy granulomas are now uncommon, because Gelfoam is not commonly used as a seal for the oval window.

Post-Stapedectomy Fistula

When a patient who has had a stapedectomy develops vertigo and a fluctuating hearing loss, the etiology is likely to include finding of a post-stapedectomy fistula. Such fistulas can occur immediately postoperatively or some time after the stapedectomy. The incidence of such fistulas varies according to the type of prosthesis and the type of seal in the oval window. The oval window should be re-explored under local anesthesia, and if the prosthesis can easily be removed this should be done and the oval window closed with connective tissue and a new prosthesis placed in position. If attempting to remove the prosthesis produces vertigo, then the edges of the fistula are freshened and the fistula is sealed with connective tissue. The use of a laser has made revision stapedectomy simpler (McGee, 1983; Gherini et al, 1988).

Otosclerotic Inner Ear Syndrome

In 1966, an association was noted between otosclerosis and vertigo (Paparella and Chasin, 1966; McCabe, 1966). McCabe coined the term "otosclerotic inner ear syndrome". The exact process by which the otosclerosis causes the vertigo is unknown, but in the majority of patients stapedectomy resulted in disappearance or improvement of the vertigo.

Otosclerosis and Endolymphatic Hydrops

Otosclerosis can be associated with endolymphatic hydrops (Sismanis et al, 1986). In fact, there is a high degree of suspicion that in some instances the otosclerotic focus can be an etiologic agent in the production of the endolymphatic hydrops (Johnsson et al, 1982; Brookler, 1984; Liston et al, 1984). It was previously thought that the hydrops saccule would be in contact with the footplate of the stapes, and this would result in an inevitable rupture of the saccule and in sensorineural hearing loss if a stapedectomy can be performed safely (Issa et al, 1983). Paparella has combined such a stapedectomy with a modification of the Cody "tack" principle (Paparella et al, 1984).

Conditions Arising in the Inner Ear

Positional Vertigo

Patients with this condition become vertiginous when the head is placed in a certain position. The vertigo can be induced by the Dix Hallpike test (1952). Schuknecht and Ruby (1973) postulated that this type of vertigo was due to the adherence of degenerated otoliths to the cupula of the posterior semicircular canal. The coined the term cupulolithiasis. Gacek (1974) described the technique of dividing the posterior ampullary nerve. This technique has continued to give good relief of benign paroxysmal positional vertigo (Gacek, 1984; Meyerhoff, 1985). Changes in the surgical technique have reduced the incidence of sensorineural hearing loss associated with this procedure (Gacek, 1984).

Møller and colleagues (1986) described what they call disabling position vertigo. They treated 21 patients by exploring cranial nerve VIII and found that in all the patients one or more arteries or veins were compressing the nerve. Sixteen patients were relieved of their symptoms after microvascular decompression of the nerve. No patients had any hearing loss, but one patient had a cerebellar contusion.

Ménière's disease

Ménière's disease was first described in 1861 when Prosper Ménière contributed six classic papers describing the clinical entity that bears his name. Clinically, the classic triad of Ménière's disease is well known and consists of episodic vertigo, fluctuating sensorineural hearing loss, and tinnitus. These are usually associated with pressure in the involved ear. Often patients do not present with these classic symptoms but have so-called atypical symptoms or patterns. Patients with vestibular Ménière's disease thus may present with only vertigo, and those with cochlear Ménière's disease may present with only hearing loss, with other symptoms occurring later in the disease process or not at all. In 1938, Hallpike and Cairns described the presence of endolymphatic hydrops in the temporal bones of two patients who had died as a complication of sectioning of cranial nerve VIII, which was done to treat Ménière's disease. To date, the chief pathologic correlate of Ménière's disease is endolymphatic hydrops.

From our recent studies it would appear that the etiologic agent in Ménière's disease is a multifactorially (racially, genetically) inherited predisposition to which environmental and individual factors have been added. The pathogenesis subsequently includes the development of endolymphatic malabsortion, and the symptoms are best explained on both a physical and chemical basis. The diagnosis can be made through a careful, detailed history in 90 per cent of cases. Audiograms first and vestibular studies and mastoid x-rays secondarily are helpful. Gibson and Prasher, 1983 have suggested that the summating potential/action potential ratio as measured by electrocohleography may be useful in the diagnosis of Ménière's disease. A trial of clinical treatment with a special emphasis on psychological support should always be the first step of treatment. If, despite and adequate trial of medical therapy, the patient still complains of disabling symptoms, then surgery is considered. For more details on Ménière's disease the reader is referred

to the previous chapter (Chap 49).

Surgical Treatment of Intractable Ménière's Disease

As the preceding chapter indicates, Ménière's disease is the most important and prevalent among the peripheral diseases, and in a sense serves as a model for diagnosis of the other diseases in which the chief symptom is vertigo. Similarly, the history of the surgical treatment of vertigo is based on the surgical treatment of Ménière's disease; thus treatment of this condition is highlighted in this chapter. It has been estimated that approximately one out of four patients seen with Ménière's disease may eventually be a candidate for surgical therapy.

Regarding preservation of hearing, labyrinthine surgery may be either conservative or ablative (destructive). Conservative procedures have as their aim preservation and if possible improvement of labyrinthine function. These include procedures on the endolymphatic sac (decompression and enhancement), sacculotomy, sympathectomy, and possibly cochleosacculotomy. A number of partially destructive (ablative) procedures are possible in treating Ménière's disease; their purpose is to destroy selectively or inactivate the vestibular labyrinth and thus abolish vertigo. They include sectioning the vestibular nerve, ultrasonography, cryosurgery, and chemical ablation by instillation via the middle ear of ototoxins. Procedures that totally destroy the labyrinth and thus the hearing include labyrinthectomy and translabyrinthine sectioning of the vestibular nerve (Table 1).

Table 1. Surgical Treatment of Intractable Ménière's disease

I. Conservative procedures (hearing preserved)
- A. Extralabyrinthine
  1. Endolymphatic sac enhancement
  2. Endolymphatic sac revision
  3. Sympathectomy
- B. Invasive of the labyrinth
  1. Sacculotomy
  2. Cochleosacculotomy
  3. Stapedectomy-sacculotomy

II. Partially destructive procedures (hearing preserved)
- A. Vestibular nerve section
  1. Through the middle fossa
  2. Retrolabyrinthine approach
  3. Retrosigmoidal approach
- B. Singular neurectomy
- C. Ultrasonic irradiation of the labyrinth
- D. Cryosurgery
- E. Exploratory tympanotomy with medical ablation

III. Destructive labyrinthectomies (hearing destroyed)
    A. Transtympanic
        1. Labyrinthectomy extended to the oval window
        2. Transcanal labyrinthectomy
        3. Transmeatal labyrinthectomy
    B. Transmastoid
        1. Transmastoid labyrinthectomy
        2. Translabyrinthine section of the vestibular nerve
        3. Horizontal canal labyrinthotomy
    C. Labyrinthectomy through the middle fossa.

