

Chapter 183: Surgery of the Vestibular System

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Successful surgery to relieve vertigo or dysequilibrium depends on an understanding of the anatomy and pathophysiology of the peripheral and central vestibular system. Vertigo or dysequilibrium results from a significant asymmetry between the input from both vestibular labyrinths. Such an alteration in input from one labyrinth may occur as the result of changes in fluid composition, disturbed physiology, infection, ischemia, trauma, degeneration, neoplasm, or surgical trauma. For clinical purposes peripheral labyrinthine disorders may be divided into pathologies that are intrinsic to the labyrinth and the vestibular nerve and those that are extrinsic to the labyrinth. The pathologies *extrinsic* to the labyrinth but affecting and disrupting its function are conditions of the middle ear, mastoid air cells, and ear canal, including infection, trauma, or neoplasm, which in some way encroach on the labyrinth and its nerve supply.

Patients troubled with vertigo from *intrinsic* pathology (Ménière's disease, vestibular neuritis) should be managed nonsurgically (reassurance, vestibular suppressants, counseling) as long as they are able to carry on a relatively normal social and work life. If vestibular symptoms remain severe and frequent enough to result in a significant impairment, then surgical management is indicated. Approximately 25% or less of patients with Ménière's disease require surgical treatment.

In general, *ablation* procedures are used successfully to treat intrinsic pathology of the labyrinth and the vestibular nerve (Ménière's disease, neoplasm, vestibular neuritis, cupulolithiasis, labyrinthitis). Selection of the ablation procedure depends on the presence and level of hearing in the affected and nonaffected ears. *Preservation* procedures, which attempt to preserve function by reversing pathophysiology of the labyrinth, are used for intrinsic pathology but generally are not as successful in relieving vestibular symptoms as the ablation procedures.

Extrinsic pathology that affects the physiology of the labyrinth and is located primarily in the middle ear, mastoid air cells, or ear canal requires preservation procedures to remove the pathology and reverse the pathophysiology of the labyrinth, thus restoring normal function. If the extrinsic pathology has damaged irreversibly the physiology of the labyrinth, an ablation procedure (labyrinthectomy) also is used to complete the removal of diseased tissue and to destroy remaining vestibular function.

Ablation Procedures

Ablation procedures eliminate the neural input to the brainstem from a disordered peripheral labyrinth by destroying the sensory and neural components of the peripheral vestibular system. The sensory cells in the five vestibular sense organs initiate the action potentials carried by the vestibular nerve. If one or both of these components are destroyed, the input from the labyrinth is eliminated and central vestibular compensation can achieve a maximal level of orientation. Relief of symptomatic vertigo thus should be 100% (Gacek, 1978; Hammerschlag and Shuknecht, 1981). Only a deficit in the patient's multisensory input (vision, proprioception, cerebellum, reticular formation) to the neuraxis could result in

incomplete vestibular compensation and be manifested as postoperative dysequilibrium.

Labyrinthectomy

Transcanal labyrinthectomy

The basis for the use of destructive labyrinthectomy is that complete removal of all sensory epithelia (five sense organs) and the peripheral nerve fibers innervating these sense organs is sufficient to eliminate the source of action potentials in the vestibular nerve. Because the vestibular sense organs are located in the vestibule, the most direct surgical approach is through a transcanal exposure to visualize the oval and round windows. However, the promontory must be removed to gain maximal exposure of the vestibule (Gacek, 1978).

Indications for destructive transcanal labyrinthectomy are found in the patient who has unilateral peripheral labyrinth disease with a severe or total loss of hearing. The level of hearing is no longer useful and therefore is not salvageable. Intractable vertigo from a disordered labyrinth may be found with Ménière's disease, postsurgical trauma, viral or bacterial postinfection, or posttemporal bone fracture.

Contraindications for labyrinthectomy are disorders of the labyrinth in which hearing is considered useful (50 dB threshold elevation or better and better than 50% word discrimination) and patients in whom the labyrinthine dysfunction affects the only ear with hearing function regardless of the hearing level. In these cases, preservation of remaining hearing should be attempted.

Labyrinthectomy may be performed under local or general anesthesia, depending on the level of vestibular function present in the diseased ear. Usually general anesthesia is necessary because of the severe vertigo and vagal symptoms the patient exhibits during manipulation and destruction of the vestibular sense organs. Local anesthesia may be sufficient and even desirable, however, if preoperative vestibular function (caloric test) is depressed profoundly. In such a patient with little or no residual vestibuloocular response following a strong caloric stimulus, the vertigo and ocular displacement observed during labyrinthectomy under local anesthesia strongly implicates that labyrinth as the source of the dysequilibrium.

The direct approach to the vestibule provided by the transcanal (middle ear) approach requires wide exposure of the vestibule between the oval and round windows (Fig. 183-1, A). The surgeon can remove the promontory by using a 1 mm cutting burr powered by an electric transcanal drill with an angled handpiece (the Shea Stapes drill). After removing the promontory bone, the surgeon extracts the entire stapes bone, achieving wide exposure of the vestibule. A sufficiently long, right angle hook (4 to 5 mm) then can be introduced superiorly into the roof of the vestibule and swept along the lateral and superior walls of the vestibule, where the superior division sense organs receive their innervation (from the superior division of the vestibular nerve) (Fig. 183-1, B). In sweeping posteriorly to anteriorly in this plane, the surgeon delivers the utricular nerve and the nerves to the lateral and superior canal cristae into the open vestibule and then removes them with a small gauge (No. 24) suction tip.

Additional light curettage of the lateral and superior walls of the vestibule then is performed with the hook to ensure complete destruction of remaining sensory and neural tissue of the superior division sense organs. A small, right angle hook is used to peel the saccular macula from the spherical recess. A special effort is made to denervate the posterior canal crista, which is located in a separate bony recess inferior to the vestibule; this maneuver is best achieved by exposing the singular nerve as it travels parallel to the osseous spiral lamina of the cochlear basal turn. The singular nerve also may be transected selectively before the removal of promontory bone if the surgeon wishes to gain experience with singular neurectomy. If the nerve is transected after the removal of the promontory bone, the singular canal should be exposed by drilling down the bone from the posterior ampullary recess to the osseous spiral lamina in a direction toward the internal auditory canal (IAC). If all the sensory and neural tissue has been destroyed in this way, vestibulotoxic solutions are unnecessary. The bony defect in the vestibule than is obliterated with Gelfoam, and the tympanomeatal flap is returned to its original position.

The control of intractable vertigo by a properly performed labyrinthectomy approaches 100%. The operative procedure characteristically is unaccompanied by significant morbidity and requires a hospital stay of 2 to 6 days for sufficient vestibular compensation before the patient's discharge. The rate at which vestibular compensation occurs depends on the (1) level of preoperative vestibular function, (2) patient's age, and (3) capacity of the multisensory inputs to achieve reorientation.

Complications are rare but include facial paralysis in those cases when the facial nerve is dehiscant in the oval window niche and instrumentation inadvertently has injured it. Cerebrospinal fluid (CSF) leak may be incurred by extending the surgery beyond the bony labyrinth in the area of the spherical recess where the saccular macula is located. Such a defect can be repaired effectively using a connective tissue graft (earlobe fat) packed into the vestibule.

Although neuroma formation within the vestibule may result from vestibular dendrite regeneration after traumatic excision of the end organs, no evidence supports the concept that such neuromas are responsible for persistent vertigo. If all peripheral sensory epithelium is destroyed, the vestibular neurons are devoid of spontaneous electrical activity. The existence of retained vestibular end organ epithelium (posterior canal crista) offers possible explanation for the few reports of persistent vertigo in patients when such neuromas were demonstrated at surgery or in temporal bones.

