Introduction

Presumably in response to evolution, the brain has developed an elaborate system of neural connections between the peripheral vestibular system, cerebellum, higher cortical centers, and other peripheral nervous systems. When a peripheral vestibular insult occurs the cerebellum rapidly responds followed by alterations by the cortical neural systems to adapt to the change in static and dynamic peripheral vestibular input. In order for the nervous system to recalibrate, the relationship between the visual, auditory, proprioceptive, and vestibular signals; repeated head, eye, and body movements are essential. Unfortunately, these movements also aggravate the symptoms of vertigo and the associated autonomic consequences of nausea, vomiting and other derangements. These unpleasant results of movement may lead the patient to pursue a posture of inactivity. It is at this point that patient education about the importance of rehabilitation be emphasized. Additionally, a certain subset of patients may benefit from formal vestibular rehabilitation therapy.

The most common vestibular disorders requiring treatment include benign paroxysmal positional vertigo (BPPV or BPV) and Meniere's disease. These disorders may be treated by either medical or surgical methods with surgery reserved for medical failures. A relatively new diagnosis which may require surgical management is superior canal dehiscence syndrome. BPPV will be discussed including medial rehabilitation and surgical techniques followed by a discussion of Meniere's disease, chemical labyrinthectomy, and surgical procedures primarily for Meniere's but also other peripheral disorders, and the superior canal dehiscence syndrome. Surgery for tumors affecting the vestibular system will not be discussed.

Rehabilitation and Surgical Management of Benign Positional Vertigo

Benign positional vertigo (BPV) is the most common peripheral vestibular disorder and is very effectively treated by vestibular rehabilitation. BPV is characterized by several findings of history and physical exam. The patient will usually offer a history of rotational imbalance that lasts for a period of seconds and will usually relate an alteration in head movement as the inciting event. On performance of the Dix-Hallpike maneuver, the posterior semicircular canal of the undermost ear is in the optimal plane of relative endolymph motion. The nystagmus produced by BPV has several distinct characteristics. The nystagmus is predominately rotational superior-laterally to the affected down-side ear, has a brief latency of onset of 5-
15 seconds, has duration of less than 30 seconds, and is fatigable with repetition of the provocative maneuver. Additionally, the otolaryngologist should be aware of the recently described horizontal semicircular canal variant of BPV. In this variety the provocative maneuver is in the supine-head lateral position. The duration of the true vertigo is between 30 seconds and one minute; the latency is no more that 3 seconds, and there is no fatigability of the response.\(^1\)

Physical therapy exercises for peripheral vestibular disorders were originally described by Cawthorne in 1954. Exercises specific to the treatment of BPV have been described by many different authors. Semont was the first to describe and gain acceptance of the use of rapid single-treatment approach, the liberatory maneuver, for BPV. He claimed a 83.96\% success rate after one maneuver and 92.68\% success with two maneuvers with only a 4.22\% recurrence. Others have had less success with the liberatory maneuver, and some have claimed that it is too violent. Two common rehabilitation efforts used to treat BPV are the Brandt and Daroff exercises and the Epley canalith repositioning maneuver; these are described in further detail.\(^1,3\)

The therapeutic protocol for the Brandt and Daroff exercise is as follows. The patients are seated with their eyes closed and tilted laterally to the precipitating position, with the lateral aspect of their occiputs resting on the bed to ensure proper plane-specific stimulation of the posterior semicircular canals. They remain in this position until the evoked vertigo subsides, and then they sit up for 30 seconds before assuming the opposite head-down position for another 30 seconds. In the case in which vertigo occurs in the opposite position (bilateral BPV), the posture is maintained until cessation of the symptoms. The sequence of positioning at each session is repeated until the vertigo subsides. The maneuver is carried out by the patient every three hours while awake and is terminated after two consecutive vertigo-free days. With this maneuver Brandt and Daroff reported 66 of 67 patients experienced complete relief of the positional vertigo within three to fourteen days, with most requiring seven to ten days. Two of the 66 patients experienced a recurrence of BPV after several months, but each responded to a second course of therapy. Additionally, the one patient whose condition did not improve was found to have a perilymphatic fistula mimicking BPV.\(^4\)

The Epley canalith repositioning procedure (CRP) is based on the theory that BPV is caused by free-floating densities, canaliths moving freely in the endolymph of the posterior semi-circular canal. CRP was designed, with the use of head maneuvers and induced vibration, to cause free canaliths to migrate by gravitation completely out of the PSC, by way of the common crus, to the utricle, where they no longer would affect the dynamics of the semicircular canals. The target SSC is predetermined by the Hallpike maneuvers and the sum of the latency and duration of the induced nystagmus is noted. This provides an estimate of the time required for the canalith bolus to gravitate through 90 degrees. Patients are premedicated with a transdermal scopalamine patch the previous night, or 5 mg diazepam orally one hour earlier. The operator is located directly behind the patient, an assistant at the patient’s side. The five maneuvers with only a 4.22\% recurrence. Others have had less success with the liberatory maneuver, and some have claimed that it is too violent. Two common rehabilitation efforts used to treat BPV are the Brandt and Daroff exercises and the Epley canalith repositioning maneuver; these are described in further detail.\(^1,3\)