Our Policy for Intractable Ménière's disease

The two primary surgical procedures for Ménière's disease are endolymphatic sac procedures and vestibular nerve sections. An important year in the history of treatment of Ménière's disease was 1928, for in that year Portmann described the first endolymphatic sac procedure. Also at that time Guild described the "longitudinal flow" of endolymph or "out-processing" toward the endolymphatic sac. Dandy, in 1928, described transection of the vestibular nerve as treatment for Ménière's disease. We can all thank House, who repopularized both of these methods, which have stood the test of time and have benefited many patients to date.

Our philosophy and policy is that conservative treatment comes first. Medical treatment with psychological support precedes any consideration of surgery. If deafness and/or vertigo become(s) intractable, then a conservative surgical procedure, endolymphatic sac enhancement/shunt, can be considered. We consider endolymphatic sac enhancement to be "an extension of conservative treatment" because it has minimal risks and appears to affect pathophysiology.

In our opinion, endolymphatic malabsorption is the fundamental pathogenetic factor in Ménière's disease and a sac procedure helps counteract this. The chance of retaining hearing is excellent and in some patients hearing is improved. To date, in more than 1000 endolymphatic sac enhancements done by one author (MMP), the main complication has been deafness, which occur in approximately 2 per cent of patients postoperatively. Wound infection was our chief complication, and it has since been resolved. There have been no deaths, no permanent facial paralysis (one patient had temporary paralysis), and otherwise no serious complications.

If a patient experiences a good result (relief of vertigo) for a period of time and then presents with a recurrence of vertigo, an endolymphatic sac revision can be considered. The opportunity with these patients for vestibular nerve section for vertigo is discussed. The risks, complications, and percentage chance for permanent relief of vertigo is given for each procedure. We feel that each surgeon must assess his or her own individual percentage of success to be presented to the patient, and this percentage will weigh heavily in that particular surgeon's recommendations. It is our feeling that a well-educated patient should have a strong influence in the decision for revision versus nerve section. If deafness recurs without significant vertigo,

an endolymphatic sac revision is considered.

Vestibular nerve section, whether done via the translabyrinthine, retrolabyrinthine, or retrosigmoidal approach or using the approach through the middle fossa, represents an intracranial procedure with attendant risks. We do not consider the procedure the first choice in treating vertigo from Ménière's disease; it always requires a routine admission to the intensive care unit for close observation postoperatively. Potential intracranial risks include leakage of cerebrospinal fluid, meningitis, and death. Nevertheless, this is a good operation for intractable vertigo of peripheral origin and can be done safely and efficaciously in well-trained hands. A review by McElveen and associates (1984) of 52 patients undergoing retrolabyrinthine vestibular nerve section showed no deaths, three cerebrospinal fluid leaks, and one case of meningitis that resolved with medical therapy. There were no cases of facial weakness or dead ears. An evaluation of more than 200 cases is now being performed (McElveen, personal communication, 1986) with similar findings for complications. Most neurotologists quote success rates for relief of vertigo of 90 to 95 per cent or better, depending on the specific approach used.

Currently our policy also includes trying to avoid labyrinthectomy as much as possible, even in deafened ears, because (1) the contralateral ear can become involved or deaf, and (2) a possible future cochlear implant will be compromised by labyrinthine trauma and scar tissue. To summarize our view, in the treatment of Ménière's disease:

1. The most important medical management is psychological support, which includes explaining the disease to the patient and describing the various expectations regarding the natural history of the disease and/or its therapy.

2. If vertigo is uncontrolled with medical management, an endolymphatic sac enhancement/shunt is the initial surgical procedure considered.

3. If symptoms recur, an endolymphatic sac revision procedure and vestibular nerve section are considered, and each case is individualized.

4. We do not recommend sectioning the vestibular nerve as the initial surgical procedure, although it is a very good alternative (success rate of 95 per cent) in those cases in which procedures to enhance the endolymphatic sac fail.

Conservative Procedures

Endolymphatic Sac Surgery

Historical Considerations

> The second major step in the story of Ménière's disease after Ménière had identified it as a labyrinthine disorder in 1861 was the research into the endolymphatic sac that was carried out in my laboratory from 1921 to 1926. This research revealed the role played by increased pressure within the labyrinth and led to the first operation to decompress the endolymphatic sac on February 26, 1926. The era of endolymphatic surgery had begun.
>
> - Georges Portmann

Portmann's first procedure was performed with mallet and gouge and the sac was simply incised with a small knife. In 1965, Portmann described excellent results in long-term relief of vertiginous symptoms with his procedure. The operation had, however, been used relatively infrequently even by Portmann, and reports by other otologists (W. F. House, 1962) were often not enthusiastic about the procedure's value. The operating microscope and modern otologic techniques made Portmann's operation much safer, and interest in the concept of drainage from the sac was revived by House (1962). In House's technique, the inner wall of the sac was incised as well. A communication was created between the endolymphatic sac and the subarachnoid space, and a Teflon tube was placed into this aperture to ensure its patency. His early success with this technique showed satisfactory results; thus House abandoned simple drainage in favor of this shunt surgery.

Naito (1962) created a permanent fistula between the interior of the sac and the subarachnoid area. Shea in 1966 reported his series for which a Teflon wick had been placed from the sac to the mastoid cavity; he believed the results were promising. Shambaugh, Jr, (1966) subsequently noted that even when he was unable to identify the sac, simple decompression of the dura around the sac seemed to effect satisfactory results. Plester's (1972) technique included a large incision in the sac through which a triangular piece of Silastic was inserted and the sac covered with a free graft of muscle.