Transmastoid translabyrinthine labyrinthectomy

The transmastoid translabyrinthine approach to labyrinthectomy differs from the transcanal procedure only in that a more direct approach is carried out through a postauricular incision and a mastoidectomy exposure. Although this approach is more indirect, it may be the approach of choice when it is necessary to remove extrinsic disease, such as chronic inflammatory tissue in the mastoid and middle ear compartments, that has produced labyrinthitis. This method also may be preferred when trauma (temporal bone fracture) or previous surgery (mastoidectomy) has distorted the anatomy of the temporal bone. In these cases exposure of the entire bony labyrinth is necessary to perform complete extraction of extrinsic pathology as well as ablation of the vestibular sense organs.

Total obliteration of the middle ear and mastoid compartments with muscle flaps and free adipose tissue grafts may be performed after complete removal of diseased and normal epithelium in the middle ear, mastoid compartment, and ear canal (Gacek, 1976). Suture closure of the cartilaginous ear canal skin flaps seals the obliteration tissue. Total obliteration of the surgical defect in a nonhearing ear provides rapid healing and eliminates the need for mastoid cavity precautions and care.

Indications for transmastoid labyrinthectomy are the same as for transcanal labyrinthectomy, that is, intractable vertigo and severe hearing loss in the affected ear and a normal contralateral ear. The translabyrinthine approach especially is indicated in those patients with chronic inflammatory disease of the middle ear and mastoid cavity that has produced irreversible disease of the vestibular and auditory labyrinth. This approach allows control of the chronic infection as well as the exposure and destruction of labyrinthine sense organs. A particular advantage of transmastoid translabyrinthine labyrinth ablation is that the posterior surgical approach may spare a sufficient number of cochlear neurons in the pars inferior of the labyrinth. It is possible, therefore, that implanting an intracochlear prosthesis can restore hearing in the contralateral ear. Contraindications are the same as for transcanal labyrinthectomy, that is, a patient with a useful level of hearing.

Transmastoid labyrinthectomy requires exposure of the vestibule from the posterior direction (Fig. 183-2). This procedure is accomplished by precise identification of the tympanic and vertical sections of the facial nerve as well as the posterior and middle fossa dural plates. The surgeon then can perform adequate and safe removal of the bony labyrinth. This removal is necessary to expose the sense organs supplied by the superior division of the vestibular nerve and to provide exposure of the posterior ampullary recess for extraction of the posterior canal crista. This approach does not permit direct visualization of the singular nerve or the saccular macula in the spheric recess. Thus it does not equal the exposure of the vestibular sense organs afforded by transcanal labyrinthectomy.

If complete destruction of the vestibular sense organs is accomplished through this approach, the incidence of complete relief of vertigo should approach 100%. A slightly higher incidence of facial paralysis is associated with this method of labyrinthectomy because of the skeletonization of the facial nerve that is required for exposure of the labyrinth. However, this temporary weakness is infrequent (10%), and excellent functional recovery usually results.

Vestibular nerve transection

Transection of the vestibular nerve is based on pathophysiologic knowledge that the vestibular nerve carries the abnormal action potentials produced by a diseased peripheral sense organ or diseased first-order vestibular ganglion cells. Transection of the vestibular nerve, therefore, eliminates the transmission of these abnormal action potentials and prevents the possibility of nerve regeneration by excision of the ganglion. Vestibular nerve transection may be total or partial.

Total vestibular nerve transection

Transmastoid, translabyrinthine technique. Indications for the transmastoid total vestibular nerve transection are primarily in those patients with intractable or recurrent vertigo and severe sensorineural hearing loss resulting from a peripheral disorder such as temporal bone fracture or previous incomplete labyrinthectomy. The pathologic reaction usually results in a fibrous obliteration of the labyrinth, with isolation of any remaining sensory tissue. Because revision labyrinthectomy would be unpredictable due to the difficulty in identifying and destroying remaining sensory tissue, vestibular nerve transection offers a more certain technique to ablate the peripheral vestibular input. This approach to the IAC also may be used to rule out a lesion such as a vestibular schwannoma or arachnoid cyst that may escape detection by radiologic contrast techniques. This procedure is contraindicated in patients who have useful hearing in the diseased ear and in most patients with poor hearing since labyrinthectomy is preferable.

The technique requires an extension of the approach used to perform a transmastoid labyrinthectomy (Fig. 183-3, A). The facial nerve is skeletonized along its tympanic and vertical segments. The bone of the labyrinthine capsule is removed, exposing the posterior and middle cranial fossae dura until the IAC is reached. The surgeon safely removes bone adjacent to these dural surfaces by using diamond burrs. The bone surrounding the IAC should be removed for at least one-half the circumference of the canal and for its entire length, from the internal auditory meatus to its distal end near the vestibule. The proximal end of the labyrinthine segment of the fallopian canal should be exposed to identify the vertical crest of the IAC, which separates the facial nerve from the superior vestibular nerve. After incising and reflecting the dura along the IAC's entire length, the surgeon can identify the superior vestibular nerve lateral to the vertical crest and separate it from the facial nerve with a right-angle hook. Although further separation of these two nerves may be performed with a blunt dissector, at times a micro-scissors is necessary to transect the vestibulofacial nerve anastomosis. Sharp transection of this anastomosis prevents traction injury to the facial nerve fibers. The superior division is retracted posteriorly with a suction tip, and the inferior division branches (to the posterior canal crista and to the saccular macula) similarly are transected and reflected posteriorly (Fig. 183-3, B). The cochlear nerve then may be transected, if indicated, to relieve tinnitus; however, the relief of tinnitus does not correlate well with cochlear nerve transection.

After the vestibular (and, if necessary, cochlear) nerves have been reflected posteriorly, bipolar cautery should be applied to the proximal end of the eighth cranial nerve to obliterate the blood supply. The nerves then are transected with microscissors at the level of the internal auditory meatus (Fig. 183-3, C). The CSF fluid leak through the dural defect of the IAC is obliterated with a connective tissue graft (adipose tissue) from the abdominal wall. This obliteration should be augmented to the level of the mastoid cortex, and the wound closure is completed without drainage.

The results are excellent for the relief of vertigo and should range between 95% to 100% if patients have been selected properly. A temporary facial paralysis occasionally may result because of the skeletonization of the fallopian canal to obtain adequate exposure of the vestibule. The incidence of this side effect is less than 10%. A CSF leak may persist if the IAC is sealed inadequately. A connective tissue seal with adipose tissue is quite sufficient.

Transcochlear technique. Some surgeons have advocated an approach to the nerves of the IAC through the medial wall of the vestibule and the cochlea as a more complete ablation procedure than labyrinthectomy (Silverstein, 1976). This transcochlear approach is an extension of the total transcanal labyrinthectomy, with the additional removal of the medial wall of the vestibule and the osseous spiral lamina of the cochlea. The lateral (distal) end of the IAC is exposed, and the cochlear and vestibular nerve branches can be transected peripherally. Because exposure is limited to the distal end of the IAC, resecting the vestibular ganglion is not possible. It has been suggested that this form of nerve transection ensures more complete ablation of vestibular function than labyrinthectomy and also may provide relief from tinnitus by transection of the cochlear nerve. Basic physiologic principles, however, hold that total destruction of vestibular sense organs is sufficient to ablate completely the action potentials traversing the vestibular nerve toward the brainstem level. Thus it is not logical that nerve transection will provide more complete ablation. Also, the relief of tinnitus is not a consistent result for cochlear nerve transection.