For those individuals not responding to rehabilitative exercises, surgical options may be considered.
Surgical procedures designed specifically for treatment of incapacitating symptoms due to BPV include singular neurectomy and posterior semi-circular canal occlusion (PSCC). These procedures are designed to eliminate responses from the PSCC either by deafferentation (singular neurectomy) or by preventing fluid movement through the posterior canal (occlusion). Patients considered candidates for surgery typically have had unrelenting or multiple recurrences of BPV emanating from the same ear.5

Singular neurectomy was initially described by Gacek. The singular nerve exits the lateral aspect of the internal auditory canal in the singular canal and courses inferiorly and posteriorly to the PSCC ampula. The intermediate segment of the nerve is inferior-posterior to the round window niche. In this location, it is possible to sever the nerve without damage to the remainder of the inner ear. Anatomic studies have reviewed the relationship of the nerve to the round window niche and have found that the nerve is lateral to the round window membrane in 50% of ears and medial in 14-27%. When the nerve lies medial, the risk of injury to the vestibule or basal turn of the cochlea is significant. While anatomic studies suggest a significant incidence of surgically inaccessible nerves, surgical series rarely document difficulty in locating or sectioning the nerve.5

The surgery is typically accomplished via a transcanal approach. The inferior scutum may need to be lowered for optimum viewing of the round window niche. The overhang of the wound window niche is then taken down to view the nerve during transection. Patients should have immediate resolution of positional nystagmus post-operatively, however most will have some spontaneous nystagmus, usually downbeating, which persists for a few days. Published success rates for the elimination of BPV are near 90%. Persistent symptoms are seen when the nerve cannot be unequivocally identified. Complications are few but include recurrent vertigo and sensorineural hearing loss. Severe SNHL occurs in at least 5% of patients, either as a consequence of direct trauma to the inner ear of postoperative labyrinthitis. Lesser SNHL may occur in additional 20% of patients. In general, this procedure should only be attempted by surgeons with considerable experience in the temporal bone lab and even then the procedure may prove extraordinarily difficult.5

The difficulty in singular neurectomy lead to the development of the PSCC occlusion procedure as an alternate. The procedure is based on the theory that elimination of endolymph flow in the canal will prevent any stimulation of the cupula with head motion. Prior observations in animal models have shown PSCC occlusion to have no effect on sensitivity of the remaining vestibular end-organs.5

A cortical mastoidectomy is performed via a post-auricular incision, the posterior semicircular canal is identified and blue-lined. The canal is then opened with a fine pick. The canal is occluded by either laser partitioning of the membranous labyrinth followed by packing of the canal versus merely compressing the membranous labyrinth and then packing with bone wax or bone dust and fascia depending on the surgeons preference. Results in limited series of patients are similar despite the method of occluding the canal.5,6 A transient SNHL occurs in most patients and can be detected intra-operatively by electrocochleography upon packing the canal, however this usually recovers within 6-8 weeks. However, mild SNHL occurs in up to 20% of patients. Immediately post-operatively patients will feel dysequilibrium that persists for days to weeks. The average duration of in-patient stay is 4.5 days. Recurrent positional vertigo is rare, however because this technique is relatively new long term follow-up is not yet available.5,6 In comparing singular neurectomy to PSCC occlusion, additional data is necessary to determine which procedure offers the best relief of vertigo long term and which procedure provides the least risk to hearing.5 In patients with failure of these procedures vestibular nerve section may be offered.

Meniere's Disease

Meniere's disease may be the most common diagnosis for which surgical intervention may be necessary. The most difficult task for the surgeon may be appropriate patient selection. The principle preoperative objectives are definition of the underlying disease state, localizing the site of lesion, quantification of the vertiginous episodes, and assessment of the patient's hearing. If surgery is contemplated, the patient should have full understanding of the expected postoperative course and likely sequelae of surgery, such as residual dysequilibrium. History and physical exam will usually lead to an accurate diagnosis. Keep in
mind that a complaint of constant imbalance warrants that a cause other than peripheral vestibulopathy be strongly considered. The differential of peripheral vestibulopathy includes Meniere's disease, trauma, iatrogenic, delayed endolymphatic hydrops, chronic vestibular neuronitis, labyrinthitis (cholesteatoma, chronic otitis media, viral, otosyphilis) vascular insult, BPPV, autoimmune inner ear disease, superior canal dehiscence syndrome. Concurrent symptoms of tinnitus or aural pressure, or the presence of nystagmus characteristic of peripheral disorder confers supportive evidence implicating the affected ear. Audiometry or caloric testing demonstrating SNHL or unilateral vestibular weakness will confirm the involved side. Some quantification of the severity of the patient's symptoms is necessary to compare different treatment modalities and justify surgical intervention. Since surgical intervention is designed to control vestibular symptoms and does not necessarily affect hearing, the functional scale for Meniere's disease provides a useful guideline. Theses guidelines are intended for use with definite and certain cases of Meniere's disease, to be defined momentarily, however probable, possible, and non-Meniere's cases may be reported using the same general guidelines but should be specified as such, and not mixed with data from definite and certain cases. The staging system for both the functional evaluation of Meniere's disease as well as hearing are presented below.¹ The status of the patient's hearing affects the choice of potential procedure and defines hearing preservation as a second objective. When the hearing is useful, labyrinth-destroying procedures are avoided. A number of factors must be addressed to determine what constitutes useful hearing. Consideration must be given to the likelihood of contralateral disease. For instance, an estimated 15-30% of patients with Meniere's disease will develop contralateral ear involvement depending on the length of follow-up. The pure-tone average after 5-10 yrs of follow-up reaches 50-60 dB, and speech discrimination scores reach a minimum of 53% after 13 years.⁷