The first inner ear valves were implanted in 1975 and 1976 in Sweden by Stahle and co-workers and in the USA by Arenberg, who developed a one-way valve placed into the endolymphatic sac with a limb of Silastic sheeting extending into the mastoid. Morrison (1975) popularized the capillary endolymphatic shunt in a large series of patients. In this technique, a capillary tube is inserted into the lateral end of the endolymphatic duct with its distal tip inserted into a Silastic sponge. Paparella and Hanson (1976) described their method, which included with exposure of the dura, avoiding the skeletonization of the posterior semicircular canal and draining the sac via a T-tube. Kitahara's (1985) method of drainage was based on an intramastoid opening of the endolymphatic sac and folding back of the lateral wall of the sac with an insertion of Gelfoam into its lumen.

In 1987, Brackmann reported his results with use of an endolymphatic subarachnoid shunt and an endolymphatic mastoid shunt, showing no statistical differences between the two procedures. A double-blind study by Bretlau and colleagues (1980-1981) compared a group of patients who underwent endolymphatic shunt operations with a group receiving "placebo" (regular mastoidectomy) operations. According to their conclusions, although there was a tendency toward greater improvements in the group receiving shunts than in the group receiving "placebos", a statistical analysis revealed no significant differences. Paparella and Hanson (1976) had reported three patients in whom endolymphatic drainage was obviated by their anatomy, and vertigo was not affected, after the surgery. These patients were not aware that the drainage procedure had not been done: thus these inadvertently "sham" procedures had no effect on the symptoms of this small control group. Also, Pillsbury and co-workers (1983) reanalyzed the data of this important and controversial study and pointed out some flaws in the way in which the data were presented. Shea and associates (1979), through detailed examination and measurements from 40 temporal bones, established reference points by which the conservative surgeon can approach the endolymphatic sac below the level of the posterior semicircular canal. These observations make it unnecessary to risk exposure of the blue line of the posterior canal, thus minimizing risk of labyrinthine injury.

Endolymphatic Sac Enhancement

Concepts of Pathogenesis

Ménière's disease may have a progressive course, often leading to an intractable state, or it may persist as a nonprogressive condition. Medical management should be used to determine progressive versus non-progressive forms. If Ménière's disease with incapacitating vertigo and/or hearing loss progresses and becomes intractable in spite of medical management, endolymphatic sac enhancement can be considered. This procedure on the temporal bone has few complications and may be done prior to an intracranial procedure holding greater risk.

We currently find that in cases of Ménière's disease, endolymphatic absorptive dysfunction (of the endolymphatic duct or sac) can lead to hydrops, just as obstruction of the endolymphatic sac in animals does lead to hydrops (Kimura, 1967). The obstruction can be physical or functional. Gross anatomic pathologic findings in patients with Ménière's disease include decreased mastoid and periaqueductal pneumatization and anterior displacement of the lateral sinus, which reduces Trautmann's triangle. Such developmental changes likely influence the endolymphatic absorptive system later in life, leading to symptoms of Ménière's disease.

When surgery on the endolymphatic sac ameliorates symptoms and findings, it is logical that it does so by reversing function obstruction to the absorption of endolymph. We believe endolymphatic sac enhancement enhances longitudinal flow. This represents the only procedure that has the potential to reverse the pathogenesis of Ménière's disease.

We consider this surgical option for patients with intractable Ménière's disease, typical or atypical. The primary indications are vertigo and vestibular dysfunction; a secondary indication

is deafness. The results are much better for vertigo than for improvement of hearing; however, there is evidence to suggest that approximately one patient of three does have improvement of hearing. The likelihood of improving vertigo or vestibular upset is 90 per cent, of eliminating vertigo or vestibular upset is 70 per cent, of retaining hearing is 90 per cent, and of improving hearing is 30 to 40 per cent; the risk of deafness is 2 per cent, and this is usually related to infection of the wound and aditus block syndrome.

Surgical Technique

The method of endolymphatic sac enhancement has been modified since previous publications (Paparella and Hanson, 1976; Paparella and Goycoolea, 1981). Current surgical principles and steps are summarized as follows:

1. A curvilinear incision in the skin is placed at least on inch behind the postauricular crease. Then a rectangular flap of periosteum attached to the concha is shaped within the exposed field. By using a triple-layered closure (periosteum, subcutaneous tissue, and skin), the incisions in skin and in periosteum made in different locations, healing in the wound is aided and the likelihood of infection in the wound is decreased. Also, this method helps avoid postoperative depression of the skin into the mastoid cavity. The posterior incision also helps obviate scar tissue, which can grow from the wound into the region of the sac, especially when the incision is in the inferior postauricular crease, adjacent to the sac.

2. A *complete* simple mastoidectomy is done, and the aditus is widened. The incus is always exposed (the only exception being when there is a lack of mastoid aircells). The aditus ad antrum is opened widely to expose the head of the malleus. The tegmen mastoideum and mastoid tip are exposed, the posterior bony wall of the canal is thinned, and the depth of drilling in the mastoid cavity is *never* extended below the dome of the horizontal semicircular canal. The purposes of this are to gain good exposure, especially for later in the procedure; to be able to see the incus and horizontal semicircular canal for orientation; and to make possible later measurements - drilling below the dome of the horizontal canal endangers the posteroinferior semicircular canal. The enlarged aditus helps promote drainage and transfer of air between middle ear and mastoid and helps avoid a postoperative aditus-block syndrome.

3. Using the fenestrometer, measurements are made from the fossa incudis 10 mm along the axis of the horizontal semicircular canal and 12 mm (approximately 45 degrees) from the linea temporalis. The zone of the solid angle (containing the canals) is demarcated so that further surgery will not enter this zone. These measurements, based on earlier anatomic dissections (Paparella and Hanson, 1976) create a demarcated zone to protect the canals and to serve as a landmark for further dissection.

4. The lateral sinus is skeletonized and decompressed throughout its length in the mastoid. Bone over Trautmann's triangle is thinned and removed with mastoid curets or a rongeur. Since the lateral sinus is characteristically prominent in an anterior and medial location in Ménière's disease, and is often associated with hypopneumatization of air-cells and a small or nonexistent

Trautmann's triangle, decompression of the lateral sinus enhances subsequent decompression of Trautmann's triangle and the contiguous dura below the solid angle.

5. Immediately below the demarcated bony zone, an infralabyrinthine cell tract is searched for. Purposeful exaggerated drilling of bone is done toward the jugular bulb to expose as much dura as safely possible in this area. Often infralabyrinthine cells do not exist, and this exposure is done through solid bone. In restrictive mastoids (sclerotic or diploic), the anteriorly located facial nerve should be watched for and avoided. The purpose is to expose infralabyrinthine dura because the main body of the sac and its lumen often lie within this area and not posterior to the posteroinferior semicircular canal. This also helps dural decompression.