Indications are the same as for those patients who require labyrinthectomy, that is, those with profound hearing loss and intractable vertigo. However, a specific application that justifies the exposure of the IAC beyond the vestibule is exploration of the IAC when an intralabyrinthine schwannoma has been discovered at labyrinthectomy. This form of vestibular schwannoma mimics the clinical presentation of Ménière's disease; that is, severe recurrent episodic vertigo is associated with low-frequency sensorineural hearing loss (DeLozier et al, 1979). The findings and symptoms are in contrast to the minimal vestibular symptoms and retrocochlear hearing loss produced by the intracanalicular form of vestibular schwannoma. Thus this diagnosis usually is made unexpectedly at labyrinthectomy. Exploration of the IAC can be performed to rule out an intracanalicular component of the tumor. The transcochlear approach to the IAC is a useful surgical maneuver to acquire this exposure.

The transcochlear technique is similar to labyrinthectomy; however wider exposure of the middle ear should be provided, (by using end-aural incisions and widening the bony ear canal posteriorly, thus allowing use of an air-or-electric-powered high-torque drill). After exposing the vestibule and the basal turn of the cochlea by removing the promontory bone, the surgeon uses a diamond burr to remove the medial wall of the vestibule, starting with the spherical recess and the bone over the cochlea until the cribriform plate of the cochlea is entered (Fig. 183-4). The distal ends of the cochlear nerve and the vestibular nerve divisions are then visible as they emerge from the medial wall of the vestibule. The saccular nerve terminating in the spherical recess and the singular nerve traveling in the singular canal can be transected in the distal end of the IAC. Resecting sufficient length of the nerve to include the vestibular ganglion cells is not possible. Exposure of the subarachnoid space in the IAC should be sealed by inserting a tissue graft of adipose tissue or fascia into the bony defect. Packing the tissue in place for at least a week is necessary to obtain a solid seal of the dural defect.

Control of vertigo is approximately the same as with either labyrinthectomy or total vestibular nerve transection. CSF leakage is usually the main complication with the transcochlear approach to the IAC nerves, controlled by tissue seal of the canal.

Technique through middle cranial fossa. This vestibular nerve transection is used to selectively transect the vestibular nerve while preserving the cochlear and facial nerves by exposing them only in the IAC. Auditory function is preserved by avoiding surgical exposure of the labyrinth. Because the vestibular and cochlear nerves separate in the distal end of the IAC, identification of the vestibular nerve branches at the most distal end of the canal provides the most predictable way of separating vestibular from cochlear nerve fibers. In addition, transection of the distal ends of the superior and inferior division nerve branches permits retraction of these nerves to a point level with the internal auditory meatus so that the vestibular ganglion is included (Fisch, 1977). Excision of this segment thus accomplishes vestibular ganglionectomy and prevents the possibility of vestibular nerve regeneration. Such exposure of the IAC is best accomplished through a middle cranial fossa approach.

This technique is recommended for those patients with excellent hearing who have intractable vertigo, usually from Ménière's disease or chronic vestibular neuritis. The hearing level in the affected ear should not exceed a threshold elevation of 20 to 30 dB, and discrimination scores should be no worse than 80%. The magnitude and potential morbidity associated with this procedure suggest that it should be reserved for those patients with minimal hearing loss. In addition, because ablation surgery does not halt progressive endolymphatic hydrops (Ménière's disease), further deterioration of hearing may occur because of progressive hydrops. The selective ablation procedure merely alleviates the disabling vertigo.

Patients with threshold elevations between 30 and 50 dB and with word discrimination scores between 50% and 80% may be considered individually. For example, if a patient considers hearing to be useful and is aware of the possibility of future deterioration of hearing, a middle fossa vestibular nerve section may be recommended if the patient is in good health. Patients selected for this procedure should be favorable medical risks; for example, they should not be over age 60.

The surgical incision is located anterior to the root of the helix and extends directly, superiorly, or in a curved direction toward the forehead (House, 1961). The incision should stay within the hairline, extending through the temporal muscle to the squamous portion of the temporal bone, and the root of the zygoma should be palpated or seen. The surgeon performs a square or rectangular craniectomy with an electric-powered drill with cutting and diamond burrs. After the bone plates has been elevated from the temporal lobe dura, it is preserved in saline for replacement at the termination of the procedure. Administration of mannitol (1 g/kg body weight) at the start facilitates satisfactory and safe retraction of the temporal lobe. Troublesome bleeding from dural vessels can be controlled with the bipolar cautery, absorbable surgical gauze (Surgicel), and either bone wax or a polishing burr if the bleeding vessels are located in bone.

The retractor designed by House is useful for retraction of the temporal lobe. The surgeon elevates the temporal lobe dura with an elevator, proceeding from the lateral surface of the temporal bone to the superior aspect of the petrous ridge and anteriorly to a point where the facial hiatus and the greater superficial petrosal nerve can be identified. The blade of the retractor then can be inserted between the dura and the superior aspect of the petrous ridge, thus maintaining retraction of the temporal lobe. If easy insertion of the retractor blade over the petrous ridge is not possible, the surgeon can anchor the blade by fashioning a small

groove near the petrous ridge with a small diamond burr. The blade of the retractor then can be engaged in this groove to maintain satisfactory temporal lobe retraction.

Although the arcuate eminence should be a useful landmark for locating the IAC, it is not always well formed. A more reliable method is to trace the facial nerve from the facial hiatus and geniculate ganglion in a retrograde fashion through the labyrinthine segment to the IAC. This step is accomplished easily with a small diamond burr and frequent irrigation. The surgeon then exposes the position and the length of the IAC using diamond burrs in a medial direction to the level of the internal auditory meatus.

After incising the dura and retracting the dural flaps, the surgeon can identify the facial and vestibular nerves in the superior compartment of the canal and separate their distal ends at the level of the vertical crest of the IAC. If the vertical crest is not exposed, the cleavage plane between the facial nerve and the superior vestibular nerve can be identified and used as a guide to separate these nerves. The distal end of the vestibular nerve's superior division should be isolated with a right angle hook and avulsed in a medial direction (Fig. 183-5, A). The avulsed end of the superior division is then retracted medially using a small gauge (No. 20) suction tip and separated from the facial nerve by blunt dissection until the inferior vestibular division branches appear. These branches (saccular nerve, posterior ampullary nerve) then are avulsed as well with a hooked instrument and retracted in a medial direction (Fig. 183-5, B). The two divisions of the vestibular nerve are separated with a blunt instrument from the facial nerve to the level of the internal auditory meatus. The surgeon should cauterize the proximal end of the vestibular nerve with a bipolar cautery and then transect it using microscissors (Fig. 183-5, C). In this way the vestibular ganglion and its proximal and distal dendrites are excised. A small piece of temporal muscle fascia, obtained at the beginning of the procedure, then is used to obliterate the dural defect in the IAC; it is held in place by the expanding temporal lobe.

It is useful at the end of this procedure to tack the temporal lobe dura to the bony edges of the craniectomy defect with nylon sutures. In this way the dura is restricted from any further dissection if an extradural hematoma occurs post-operatively. Controlling all epidural bleeders with bipolar cautery and also the branches of superficial temporal artery with tightly knotted silk sutures to reduce the incidence of postoperative bleeding is extremely important. The bone plate removed during the craniectomy then is placed on the temporal lobe surface, and the temporal muscle is closed with absorbable sutures. After closure, the wound is connected to negative pressure drainage.

The results for the control of vertigo, if both superior and inferior division branches have been transected and excised, are approximately 98% to 100% (Glasscock et al, 1984b). The incidence of sensorineural hearing loss should be no greater than 10%, and temporary facial paralysis may occur in approximately 25% of patients. The incidence of facial paralysis probably is related to the method used to locate the IAC. The reliability of this technique in locating the IAC and the temporary nature of the facial weakness justify its use.

In addition to the 10% incidence of sensorineural hearing loss and 25% incidence of temporary facial paralysis, the more serious but infrequent complications include (1) temporal lobe aphasia secondary to edema, (2) epidural hematoma, (3) subdural hematoma, and (4) meningitis.

Retrolabyrinthine technique. The basis for using retrolabyrinthine vestibular nerve transection to relieve vertigo is the same as with the other procedures: to disrupt the vestibular nerve fibers carrying abnormal action potentials from the vestibular sense organs (Silversteni and Norrell, 1980). The retrolabyrinthine approach is used to expose the posterior fossa dura in the mastoid cavity; incision of the dura exposes the cerebellopontine angle. Retraction on the cerebellum slightly stretches CN VII and VIII, bringing them into view across the cerebellopontine angle.

The advantages of this technique are (1) easier exposure than with the middle fossa approach, and (2) lower incidence of facial paralysis because the facial nerve is not used to locate the surgical field. The procedure is advocated as having the same success rate in relieving vertigo.

The disadvantages of this procedure are as follows:

1. The cleavage plane between the cochlear and vestibular nerve does not accurately divide the vestibular nerve fibers from the cochlear nerve fibers. Thus, a certain degree of error is introduced, either some cochlear fibers are sacrificed or some vestibular fibers are spared by surgically developing this cleavage plane.
2. Transecting only the proximal axons and leaving the vestibular ganglion intact theoretically allows for the possibility of regeneration across the transected region.
3. The traction and manipulation associated with dissection of the nerves raises the possibility of mechanical disruption of cochlear nerve fibers.
4. Because the blood supply to the labyrinth (the labyrinthine artery) is proximal to its peripheral branching at this point, injury to the main vascular supply to the labyrinth is more likely than in the distal IAC, where peripheral branches supplying the cochlea are not in the surgical field.
5. The formation and retraction of a posterior fossa dural flap containing the endolymphatic sac present an opportunity for injury to the sac and duct during the exposure of the cerebellopontine angle.

Indications for this procedure are essentially the same as for the patients selected for middle fossa vestibular nerve transection. These patients have unilateral peripheral labyrinthine disease, usually Ménière's disease or vestibular neuritis, and excellent hearing. The retrolabyrinthine technique is contraindicated in patients with poor hearing in the involved ear or with intractable vertigo and excellent hearing in only one ear with hearing ability.

A postauricular incision and a simple mastoidectomy approach expose the posterior fossa dura in the mastoid compartment. Removing bone as close to the labyrinthine capsule as possible is necessary to achieve maximal exposure of the cerebellopontine angle. After exposing the posterior fossa dura, the surgeon incises it, then identifies the cerebellopontine angles by retracting the floccular lobe of the cerebellum. The retraction provides exposure of CN VII and the common trunk of CN VIII in the cerebellopontine angle by applying slight

traction to the nerves (Fig. 183-6, A). A shallow groove on the dorsal surface of CN VIII shows the separation of vestibular from cochlear nerve fibers. This cleavage plane is developed through the CN VIII complex with knives and hooks until the caudal half of CN VIII containing vestibular nerve fibers is transected (Fig. 183-6, B and C). Blood supply travels to the labyrinth ventral to the CN VII and the CN VIII complex and must be avoided during this transection procedure.

Unfortunately the cleavage plane is not developed easily through CN VIII. Therefore, the interface between cochlear and vestibular nerve fibers may not be followed accurately by the surgical cleavage plane (Rasmussen, 1940). A nerve transection may result in either an undesirable transection of some cochlear fibers or an incomplete transection of vestibular nerve fibers. The dural defect is closed with a tissue graft, preferably adipose tissue, to seal the CSF leakage.

The results are reported to be as good for control of vertigo as those of the middle fossa vestibular nerve section (House et al, 1984); the preservation of hearing also is reported to be similar. Theoretically these results should not be equal to those achieved with the middle fossa nerve transection. House et al (1984) claim temporary facial paralysis is less common with the retrolabyrinthine approach than with the middle fossa approach to the IAC.

Complications that may occur relate to the exposure of the subarachnoid space in the cerebellopontine angle. If the dural defect is adequately sealed neither CSF leakage nor meningitis should result.

Partial vestibular nerve transection

Posterior ampullary technique (singular neurectomy). The rationale for using singular neurectomy is based on the large amount of histologic and clinical evidence indicating that the posterior canal crista of the downmost ear in the provocative position is responsible for the vertigo and rotatory nystagmus seen in cupulolithiasis. The physician makes diagnosis by placing the patient in the provocative position and observing a rotatory nystagmus, which occurs after a latent period of 1 to 4 seconds, has a duration of approximately 25 to 30 seconds, fatigues rapidly, and is seen again in the reversed direction when the patient resumes the starting position. The nystagmus is fatigable on repeat testing. Neither an electronystagmographic recording nor Frenzel's glasses are necessary to view the nystagmus present in cupulolithiasis. Because most patients with this disorder have normal or useful hearing, extralabyrinthine denervation of the posterior canal sense organ should result in selective ablation of the pathologic sense organ while preserving auditory function. Furthermore, sense organs of the operated and contralateral ears assist vestibular compensation.

The rationale for using neurectomy as an adjunct in transcanal labyrinthectomy is that the posterior canal sense organ is located in a separate bony recess apart from the superior division sense organs in the vestibule. This location renders it inaccessible. Thus incomplete destruction of the sense organ may result in residual vestibular function and symptomatic vertigo or ataxia. Performing singular neurectomy ensures that the sense organ has been denervated.

Singular neurectomy is indicated for two clinical conditions (Gacek, 1985): (1) chronic benign paroxysmal positional vertigo (cupulolithiasis) in which hearing is normal and the positional vertigo is disabling, as indicated by a duration of at least 1 year; and (2) as an adjunctive maneuver in transcanal labyrinthectomy to ensure denervation of the posterior canal crista.

The contraindications for singular neurectomy are found in those patients who have positional vertigo with nystagmus that does not fit the characteristics of nystagmus seen in cupulolithiasis. These patients may have a central vertigo manifested by a nonfatigable and direction-changing form of positional nystagmus or other forms of peripheral positional nystagmus. A horizontal or vertical nystagmus indicates that other sensory disorders are responsible. In addition, singular neurectomy is not indicated for those patients with the acute form of cupulolithiasis in which the positional vertigo may improve and completely disappear within 6 to 12 months after onset. It also is not indicated in those patients who have chronic cupulolithiasis but are not disabled sufficiently to warrant surgery. These patients are able to tolerate positional vertigo by avoiding the provocative position.

Singular neurectomy is performed through a transcanal tympanomeatal flap approach with the patient under local anesthesia (Fig. 183-7). Exposure of the round window niche is essential and may require curettage of the inferior bony canal wall. The surgeon must remove the bony overhang of the round window niche with a microdrill (Shea Stapes drill) to visualize the entire round window membrane. The round window membrane is the key landmark for locating the singular canal, which is found inferior to the attachment of the most posterosuperior portion of the round window membrane. The surgeon cannot view this portion of the round window membrane easily and thus must identify it by inward displacement of the stapes in the oval window. After identifying this portion of the round window membrane, the surgeon uses a small (0.5 mm) diamond burr to drill a depression in the floor of the round window niche adjacent to the attachment of the membrane's posterosuperior portion. At a depth of approximately 1 to 2 mm, the singular canal usually can be identified, as confirmed by the patient's expression of disequilibrium or rotatory vertigo. An observer may identify the nystagmoid movements. The white, myelinated nerve fibers of the singular nerve can be seen in the exposed canal. After transecting the nerve with small hooks or picks, the surgeon obliterates the bony defect with Gelfoam.