6. The dura contiguous with the decompressed lateral sinus is firmly decompressed (pushed down), especially below the solid angle. Care must be taken not to traumatize dura above the sac, which is often thin; this can lead to spinal fluid drainage. Sometimes a bony shelf below the dura (intracranial side) hampers complete decompression of the dura. Because of anatomic factors, the dura is often tight in this region, and decompression counteracts this tightness and assists sac enhancement later.

7. The sac is entered beneath the solid angle. Sac epithelium is visualized. A Whirlybird drill or other instruments help to bluntly and easily identify the lumen, especially in the infralabyrinthine region. The entrance to the sac should not be opened to the mastoid cavity so as to prevent fibrous or granulation tissue from invading the sac.

8. One or two Silastic T-struts are placed within the sac. Exact width and length depend upon size of the lumen. The tail of the strut is placed outside the sac between dura and bone. "Spacers" or strips of Silastic, as many as possible, are folded above and below the sac between the dura and the bone to decompress the dura and contiguous lateral sinus permanently. All silicone is placed or contained between bone and dura and not in the mastoid. Rubber strips and spacers serve as a soft, springlike mechanism for decompression of dura and sac. T-struts within the lumen enlarge the lumen and help the passive transfer of nanoliters of endolymph. It is important to open and treat the lumen of the sac, as well as the surrounding region. This observation is emphasized during revisional sac surgery.

9. A Silastic "apron" is placed to cover this region, followed by a large piece or two of Gelfoam, dipped in a steroid antibiotic solution and loosely placed to hold the "apron" in place. The purpose here is to help keep fibroblasts from invading the region of the endolymphatic sac. Previous techniques using pedicle grafts, temporalis fascial grafts, and protection by gold foil have been replaced by this more effective and simpler method.

10. As was done before opening the lumen of the sac, all bone dust and debris are removed meticulously. Bone dust can enter the middle ear through the aditus and should be irrigated out and removed. All bleeding is stopped and the wound is closed using a triple-layered closure: first the periosteal flap, then the subcutaneous tissue, and finally the epithelium. The purpose is to ensure healthy, rapid healing and to avoid infection of the wound.

11. A ventilation tube is placed in the tympanic membrane, and the middle ear is suctioned through this site. The ventilation tube may not be used if drainage of cerebrospinal fluid occurs. The goal is to promote drainage of operative fluids from the middle ear and mastoid in the immediate postoperative period (2 months). In addition, this promotes ventilation to the middle ear, and especially the mastoid via the enlarged aditus, so as to discourage formation of tissue in the mastoid, both in the short term and long term, and in particular to help avoid aditus block and long-term formation of tissue (granulations, scar, and bone) around the sac, which can lead to subsequent recurrent symptoms and signs of Ménière's disease. The tube has also been helpful in preventing otitis media and barotrauma from flying. The ventilation tube usually extrudes spontaneously in a year or so and is not replaced. The patient's symptoms are not affected when the tube extrudes.

Results

During the past 21 years, approximately 1000 endolymphatic sac enhancement procedures have been performed, usually for intractable vertigo and classic Ménière's disease, sometimes for atypical intractable Ménière's disease, and including 5 per cent revisions to date. In any assessment or comparison of results, the patient profile within the study (entry criteria) is just as important as the method of treatment used. For example, many surgeons prefer to operate early in the course of Ménière's disease, although our policy is more conservative. In our preceding reports, the average duration of disease was six years prior to sac enhancement (Paparella and Hanson, 1976; Paparella and Goycoolea, 1981).

For this report, we randomly selected 100 patients whose chief complaint was vertigo and whose speech reception thresholds ranged between 30 dB and 60 dB; we had at least a 1-year follow-up. We excluded patients with vestibular Ménière's disease, cochlear Ménière's disease, syphilitic Ménière's disease, otosclerosis and Ménière's disease, delayed Ménière's disease in a deaf ear, and so forth. The average age was 43 years. Males (56) slightly exceeded females (44) in number. Our results are displayed in Table 2 using the American Academy of Ophthalmology and Otolaryngology (AAOO) 72 criteria and American Academy of Otolaryngology - Head and Neck Surgery (AAO-HNS) 85 criteria.

Table 2. Results of Endolymphatic Sac Enhancement Procedures in 100 Randomly Selected Patients

AAOO 72 Criteria 1

"Best"*					"Worst"*			
Preoperative Audiogram								
A	**B**	**C**	**D**		**A**	**B**	**C**	**D**
36%	**56%**	**3%**	**5%**		**41%**	**51%**	**3%**	**5%**

AAO-HNS 85 Criteria 1 Compared With "Best" and "Worst" Preoperative Audiogram**

Class of Vertigo		**"Best" Hearing Class**		**"Worst" Hearing Class**		**Class of Disability**	
V0	72%	H1	36%	H1	42%	D0	56%
V1	12%	H2	56%	H2	50%	D1	26%
V2	8%	H3	8%	H3	8%	D2	8%
V3	8%					D3	0%
V4	0%						

* AAOO( ): 72 criteria are interpreted as follows in order to incorporate results for both hearing and vertigo: Class A refers to absence of vertigo, hearing improved; B refers to absence of vertigo (34% for "best" or 21% for "worst" category). The remaining percentage under B refers to vertigo controlled; B also refers to hearing unchanged; C refers to worsening of hearing but improvement of vertigo; D refers to worsening of hearing and failure of control of definitive spells.

** AAO-HNS: V0 = complete control of definitive spells; V1 = substantial control of definitive spells; V2 = limited control of definitive spells; V3 = significant control of definitive spells; V4 = worst control of definitive spells.

Complications

Complications can occur during or subsequent to surgery. General comments cover all patients operated on to date. During surgery, bleeding, particularly from the lateral sinus, and drainage of spinal fluid are the major complications. First pressure with Gelfoam soaked in adrenaline, followed by adrenaline tapes, can be used to control the bleeding. If drainage of spinal fluid occurs, the patient's head is elevated and the area is packed with Gelfoam. Occasionally mastoid air-cells are nonexistent, and therefore an antrum and the landmarks around the aditus cannot be identified. In such a case, the lateral sinus is decompressed, the contiguous dura and lateral sinus are pushed down, and the sac is treated as described above. Although the method described demarcates and protects the solid angle, inadvertent opening of the posterior or horizontal canal nevertheless should be avoided and anatomic variants looked for.