The location of the singular nerve may vary from patient to patient. Usually only a portion of the singular canal protruding into the bony defect superiorly can be identified. One uncommon variant is for the singular nerve to be exposed completely because of its inferior location in the round window niche. Another uncommon location is medial to the attachment of the round window membrane, where the surgeon cannot see it; in this case local anesthesia is necessary because positive identification of the nerve depends heavily on the responses of the patient when the canal is probed.

The major significant complication of singular neurectomy is sensorineural hearing loss because of trauma to the cochlea.

The incidence of sensorineural hearing loss in over 100 cases is 4%. Maintaining a bridge of bone between the bony defect needed to expose the singular canal and the attachment of the round window membrane minimizes the risk. A CSF leak from the singular canal occasionally may occur after the surgeon has probed the proximal end. This leak can be controlled easily, however, by using adipose tissue to obliterate the bony defect.

A series of 102 singular neurectomies performed in 95 patients demonstrated complete relief of positional vertigo in 99 patients (97%) (Gacek, 1991). The remaining three patients improved but still experienced minor symptoms in the provocative position. Bilateral sequential singular neurectomies were performed in 7 of these patients and unilateral neurectomy in 88. The reasons for incomplete relief in three patients are attributed to incomplete transection of the nerve either because of a doubled or a partitioned singular canal.

Although a number of otologic surgeons have found this procedure technically achievable, Parnes and McClure (1990) have suggested alternate approaches for rendering the posterior canal functionless to relieve paroxysmal positional vertigo. They have suggested that plugging the posterior semicircular canal with bone wax or bone paste to mechanically compress the membranous canal through a transmastoid approach may be an easier maneuver to inactivate the posterior canal cupula. However, this approach, which most certainly is associated with traumatic labyrinthitis, requires a satisfactory group of control patients to prove that the effect was due specifically to canal occlusion and not a generic effect from the surgical labyrinthitis. Furthermore, it is debatable whether the endolymph fluid compartment of the posterior semicircular canal can be sufficiently immobilized by occlusion to inactivate a gravity-sensitive cupula. Cupular deflection by gravity is still possible because of the nonrigid membranous compartment whose flexible walls permit endolymph displacement. The extremely high incidence of complete relief by singular neurectomy performed under local anesthesia appears more desirable, especially considering the low incidence (4%) of sensorineural hearing loss.

Superior vestibular nerve transection. Transection of the superior division of the vestibular nerve by way of a middle cranial fossa exposure of the IAC represents a compromise procedure for those patients who are candidates for total selective vestibular nerve transection through the middle cranial fossa approach (Ménière's disease, vestibular neuritis) but in whom anatomic variants in the IAC prevent safe and complete transection of the inferior division branches. Examples of such anatomic variants are (1) an extremely narrow IAC, (2) a prominent vascular loop of the anterior inferior cerebellar artery obscuring the inferior division branches, and (3) a swollen facial nerve in the superior portion of the IAC obstructing a view of the inferior division branches.

The technique is identical to the middle fossa approach to the IAC, with incision and retraction of the dural covering of the canal as necessary to expose the nerves in the IAC. The superior division of the vestibular nerve can be identified consistently and transected by avulsion. Any of the variations just mentioned may make impossible visualization of the inferior division branches of the vestibular nerve. Limiting the nerve transection to only the superior vestibular division, relying on this partial ablation to alleviate the patient's vertigo, is safer. If vertigo does persist or recur either in an episodic form or as positional vertigo (cupulolithiasis), the surgeon may assume that the posterior semicircular canal crista is responsible. If the patient remains disabled or symptomatic, the surgeon can suggest a singular

neurectomy through a middle ear approach.

Medical ablation (streptomycin sulfate)

A description of a medical form of peripheral vestibular ablation is important because it represents the most reliable method of selective vestibular ablation. Many histopathologic observations have demonstrated that the degenerative effect of streptomycin sulfate administered parenterally is on the vestibular sensory hair cells (Schuknecht, 1957). The effect is more severe in the vestibular sensory cells of the cristae than on the maculae of the utricle and saccule.

Streptomycin sulfate administered parenterally is carried through the bloodstream into the perilymphatic space of the labyrinth, where it contacts the type I and type II vestibular hair cells of the neurosensory epithelium. The effect initially is selective for the vestibular system. If the streptomycin sulfate is administered to a point at which the vestibuloocular reflex to an ice water stimulus is absent, no cochleotoxic effect will occur. The danger of cochlear toxicity is significant only when the end point of vestibular ablation has been surpassed. This method of ablating the vestibular sensory epithelium has found great therapeutic use and proved to be safe and effective to treat bilateral Ménière's disease, and to manage disease that affects the only-hearing ear. It is the safest way to selectively ablate vestibular function while preserving hearing.

Patients selected for this treatment must be evaluated carefully. The treatment should be restricted to those who are severely incapacitated by the episodic vertigo of bilateral Ménière's disease and have excellent capabilities of vestibular adaptation. A reliable general rule limits medical ablation to patients who are age 55 or younger and in good physical health.

Streptomycin sulfate is administered intramuscularly in divided doses of approximately 2 to 3 g/day parenterally while the patient is monitored in the hospital. Vestibulo-ocular reflex (caloric-induced nystagmus) and hearing are tested daily. Streptomycin ablation of vestibular function is considered complete when the nystagmus response to ice water stimulus (5 mL) is absent in both ears or in the remaining ear with hearing ability.

Excision of vestibular schwannoma

The diagnosis and surgical treatment of vestibular schwannoma are discussed in Chapters 188 and 192, respectively.

Preservation Procedures

The surgical procedures included in this category primarily are used in the control of episodic vertigo caused by progressive endolymphatic hydrops (Ménière's disease). Generally these procedures are not as effective as ablation techniques to relieve episodic vertigo. The successful control of vertigo following these preservation techniques ranges from 50% to 70%. Preservation techniques attempt to reverse the progression of endolymphatic hydrops and preserve the function of both the vestibular and the auditory divisions of the labyrinth. These procedures, except for ultrasound or cryosurgical labyrinthectomy, are designed to alter

the drainage of endolymph and relieve the progressive endolymphatic hydrops of Ménière's disease.

Endolymphatic sac surgery

The rationale for endolymphatic sac surgery is that because the endolymphatic sac contains the major component of the resorptive epithelium in the membranous labyrinth, decompression or drainage of the sac should provide for better drainage of endolymph. Theoretically, decompression or drainage of the endolymphatic system should decrease the increased volume of endolymph (House, 1962; Shambaugh, 1966). Support for this surgical approach comes from animal experiments, which demonstrate endolymphatic hydrops (EH) in various animal species after destruction of the endolymphatic duct or sac. The development of EH requires various periods in different animal species, occurring most readily in rodents such as guinea pigs, but taking several years in cats or monkeys.

Advantages of the endolymphatic sac procedures are that (1) they are easy to perform, (2) they can be carried out under local anesthesia or a short general anesthesia, and (3) they do not sacrifice hearing function. Disadvantages are the following:

1. It is usually difficult to identify the lumen of the endolymphatic sac.
2. The site of obstruction in the endolymphatic system is variable; it may be located proximal to the sac in the region of the duct. This obstruction may take the form of dilated membranous structures occluding the lumen of the endolymphatic duct.
3. Foreign materials used to shunt the endolymphatic sac into either the mastoid cavity or the subarachnoid space invariably become encapsulated by fibrous tissue, precluding a patent drainage system.
4. Because vestibular signs have been absent in the animal models of EH after endolymphatic duct obliteration, some doubt exists that the EH is always responsible for the vertigo.

Endolymphatic sac surgery is indicated in those patients with intractable episodic vertigo when hearing in the involved ear is useful. The type of surgical drainage performed on the sac usually takes one of two forms: (1) decompression of the endolymphatic sac by removal of the mastoid layer of the posterior fossa bony plate and (2) shunting of the endolymphatic sac lumen into the mastoid air cell system or into the CSF space of the posterior fossa.

Endolymphatic sac surgery is contraindicated in patients who have EH affecting an only-hearing ear in patients in whom the affected ear has no useful hearing ability.

The technique for identification and surgical drainage of the endolymphatic sac is to perform a wide, simple mastoidectomy, with the ear canal wall intact (Fig. 183-8, A). This may be accomplished under local or general anesthesia. The endolymphatic sac can be identified after skeletonization of the dura covering the sigmoid portion of the lateral sinus and posterior fossa. The sac is anterior to the sigmoid sinus and posterior to the posterior

semicircular canal within layers of the inferior half of the posterior fossa dura. It appears as a thickened triangular area of dura, and the lumen usually is identified by incising the lateral wall of the sac until a lumen is encountered. This lumen then is incised widely for the surgical manipulation required for drainage. The shunt from the endolymphatic sac to the mastoid cavity requires that a prosthetic device of Teflon or Silastic be placed into the lumen of the sac that extends out into the mastoid cavity (Fig. 183-8, B). This layer of artificial material then is covered with a temporalis fascia graft for stabilization. If the shunting is accomplished medially into the subarachnoid posterior fossa space, the medial wall of the endolymphatic sac is incised, and a drain tube is inserted to communicate the endolymphatic sac and the subarachnoid posterior fossa space (Fig. 183-8, C). The great pressure of CSF casts doubt about the possible effectiveness of establishing drainage from the endolymphatic sac into the subarachnoid space against the pressure gradient.

The relief of episodic vertigo from endolymphatic sac surgery ranges from 50% to 70% in various series (Glasscock et al, 1984a). Hearing is preserved in most patients, with hearing loss occurring in less than 25%. The disappointing incidence of recurrent or persistent vertigo may be related to the technical problems associated with endolymphatic sac surgery; these disadvantages have been outlined earlier. In addition, a study of patients in whom only mastoidectomy and exposure of the sac were performed without either decompression or shunting has demonstrated similar results to a comparable group of patients in whom the sac was decompressed or shunted (Thomsen et al, 1981). The similarity of results (relief of vertigo, preservation of hearing), regardless of the type of procedure performed on the sac and whether *any* modification is performed on the sac, strongly suggests that the beneficial results cannot be attributed to an alteration of the fluid physiology in the labyrinth.

The primary postoperative complications are (1) facial paralysis (usually temporary), (2) fistulization of the labyrinth (posterior semicircular canal), (3) injury to the sigmoid sinus, (4) injury to the posterior fossa dura with CSF leakage, and (5) hearing loss resulting from injury to the labyrinth.

Cochleosacculotomy

Schuknecht (1982) popularized cochleosacculotomy, in which the surgeon creates a fistula between the perilymphatic and the dilated endolymphatic compartments by inserting a hook through the basal turn of the cochlea. If the fistulization interrupts the bone of the osseous spiral lamina, permanent fistulization should occur. Animal studies have supported this hypothesis by showing fistula formation with little damage to the sensory structures of the labyrinth, as revealed by light microscopy. Successful permanent perilymph-endolymph fistulization varies with species, however, and the predictability of this concept in human beings is not known.

The cochleosacculotomy procedure is indicated in patients with intractable vertigo from EH when useful hearing is present. It is particularly suited to those patients who are candidates for a simple procedure performed under local anesthesia. Thus patients who are age 60 years or who are poor medical risks for general anesthesia are candidates. The operation is simple and is associated with little morbidity to the facial nerve, subarachnoid space, or brain. The procedure is contraindicated in patients with only one functioning ear who have Ménière's disease or in those who have unilateral Ménière's disease with poor

hearing.

The operation is performed through a transcanal approach to the middle ear. Complete visualization of the round window niche and the oval window should be obtained. The exposure of the round window niche and membrane may require removal of the bony overhang of the round window niche with a microdrill. A 4-mm, right angle hook then is introduced superiorly through the round window membrane and the osseous spiral lamina of the hook portion of the cochlea's basal turn (Fig. 183-9). The surgeon may detect the penetration of the spiral lamina by the hook by manipulating the stapes footplate with the tip of the hook. The defect in the round window membrane then is obliterated with Gelfoam, and the tympanomeatal flap is returned to its original position.

The results reported thus far, although followup periods are less than 2 years, indicate that relief of vertigo may be achieved in 60% to 75% of patients and hearing maintained in 50%.

The complications of the procedure are (1) hearing loss resulting from trauma to the cochlea or (2) recurrent vertigo because of incomplete fistulization of the perilymphatic and endolymphatic spaces.

Ultrasound alteration of labyrinth

The use of ultrasound irradiation of the labyrinth is based on ultrasound delivered through a probe generating heat or some other form of energy that is destructive to the vestibular labyrinth (Barnett and Kossoff, 1977; Pennington et al, 1980). Therefore, administering and directing it toward the vestibular sense organs can render the sense organs nonfunctional without surgically entering the bony labyrinth system. The ultrasound energy may be administered through a round window or a lateral canal technique.

Round window technique

The round window technique for ultrasound labyrinthectomy requires exposure of the round window membrane through a transcanal approach. A special probe that fits into the round window niche then is directed superiorly through the basal turn of the cochlea (Fig. 183-10, A).

This procedure is *indicated* in patients with endolymphatic hydrops who have intractable episodic vertigo and useful hearing. It is *contraindicated* in patients with only one functioning ear who have Ménière's disease or in those who have nonuseful hearing.

Although the round window technique of delivering the ultrasound energy risks the function of the extreme basal end of the cochlea, its proponents state that this does not produce any significant hearing loss to the upper turns of the cochlea, where speech frequencies are located. Thus the ultrasound energy can be directed through the hook portion of the cochlear basal turn into the vestibule, where the vestibular sense organs are located.

The ultrasound destruction of the cristae ampullares (ampullary crests) is monitored by checking the nystagmus response with the patient under local anesthesia. Initially an irritative nystagmus is produced, which beats toward the operated ear; it then reverses direction (paralytic nystagmus), indicating destruction of canal function. Unfortunately with the round window approach, end point stimulation of the labyrinth with a cold caloric test to determine complete destruction of vestibular function is not practical.

Lateral canal technique

Ultrasound energy may also be delivered through the lateral and posterior semicircular canal approach. Because this technique requires that the labyrinth capsule of the amputated end of the lateral canal be thinned down to the endosteal bone layer, a simple mastoidectomy first must be performed with the patient under local anesthesia to expose the lateral canal. The purpose of the thin layer of bone is to admit the ultrasound energy efficiently without violating the perilymphatic space.

A special ultrasound probe is placed over the thin bone and directed toward the ampullae (Fig. 183-10, B). The energy is delivered at a conservative wattage to destroy the lateral and superior canal cristae. As with the round window technique, an irritative nystagmus is produced initially, reflecting the heat insult into a paralytic nystagmus when complete ablation occurs. Confirmation of injury to the superior division sense organs then is obtained in the form of an absent vestibuloocular response when a cold stimulus (etylchloride-saturated cotton ball) is placed in contact with the thinned-down portion of the bony lateral canal.

Although the original technique with lateral canal ultrasound irradiation described only the lateral canal application, the unsatisfactory incidence of recurrent vertigo suggests that the posterior canal crista also should be irradiated through a similar technique. Thus, similar preparation of the posterior semicircular canal also should be performed, with the ultrasound probe directed toward the ampullated end (inferior) of this semicircular canal. The irradiation is monitored again by the appearance of nystagmus that eventually reverses direction.