After surgery, the most serious complication is severe sensorineural hearing loss or "dead" ear, which has occurred in approximately 2 per cent of patients to date. In this regard, postoperative infection of the wound has been our chief nemesis, leading to a revised method of treating the wound, as described. Infection of the wound not only can encourage scar tissue immediately over the sac but can also spread to the labyrinth. Conductive hearing losses are seen in a few patients due to bone debris and other mastoid contents that enter the middle ear. In certain patients, exploratory tympanotomy and removal of bone in the region of the footplate have helped to correct conductive hearing loss.

Symptoms of Ménière's disease may recur after an initially good result, usually several years later. This is due to postauricular depression of the wound, and to bone and scar tissue that grow into the extrasaccular region, causing obstruction. We have had no other serious complications to date. We have had no deaths and only one temporary case of palsy of the facial nerve.

Endolymphatic Sac Revision

A subset of patients with intractable Ménière's disease have a good result for months or years following endolymphatic sac enhancement, only to develop recurrent symptoms of Ménière's disease, including vertigo and deafness. In these cases, as we stressed before in this chapter, we consider an endolymphatic sac revision and vestibular nerve section. The decision is made by the patient after careful discussion of the pros and cons of both procedures. To date, endolymphatic sac revisions have been done for 5 per cent of our patients who have had endolymphatic sac enhancements. In these cases, we have a unique opportunity to study Ménière's disease in patients, thus making it possible for us to illuminate new points of its pathogenesis and treatment.

Our revisional procedures have revealed extrasaccular fibrosis or granulation tissue, osteoneogenesis, and aditus bloc syndrome resulting in saccular obstruction and a tight contiguous dura. A recent case is illustrative: a 36-year-old man developed intractable Ménière's disease, characterized by incapacitating and frequent bouts of vertigo, fluctuating sensorineural hearing loss, pressure, tinnitus, and intolerance for loudness, in the left ear. This had grown progressively worse for seven years. A sac enhancement procedure was done on the left ear in January of 1981. Subsequently, the hearing in the left ear improved. The patient was free of vertigo for more than 4 years, until 1985, when disabling vertigo and other symptoms of Ménière's disease recurred. Along with vertigo, the patient developed actual deafness (not hearing loss), confirmed by the live voice and tuning-fork tests plus three separate audiograms.

At this point, the following procedures were considered: revision of the endolymphatic sac enhancement and vestibular neurectomy. Endolymphatic sac enhancement was chosen by the patient and was done in September, 1985. Trautmann's triangle was characteristically reduced, with copious amounts of bone and scar tissue from the wound growing into the region around the sac. The infralabyrinthine body cell tract was aggressively enlarged, and bone and scar tissue were removed. After revision, the patient again was vertigo-free and, surprisingly, the hearing

improved to a level that surpassed the original preoperative level. Discrimination in that ear became 100 per cent, with a speech reception threshold of 40 dB.

We have made similar but not so dramatic observations in other patients who received revision procedures. Pressure and obstruction recur in the region of the sac postoperatively, owing to extrasaccular fibrosis and formation of bone. After this obstruction is alleviated and the absorption of endolymph is once again enhanced, most patients return to a symptom-free state. Exaggerated attempts are made to prevent further formation of bone and scar tissue.

This subset of patients provides a built-in control with which we can compare the "before" and "after" of a pathologic, iatrogenic lesion causing malabsorption of endolymph and symptoms of Ménière's disease. Upon reversal of that lesion (enhancing absorption of endolymph), improvement of Ménière's disease and its symptoms often occurs. Results are better for vertigo and deafness from endolymphatic sac *revision* than from initial endolymphatic sac enhancement procedures. This is because the patient has had a good result from the primary procedure, and then iatrogenically induced bone and scar tissue have recreated the pathogenesis years later; correction of these iatrogenic secondary lesions provides a better chance for a beneficial result than with the initial procedure.

Consideration Concerning Why Enhancement of the Endolymphatic Sac Works

We hypothesize that the ability of the endolymphatic sac to absorb endolymph is enhanced because of (1) decompression, (2) passive diffusion, (3) osmotic pull, and (4) alteration of blood supply. There are certain surgical objectives designed to enhance absorption of endolymph. The first, and perhaps most important, is decompression. Decompression is done in a number of ways, including complete simple mastoidectomy and removal of bone widely over Trautmann's triangle, the lateral sinus, and especially the area of the infralabyrinthine cell tract. The dura is decompressed from the bone with concentrated effort and Silastic spacers are used to separate the dura softly from the bone.

In addition, passive diffusion of endolymph is allowed to take place along inert alloplastic surfaces of material placed in the expanded lumen of the sac. At the time of revision, we usually find that the silicone sheeting (T-strut) within the sac is yellow, suggesting extracellular transudation. Thus, it is likely that this procedure creates an extracellular electrolytic environment of sodium ions, which in turn creates an osmotic pull on endolymph in the opposite vestibular end of the endolymphatic duct, which is high in potassium. This same principle applies to other methods, such as the subarachnoid shunt, in which spinal fluid (similar to extracellular fluid) enters and bathes the lumen of the sac. Another surgical principle that likely enhances absorption of endolymph is creation of an altered or collateral blood supply.

Cervical Sympathectomy

The cervical sympathectomy was first used by Mogan and Baumgartner in 1934 in a case of Cogan's syndrome, with good results in the relief of vertigo. The aim of this procedure is to interrupt the distribution of the sympathetic nervous system to the head, thus enhancing the blood supply to the labyrinth. The procedure may be preceded by blockage of the stellate ganglion, although failure to obtain amelioration of the symptoms by that means is not necessarily a contraindication to sympathectomy. The sympathectomy technique includes exposure of the cervical chain and (usually) uncovering of that portion from the third thoracic ganglion to just above the stellate ganglion. The main indication has been for patients with bilateral disease in which the better ear is believed to be the one which is active in causing the symptoms.

Conservative Procedures Invasive of the Labyrinth

Sacculotomy

This procedure represents an attempt to decompress the endolymphatic system via a puncture of the saccule through the oval window. Indications for it are mainly seen in patients with Ménière's disease with disabling vertigo. Two techniques have been described and promoted: one by Fick (1964) and another by Cody (the "tack" procedure) (Cody and McDonald, 1983). Both procedures use a transcanal approach via a tympanomeatal flap.

After good visualization of the stapes and footplate is obtained, a fenestration is made into the footplate, in order to rupture the saccule. In the Fick procedure, a sharp pick is passed deeply into the vestibule to create a fistula within the saccule. Gelatin or connective tissue is then used to cover the fenestra in the oval window. The Cody tack procedure attempts to create a more permanent fistula in the saccule by placing a permanent tack through the fenestra in the footplate. Cody reported successful control of vertigo in 86 per cent of patients, with 5 per cent having improvement in hearing and 78 per cent having improvement in tinnitus. Cody reported successful control of vertigo in 79 per cent (290 patients), with hearing loss occurring in 35 to 40 per cent of patients. Unfortunately, these results have not been reproduced by other investigators. With reports of early recurrence of vertigo and postoperative loss of hearing, these procedures have not found widespread acceptance.

Cochleosacculotomy

Schuknecht introduced the cochleosacculotomy procedure in 1982. Prior experimentation had demonstrated that a penetrating injury to the cochlear duct associated with fracture of the osseous spiral lamina was apt to lead to nonhealing and permanent fistulization (Schuknecht and Neff, 1952; Schuknecht and Sutton, 1953; Schuknecht and Seifi, 1963). The procedure (Schuknecht, 1982) is performed under local anesthesia with initially a tympanomeatal flap to gain adequate exposure of the oval and round windows. The round window niche is removed with small cutting burs, allowing full visualization of the round window membrane. A 3-mm right-angled pick is then introduced through the round window membrane in the direction of the

oval window and passed to the full depth of 3 mm. This passage will carry through the cochlear partition, causing a fracture disruption of the osseous spiral lamina and cochlear duct. The tip of the pick should lie at the center of the oval window with rupture of the dilated saccule.

Stapedectomy-Sacculotomy

We have had opportunities to treat patients in whom otosclerosis and Ménière's disease coexist. In these cases, a stapedectomy is followed by replacement using a prosthesis made of wire and connective tissue that is usually 4.5 mm long from the top of the loop to the bottom of the graft. The tip of the wire is sharpened on the bias and extended 0.75 to 1 mm from the bottom of the graft to decompress the saccule. A central hold should always be made through the footplate prior to removal of the footplate, to avoid labyrinthine trauma from saccular decompression and to avoid sudden removal of a portion of the saccular wall attached to the footplate in certain decompensated cases.

Partially Destructive Procedures

Vestibular Nerve Section

Historically not a new idea, sectioning of the vestibular nerve has been attempted by neurosurgeons such as Frazier (1912) and Dandy (1941) since as early as the turn of the century. Rates of cure for vertigo were reported to be as high as 90 per cent, but the unacceptably high rates of dysfunction of the facial nerve and of loss of hearing prevented widespread acceptance of the procedures.

In the early 1960s, William House (1961) developed the middle fossa approach for selective sectioning of the vestibular nerve. With the aid of the operating microscope, he was able to obtain high rates of cure (> 90 per cent) while preserving both hearing and function in the facial nerve. The disadvantage of the procedure was that it was an unfamiliar approach for the otologist and required retraction of the temporal lobe. In 1980, Silverstein and Norrell introduced the retrolabyrinthine approach for sectioning the vestibular nerve, with resulting cure of vertigo in several series of 95 per cent or better (McElveen et al, 1984; Kemink and Hoff, 1986). We believe that this has become the most widely used approach for sectioning of the vestibular nerve. In 1985, Silverstein and colleagues (Silverstein et al, 1987) began using a retrosigmoidal approach with drilling of the posterior lip of the internal auditory canal in an attempt to obtain a more complete section of the vestibular nerve. They are currently analyzing these results in comparison with results of retrolabyrinthine section. These three procedures will be discussed briefly; the goals of all three are to denervate the vestibular end-organ while preserving residual hearing. Intraoperative monitoring of the facial nerve (Harner et al, 1986) and of the cochlear nerve (Sabin et al, 1987) has improved the safety of these procedures.

The most common problem treated by neurectomy is Ménière's disease when disabling vertigo is uncontrolled by more conservative means. Other causes of peripheral vestibular dysfunction such as trauma, postoperative damage, uncompensated vestibular neuritis, and so

forth, are also indications for neurectomy. A reproducible and significantly reduced vestibular response on testing by electronystagmography is a valuable preoperative diagnostic tool for recommendation of a vestibular neurectomy in these other, non-Ménière's cases.

Sectioning the Vestibular Nerve Through the Middle Fossa

This procedure (Glasscock et al, 1984) is performed with the patient supine and the diseased ear up. The surgeon sits at the head of the table. A vertical incision begins about 1 cm anterior to the tragus and is carried vertically. A rectangle of bone (approximately 4 x 4 cm) is removed from the squamous portion of the temporal bone. The temporal lobe and dura are then carefully elevated, exposing the floor of the middle cranial fossa. Using a high-speed drill, the internal auditory canal is opened, superior and inferior vestibular nerves identified, and the nerves sectioned.

The advantage of the middle fossa neurectomy is that the vestibular nerves are identified in a more lateral position with respect to their course from the brain stem. In this position, they have separated from the cochlear nerves and are identified as two separate nerve bundles. This ensures a more complete vestibular nerve section. Published data show a 90 to 95 per cent rate of success in controlling vertigo (Glasscock et al, 1984; Garcia-Ibanez and Garcia-Ibanez, 1980; Fisch, 1977). Hearing is preserved in 90 per cent or better, with hearing remaining at preoperative levels or better in 75 per cent (Silverstein and Norell, 1982). Transient facial palsy may be seen in 4 to 10 per cent of patients, and a subdural hematoma in 1.8 per cent (Glasscock and MIller, 1977). These risks and the unfamiliarity of the approach have relegated this to a second-line procedure behind the retrolabyrinthine vestibular neurectomy.

Retrolabyrinthine Vestibular Neurectomy

This surgical procedure has been well described in the literature (Silverstein and Norell, 1982; House et al, 1984). Briefly, the patient is placed supine in the usual otologic position. A complete mastoidectomy is performed with identification of the lateral and posterior semicircular canals. The sigmoid sinus is decompressed and the dura of the posterior fossa completely removed from the bone. The cerebellopontine angle is entered by creating a flap of dura with incisions medial and parallel to the sigmoid sinus and superiorly parallel to the superior petrosal sinus. The flap is draped over the posterior semicircular canal.