The long-term results for permanent control of vertigo after ultrasound irradiation falls in the range of 50% to 60%. Short-term results (1 to 1.5 years) may range around 80% to 85%, but with continued follow-up this percentage is diminished.

The most serious complication of ultrasound irradiation is sensorineural hearing loss. If the bony labyrinth has not been fistulized, however, this problem can be kept at a minimum. Significant sensorineural hearing loss is estimated conservatively at less than 25%. The other significant complication is facial nerve paralysis when the lateral canal technique is used. This complication is related to the heat generated by the ultrasound probe and the proximity of the placement of the probe to the facial nerve. Facial pain or ear pain during the irradiation usually indicates facial nerve injury. Reduced power of the ultrasound energy, therefore, is advisable if the patient complains of facial or ear pain; this is a practical technique for avoiding facial paralysis. The facial nerve paralysis is temporary, and a good functional recovery occurs.

Cryosurgical alteration of labyrinth

Wolfson (1984) has advocated the use of cryosurgery to destroy vestibular function by application to the lateral canal. The technique is similar to that used for ultrasound irradiation of the labyrinth through the lateral canal. It can be performed through a simple mastoidectomy approach with the patient under local anesthesia. Its primary use is for those patients who have good hearing and intractable vertigo. It is contraindicated in patients who have poor hearing or have only one functional ear with symptomatic endolymphatic hydrops. The procedure is performed easily and is associated with minimal risk to the facial nerve or the intracranial structures. The control of vertigo is reported to be from 50% to 60% (Wolfson, 1984).

Miscellaneous Preservation Procedures for Vertigo

Repair of bony labyrinth fistula

Fistulization of the bony labyrinth may occur over one or more of the semicircular canals or the lateral cochlear wall. Bone erosion from chronic infection usually occurs over the ampullated end of the lateral canal or the superior canal; occasionally fistulization of the posterior canal may occur.

Episodic vertigo on compression of the ear canal is the typical complaint in patients with fistulization of the vestibular labyrinth. A low-grade circumscribed labyrinthitis or serous labyrinthitis frequently accompanies the fistulization of the bony labyrinth. As a result, various degrees of disequilibrium or ataxia also may be present. Although the presence of the fistula may be detected preoperatively by a positive fistula response on pneumatic otoscopy, a negative response does not indicate the absence of a fistula. A barrier to induced pressure change in the form of soft tissue or cholesteatoma debris may produce a false-negative response on pneumatic otoscopy. The surgeon thus should expect a bony labyrinth fistula in *all* patients undergoing surgery for chronic middle ear and mastoid disease, particularly when cholesteatoma is present. The surgeon may suspect fistulization of the lateral cochlear wall if a sensorineural hearing loss is present preoperatively, but this can be confirmed only intraoperatively with careful surgical exploration.

Repair of a bony labyrinth fistula is indicated when vertigo accompanied by a positive fistula response is present in the patient with chronic middle ear disease. If hearing in the affected ear is useful, repair of the bony fistula is indicated, depending on the size of the bony fistula and whether the cholesteatoma membrane can be removed safely from the exposed membranous labyrinth. If, on the other hand, profound or total loss of hearing is present, repair of the fistula is unnecessary, and a labyrinthectomy should be performed to explore the vestibule and prevent further complications from progression of the inflammatory process.

The surgical technique for repair of a bony labyrinth fistula depends on (1) the size and location of the fistula, (2) the function of the involved and opposite ears, and (3) the surgeon's experience (Gacek, 1974). A consistently successful approach for identification of the fistula is to evacuate the contents of the cholesteatoma sac and palpate the prominent bony landmarks of the labyrinth through the intact cholesteatoma matrix. When a fistula is found, as indicated by a lack of resistance to the palpating instrument, the surgeon should perform

a radical mastoidectomy and remove all diseased inflammatory tissue throughout the mastoid and middle ear compartments, except for the segment over the fistula. When all bleeding and significant bone removal has been completed, the surgical removal of the cholesteatoma membrane over the fistula can be initiated. In general, if the fistula over the vestibular labyrinth is no larger than 2 or 3 mm, safe removal of the matrix from the underlying endosteal membrane usually is possible because the matrix is not sufficiently adherent to the endosteal membrane. If the fistula is larger than 3 mm, the cholesteatoma membrane may or may not be firmly adherent to the endosteal membrane. Using a fine-gauge (No. 24) suction tip and a delicate elevator (sickle knife or a dental elevator), the surgeon must determine the degree of adherence by carefully elevating the cholesteatoma membrane (Fig. 183-11, A and B). If the matrix can be peeled off the endosteal membrane easily, then complete removal should be attempted. On the other hand, if elevation of the matrix produces a small tear in the endosteal membrane, the matrix should be returned to its original place. The middle ear then is reconstructed around the area of fistulization. If the matrix has been removed completely, the area of bone erosion surrounding the fistula can be repaired by fashioning a "cap" from cortical bone (Fig. 183-11, C). This bony cap is sculptured carefully with a diamond burr to fit precisely the bony defect and is anchored with a layer of temporal fascia. The stabilization will allow fibrosis and osteogenesis to complete the repair of the bone defect.

A bony fistula of the lateral cochlear wall in a hearing ear should not be manipulated surgically because sensorineural hearing loss will result from even atraumatic removal. Only in the case of a nonhearing ear when total labyrinth destruction and exploration is planned should removal of cholesteatoma membrane from a cochlear fistula be attempted.

Repair of oval or round window fistula

The presence of a round window or oval window perilymphatic fistula is difficult to diagnose precisely preoperatively and to identify intraoperatively. Although almost any history may be obtained in patients who develop spontaneous perilymphatic fistula, the presence of a sudden middle ear pressure change preceding the loss of hearing and the appearance of vestibular symptoms strongly suggest perilymphatic fistula. Because a perilymphatic fistula may heal spontaneously, an initial observation period lasting 7 to 10 days should be allowed; bed rest and sedation can be incorporated into the observation period.

Persistence of hearing loss and vestibular symptoms after this period of conservative management indicates the need for exploratory tympanotomy to search for an oval or round window fistula. Careful exposure and examination of the annular ligament and the round window membrane should be performed to identify a defect consistent with a perilymphatic fistula. Although exposure of the oval window usually is attained easily, exposure of the round window membrane requires removal of the bony overhang of the round window niche. This is done with a small cutting burr powered by an electric middle ear drill (Shea Stapes drill). An accurate decision on the presence of a fistula must be based on direct visualization and examination of the two windows. Clear fluid emerging from a defect in the round window membrane or the annular ligament is *required* for the surgeon to judge that a fistula is present. Clear fluid welling up in the middle ear space (round window niche) represents *indirect* evidence of perilymphatic fistula. If a fistula has been identified in either the annular ligament or the round window membrane, repair with a tissue graft is recommended.

When a definite fistula in the oval or round window has been repaired with a tissue graft, recovery of auditory function and elimination of the vertiginous symptoms generally are favorable. If the fistula has been repaired beyond 2 to 3 weeks after its onset, recovery of function is more unpredictable. Generally, the longer the interval from onset to repair, the poorer the chances for recovery of hearing. Vestibular symptoms may improve even with loss of vestibular sensitivity because of vestibular compensation.

Repair of poststapedectomy complications producing vertigo

Two complications after stapedectomy may be responsible for significant vertigo and sensorineural hearing loss: (1) long stapedectomy prosthesis and (2) poststapedectomy granuloma. Both are correctable by well-timed surgical intervention.