With gentle retraction of the mobile sigmoid sinus and the cerebellum, the seven and eighth nerve complex is identified. Using a sharp hook or scissors, the superior half of the eighth nerve complex (vestibular bundle) is sectioned. At this level of identification, the eighth nerve complex has not separated into distinct cochlear and vestibular nerve bundles. An obvious cleavage plane is seen in approximately 80 per cent of cases. In the 20 per cent that do not have an obvious cleavage plane, the superior half is sectioned.

The advantages of the retrolabyrinthine approach are that its anatomy is more familiar and consistent for the surgeon, retraction of the temporal lobe can be avoided, and the facial nerve

is relatively safe, since it has a more divergent path at this level. Various series have placed the rate of success in controlling vertigo at 90 to 95 per cent, which is comparable to results for neurectomy using the approach through the middle fossa (Silverstein and Norell, 1982; McElveen et al, 1984). In the McElveen series of 52 patients, a success rate of 93 per cent was found, with no incidence of total deafness, facial palsy, or complications involving cerebrospinal fluid. In a recent review of 200 patients undergoing retrolabyrinthine neurectomy, similar results have been found (McElveen, 1987).

Retrosigmoidal Vestibular Neurectomy

Silverstein and colleagues (1987) recently introduced retrosigmoid neurectomy in an attempt to identify the vestibular nerves in a more lateral position after their division from the cochlear nerve. Thus it was felt that a more complete section could be accomplished. In this approach, the cerebellopontine angle is entered posterior to the sigmoid sinus through a craniotomy opening, with some retraction of the cerebellum required. With more posterior visualization of the internal auditory canal, the posterior lip is drilled away until the singular canal is identified near the inferior aspect of the internal auditory canal. The superior vestibular nerve is sectioned, as well as the posterior ampullary nerve. The remaining inferior vestibular nerve fibers (saccular nerve) are not transsected, as they are intimately associated with the cochlear nerve.

Final results with this procedure await more time and experience to see if it gains the acceptance now held by the retrolabyrinthine neurectomy. It appears that retrolabyrinthine vestibular nerve section has become the most widely used of the three procedures. All three procedures do carry a higher risk than other procedures for control of vertigo, but in well-trained hands there has been a low incidence of postoperative complications. Each different technique for vestibular nerve section carries particular complications; all of these techniques share the risk of wound infection, leakage of cerebrospinal fluid, meningitis, hearing loss, deafness, facial weakness, facial palsy, stroke, coma, and death. An example of technique-inherent complication is the infection of the abdominal wound in those procedures in which fat is harvested from the abdominal wall (Kemink and Hoff, 1986; Silverstein et al, 1987).

Singular Neurectomy

Gacek (1974) first reported this procedure for relief of vertigo associated with benign positional vertigo. The procedure is based on the theory of "cupulolithiasis" with resulting sensitivity to gravitational changes in the cupula of the posterior semicircular canal. Singular neurectomy is a surgical procedure for selective transection of the posterior ampullary nerve in the singular canal. Since benign postural vertigo is usually a self-limiting process, an extended trial of medical therapy and positional exercises for up to 1 year is recommended prior to surgical intervention.

Gacek's technique involves a transcanal approach under local anesthesia and raising of a tympanomeatal flap. Bony overhang of the round window niche is then removed to fully expose

the round window membrane. The singular canal is then approached by drilling just inferior to the attachment of the round window membrane within the floor of the niche. A 1-mm bony margin is left between the membrane and the surgical defect created in the floor of the niche, in order to protect the adjacent cochlea. The posterior ampullary (singular) nerve is identified at a depth of roughly 1.5 to 2 mm. It is then removed, using a small hook.

The major disadvantage of this procedure remains the significant risk of sensorineural hearing loss and the technically difficult approach, with inability to identify the nerve. Nevertheless, singular neurectomy appears to be an effective method of treating intractable benign paroxysmal positional vertigo. Since only a partial vestibular destruction occurs, compensation for the patient is much less than for total vestibular neurectomy (which is advocated for patients in whom the singular nerve is not identified).

Ultrasonic Irradiation

Ultrasonic irradiation for Ménière's disease was introduced by Arslan (1953). By irradiating the labyrinth with ultrasound, he hoped to perform a selective labyrinthectomy, destroying the cristae of the semicircular canals while preserving the organ of Corti. Initially, the ultrasound was applied to the lateral semicircular canal through an approach as in a simple mastoidectomy (Arslan, 1953; Stahle, 1976). Later, Kossoff and associated (1967, 1972) developed a probe that allowed ultrasonic irradiation to the round window via a tympanostomy incision. As the ultrasonic irradiation is absorbed, heat is given off that dissipates quickly throughout the fluids of the inner ear, causing selective injury to the membranous labyrinth.

Cryosurgery

Cold has been applied to the lateral semicircular canal. This modality does not appear to affect the hearing, which continues to fluctuate post-cryosurgery in the same manner as prior to it (Wolfson, 1966).

Gentamicin Therapy

Gentamicin is irrigated into the middle ear via a needle through or a hole in the tympanic membrane. By a combination of vestibular and audiologic testing, it is attempted to find a dose that will be absorbed into the inner ear and destroy vestibular but preserve auditory function.

Destructive Procedures (Hearing Destroyed)

As mentioned previously, criteria for destructive surgery for vertigo (destroying hearing) have narrowed. With newer procedures that conserve hearing providing higher rates of cure of vertigo, any residual hearing in a patient deserves careful consideration. This is especially important in Ménière's disease, in which bilaterability often develops. With data emerging from patients receiving cochlear implants, even a small amount of hearing has been shown to be important. However, in profoundly or totally deafened ears with residual, damaged vestibular

function, destructive procedures are still used to control vertigo.

Transtympanic Labyrinthectomy

Labyrinthectomy Extended to the Oval Window

This procedure may be indicated in elderly patients or patients in whom general anesthesia is hazardous. It may be performed under local anesthesia and involves a relatively short operating time. A tympanomeatal flap is raised, allowing good exposure of the promontory, oval window, and round window. The stapes is then removed, and with an appropriate cutting bur the bone between the oval and round window is removed. As much of the membranous labyrinth as possible is then removed with a hook. The tympanomeatal flap is replaced and the procedure terminated.

Transcanal Labyrinthectomy

Schuknecht (1957) described a procedure for transcanal labyrinthectomy that may be performed under local or general anesthesia. A tympanomeatal flap is elevated as for a stapedectomy, and the stapes is then removed. The saccular and utricular maculae are then removed from the vestibule through the oval window. With a blind sweep of a 3-mm right-angled hook, the neuroepithelium is then removed from the combined lateral and superior ampullary cavity and the posterior ampullary cavity. The vestibule is then aspirated thoroughly with a 20-gauge suction tip.

Transmeatal Labyrinthectomy

Silverstein (1976) introduced the transmeatal labyrinthectomy in an attempt to improve results of the transcanal labyrinthectomy. The technique involves using either an endaural or postauricular incision to gain added exposure. A tympanomeatal flap is then elevated and wide exposure of the promontory and windows is obtained, with removal of bone if needed. The singular nerve is identified posterior and inferior to the round window membrane. The promontory of the cochlea is then removed, exposing the vestibule with the saccule and cochlear ducts, and the basal turn of the cochlea is widely opened.

With a diamond bur, the singular nerve is then followed to the internal auditory canal, if the cochleovestibular nerve is also to be sectioned.

Transmastoid Labyrinthectomy

A transmastoid labyrinthectomy offers a more complete and thorough removal of vestibular function from an ear than does the transcanal approach. After exposure of the bony labyrinth through a simple mastoidectomy, each of the three semicircular canals is systematically drilled away in its entirety. By following the canal to their ampullated ends, the vestibule may be widely opened and, under direct visualization, the neuroepithelium may be removed from

within. Hence, this procedure effectively removes all of the membranous neuroepithelium under direct visualization.

Translabyrinthine Section of the Vestibular Nerve

A transmastoid labyrinthectomy is performed as the initial part of this procedure. The internal auditory canal, which lies immediately medial to the floor of the vestibule, is skeletonized and finally bony removal is accomplished with a hook or pick. Bill's bar helps identify the facial nerve and protect it from damage during sectioning. The superior and inferior vestibular nerves and cochlear nerves may then be sectioned. Again, this procedures combines the advantages of the labyrinthectomy with removal of the membranous labyrinth and preganglionic sectioning of the vestibular nerve. It does add additional potential risks involved with intracranial surgery. Although rarely encountered, these include potential leakage of cerebrospinal fluid, meningitis, and increased risk to the facial nerve. Rates of success have been reported as well over 90 per cent (Pulec, 1964; Glasscock et al, 1980; Graham and Kemink, 1984).

Exploratory Tympanotomy (Interactions Between Middle Ear and Inner Ear)

Many diseases involve middle ear/inner ear interactions, especially through the round window. Such diseases include trauma (for example, fracture of the temporal bone, subluxation of the incus, damage to the round window, and so forth), infection (especially acute and chronic suppurative otitis media), mycoses, and tumors (for example, squamous cell carcinoma and glomus tumors). Diseases including interactions between the middle ear and inner ear are summarized in Table 3. Exploratory tympanotomy is useful for diagnosis and in certain instances treatment of such findings. The most common disease that can cause vestibular upset and deafness is perilymphatic fistula.

Perilymphatic fistulas may occur secondary to changes in pressure (Goodhill et al, 1973; Simmons, 1978) resulting from trauma, diving, or flying, or in postoperative patients (usually after stapedectomy). Perilymphatic fistulas are also associated with chronic otologic problems such as cholesteatoma that produce episodic vertigo with recurrent drainage of the ear; this specific problem is dealt with in Chapter 31 of this volume. With spontaneous fistulas, patients often have a sudden sensorineural hearing loss associated with vertigo, and often a history of trauma or barotrauma (from diving or flying) can be elicited. In patients with a history suggestive of this etiology, and in patients in whom episodic vertigo does not improve, consideration should be given to an exploratory tympanotomy. In post-stapedectomy patients, symptoms include persistent dizziness, beginning weeks after the operation. With newer techniques and better prostheses, post-stapedectomy fistulas have markedly decreased in incidence.

Table 3. Diseases Including Interactions Between the Middle Ear and Inner Ear

Diseases of the Middle Ear with Inner Ear Manifestations	**Diseases of the Inner Ear with Middle Ear Manifestations**
Congenital anomalies	Congenital anomalies
Trauma (including changes from pressure)	Trauma (including changes from pressure)
Infection / inflammation	Infection / inflammation
Tumors	Tumors
Granulomas	Otosclerosis
Ototoxic drugs	Ménière's disease
Cochlear implants	Decompensated
	With perilymphatic fistula
	Perilymphatic hypertension.

The site of leak is usually in the inferior aspect of the round window or around the annular ligament of the oval window. Preoperative electronystagmographic (ENG) evaluation may be helpful in diagnosing this elusive fistula. In a small series, Daspit and coworkers (1980) reported ENG results surgically confirmed in 90.8 per cent of patients tested. When a perilymphatic fistula is suspected from a thorough history or testing for fistula, an exploratory tympanotomy is performed under local anesthesia.

Both oval and round windows are inspected for any obvious perilymphatic leakage. The windows are then grafted with Gelfoam, fat, or similar autografting material. Even if no leakage is found, with a presumptive diagnosis of fistula the round window is often grafted, as intermittent fistulas have been postulated to occur. With a history of barotrauma, initial treatment including bed rest and close observation is recommended. Simmons (1978) recommends exploration if, after 10 days, there is an increase in hearing loss or imbalance persists.

If a fistula of the oval window is found in a post-stapedectomy patient, the graft should be left in place. The area around the fistula and grafted material is freshened and denuded. A new tissue graft is then placed and a new prosthesis is placed if possible (Althaus and House, 1973). An increased risk of hearing loss is incurred if the original graft is removed.

Perilymphatic Hypertension

A syndrome termed perilymphatic hypertension has recently been described by Paparella and colleagues (1987). The clinical diagnosis and picture of perilymphatic hypertension is comparable to that for perilymphatic fistula. The diagnosis is highly suspect in the history and confirmed by exploratory tympanotomy when a decided bulging of the round window membrane is seen. A patient or semi-patient cochlear aqueduct or modiolus is considered precursory to this condition. Perilymphatic hypertension is believed to predispose to perilymphatic fistula, which may be part of the process of resolution. Treatment for this subset of patients has consisted of paracentesis of the round window membrane followed by grafting, with improvement of hearing

in certain patients.