Long stapedectomy prosthesis

The excessively long stapedectomy prosthesis produces ataxia and ultimately a sensorineural hearing loss in patients who have undergone stapedectomy with replacement by a piston prosthesis. It is also possible, although not as common, for the excessively long prosthesis to be formed of tissue and stainless-steel wire. The increased association between hearing loss and a piston prosthesis is related to the need for inserting the prosthesis beyond the level of the oval window to prevent refixation by a fibrous or bony membrane. Because the measurement of the projection into the vestibule is critical due to the proximity of the utricular macula to the oval window, judging the precise length of the prosthesis by depression of the incus with the patient under local anesthesia is necessary. The patient's experience of dizziness or ataxia when the prosthesis is depressed indicates the prosthesis is too long, and a shorter one must be used.

Prolonged postoperative dizziness for months after stapedectomy characterizes the clinical manifestation of a long prosthesis. Ocular displacement following pneumatic otoscopy can accompany and exacerbate the vestibular experience. The recommended treatment is revision stapedectomy with extraction of the prosthesis. The surgeon then uses a shorter prosthesis and carefully monitors its length by checking the patient's response under local anesthesia. The result of this method of recognition and correction of long prosthesis is satisfactory.

Poststapedectomy granuloma

An excessive reparative response to the surgical trauma in the oval window region produced at stapedectomy causes poststapedectomy granuloma (Gacek, 1970). The progressive granulomatous response in the oval window is responsible for a serous and serofibrinous labyrinthitis, manifested by a sensorineural hearing loss and persistent or recurrent vertigo 1 or 2 weeks after stapedectomy. The diagnosis should be suspected in the patient with prolonged vertigo beyond 2 to 3 days after stapedectomy for whom a sensorineural hearing loss with markedly reduced speech discrimination scores is recorded. Careful examination of the tympanic membrane frequently reveals erythema and induration, reflecting the granulomatous mass in the middle ear. This is particularly evident in the posterosuperior quadrant of the drum.

When the diagnosis is suspected and progressive labyrinthine signs are documented, immediate exploratory surgery with removal of the entire granuloma is necessary. Removal of any granulomatous component extending into the vestibule is important to remove completely the source of the labyrinthitis (Fig. 183-12). A tissue wire graft is used to replace the original prosthesis and seal the oval window. Recovery of hearing and complete relief of vestibular symptoms are excellent if the surgery has been performed within 10 days of the original stapedectomy.

Management of serous labyrinthitis secondary to chronic otitis media

The rationale for using this procedure is that the presence of severe chronic inflammatory disease in the oval and round windows, particularly the round window niche, may produce a serous labyrinthitis by the transmission of toxic effects of the inflammatory process across the round window membrane. The clinical manifestation is that of persistent unsteadiness, intermittent dysequilibrium, or ataxia, which may or may not be accompanied by sensorineural hearing loss in an ear with chronic otitic media. Sensorineural hearing loss may be limited to the high frequencies or may manifest as a flat threshold elevation. Indications of labyrinthine capsule involvement (bony fistula) by the chronic infection are absent (negative fistula test).

The surgical procedure requires extensive and thorough exenteration of mastoid air cells, with removal of diseased granulation tissue, mucous membrane, and cholesteatoma from both compartments. The surgeon should search thoroughly for fistulization of the bony vestibular or auditory labyrinth. Finding no such bony fistula, the surgeon should make a special effort to examine the oval and round windows. Chronic inflammatory tissue and granulation tissue should be removed from the footplate, and its annular ligament should be removed from the footplate, and its annular ligament should be examined carefully for defects. Finally the surgeon should identify and explore the entire hypotympanum and the round window niche. A common finding in patients with this syndrome is a concentration of mucous membrane and granulation tissue disease in the round window niche and the tympanic sinus. This area frequently is sealed off by an overhanging ledge of bone from the posterior canal wall anterior to the vertical portion of the fallopian canal (Fig. 183-13, A). Complete exposure of the round window niche so that inflammatory tissue can be removed safely and completely from the niche requires maximal removal of bone anterior to the vertical portion of the facial nerve. This exposure depends on visualization of the vertical portion of the facial nerve by removal of the fallopian canal (Fig. 183-13, B). After completely exposing the round window niche, the surgeon should strip completely the mucosal disease from the niche and from the round window membrane itself. This may require removal of the bony overhang of the round window niche with a diamond burr.

The major complication of this procedure is injury to the round window membrane. This problem can be avoided by carefully freeing mucous membrane tissue with sharp instruments and small-cup forceps, removing the mucous membrane and granulation tissue disease piecemeal. If the mucous membrane and granulation tissue cannot be removed safely from the niche and the round window membrane, it should be left untouched. The second possible complication is temporary facial weakness caused by surgical injury to the facial nerve sheath.

Relocation of vascular loops

The neurosurgical specialty has long promoted the relocation of a vessel loop making contact with CN V, VII, and IX for the relief of symptoms related to these sensory and motor nerves (Gardner, 1959, 1962; Jannetta, 1967, 1970). It was inevitable that the same approach would be advocated for the relief of auditory and vestibular symptoms presumably caused by a vessel loop contacting or traumatizing the eighth cranial nerve. Jannetta (1975, 1980) has espoused this approach and claims relief from these symptoms after relocation with an insertion of a synthetic sponge between the vessel loop and the eighth cranial nerve. The technique has not been universally supported, however, probably because of unsatisfactory objective evidence of an unequivocal effect on auditory and vestibular symptoms after relocation of the vessel loop.

Several factors mitigate against the value of this approach for the relief of auditory and vestibular symptoms in patients where the clinical findings are not compatible with a known pathology of the labyrinth or eighth cranial nerve (that is, Ménière's disease, vestibular neuronitis, labyrinthitis, cupulolithiasis).

1. Vessel loops, particularly a loop of the anterior inferior cerebellar artery (AICA) have been identified in a high proportion of normal dissected and sectioned temporal bone specimens (Mazzoni and Hansen, 1970; Quaknine et al, 1978; Sunderland, 1945). Yet an association of the vessel loop with auditory or vestibular symptoms could not be made from these pathologic studies, particularly in temporal bone histopathology specimens (Fig. 183-14). If this were a common condition, one would expect an established correlation based on these many pathologic observations.

2. The mechanism based on trauma to the nerve as a result of pulsation from the vessel has not been convincingly demonstrated. When observed during intracranial surgery in the cerebellar pontine angle, vessel loops and the seventh and eighth nerve complex are characteristically seen swaying together bathed in CSF and displaced by the transmitted venous pulsations of the spinal fluid space. It is difficult to justify a traumatic effect unless the nerve complex were stationary. However, a large tortuous vessel such as the basilar artery (Fig. 183-15) may compress the eighth nerve against the temporal bone causing hearing loss and vertigo (Benecke and Hitselberger, 1988).

3. A control series of patients against which to compare the patients with surgically relocated vessel loops has not been included to demonstrate a known effect of the relocation procedure.

4. The results of vessel relocation surgery have been evaluated on a subjective rather than objective basis (for example, a test of auditory function or a test of vestibular function such as nystagmus response in positioning maneuver or a test of the vestibuloocular reflex sensitivity).

Until more convincing evidence is forthcoming that vessel loop relocation is responsible for the unequivocal relief of these symptoms, the value of vessel relocation surgery cannot be recommended.

Summary

A variety of surgical procedures presently are available for the control of peripheral vertigo. The successful implementation of these procedures is based on a thorough knowledge of the anatomy and pathophysiology of the vestibular system, as well as on clinical experience formed from an objective appraisal of the effects of such surgery. Although many of the procedures described are consistently successful in relieving symptoms of vertigo, the surgeon should realize that modifications or the addition of new techniques may appear as our knowledge of the histopathology and the pathophysiology of the vestibular system increases.