In patients with fluctuating hearing loss, the best preoperative audiogram should be considered. Generally, thresholds better than 70 dB and speech discrimination scores of better than 20% warrant consideration of hearing preservation surgery. The ultimate decision on utility of hearing rests with the patient. Surgical procedures should be avoided on a patient's only hearing ear.¹

Four categories are used to define the accuracy of the diagnosis of Meniere's disease. "Definite" Meniere's disease requires two or more episodes of spontaneous rotational vertigo lasting 20 minutes or longer and audiometrically documented hearing loss on at least one occasion and tinnitus or aural fullness in the affected ear and other causes excluded. "Certain" Meniere's disease adds histopathologic confirmation to the "definite" criteria. "Probable" Meniere's disease is used when only one definitive episode of vertigo has occurred despite fulfillment of all other criteria in the "definite" category. "Possible" Meniere's applies to cases that represent cochlear or vestibular variants of Meniere's disease and for which other causes have been excluded.¹ ⁸

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**TABLE 160-3. Functional scale for Meniere's disease**

<table>
<thead>
<tr>
<th>Level</th>
<th>Patient's assessment of current status of overall function</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No effect on activities at all.</td>
</tr>
<tr>
<td>2</td>
<td>I have to stop what I am doing during an attack but may resume activities when it has passed. I continue to work, drive, and engage in most activities I choose without restriction. I have made no changes in my activities in order to accommodate the dizziness.</td>
</tr>
<tr>
<td>3</td>
<td>I have to stop what I am doing during an attack but may resume activities when it has passed. I continue to work, drive, and engage in most activities I choose, but I have had to make changes in my activities in order to allow for the dizziness.</td>
</tr>
<tr>
<td>4</td>
<td>I am able to work, drive, travel, take care of my family, or engage in most essential activities, but I must exert a great deal of effort to do so. I must constantly make adjustments in my activities and budget my energy. I am barely making it.</td>
</tr>
<tr>
<td>5</td>
<td>I am unable to work, drive, or take care of my family. I am unable to do most of the active things I used to do. Even essential activities must be limited. I am disabled.</td>
</tr>
<tr>
<td>6</td>
<td>I have been disabled for 1 year or longer, and/or I receive compensation because of dizziness or balance problems.</td>
</tr>
</tbody>
</table>

From Bailey's 2nd Edition

**TABLE 160-2. Staging of hearing in Meniere's disease**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Four-tone pure-tone average*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;26</td>
</tr>
<tr>
<td>2</td>
<td>26–40</td>
</tr>
<tr>
<td>3</td>
<td>41–70</td>
</tr>
<tr>
<td>4</td>
<td>&gt;70</td>
</tr>
</tbody>
</table>

*Calculated as the mean of thresholds at 0.5, 1, 2, and 3 kHz from the patient's worst audiogram in the 6 months preceding treatment. These guidelines were designed only for certain and definite cases of Meniere's disease.
Prior to surgery for Meniere's or other peripheral vestibulopathy, an MRI scan of the brain with attention to the posterior fossa should be obtained to rule out a cerebellopontine angle tumor or brainstem lesion. Additionally, a FTA-Abs to rule out syphilis should be obtained. Surgical intervention is reserved for those patients who continue to have disabling vertigo in spite of conservative management. Usually patients whose functional status is 4 or worse are considered for surgery, however, occasionally functional status 3 may be considered. Hearing preservation and hearing destructive alternatives are considered based upon the overall hearing in the affected ear and the hearing on the contralateral ear.

Chemical Labyrinthectomy

Transtympanic aminoglycoside labyrinthectomy was first described by Schuknecht in 1956. The approach was developed to avoid significant incidence of bilateral vestibular failure seen with intramuscular injection. Recently there has been renewed interest in the use of transtympanic gentamicin to produce chemical labyrinthectomy in cases of Meniere's not responsive to medical management.

Aminoglycoside injected into the middle ear cleft is absorbed through the round window and is directly toxic to cochlear and vestibular hair cells. Different aminoglycosides are cochleo versus vestibulo-toxic. Gentamicin and streptomycin being preferentially vestibulo-toxic. Many authors have looked into different treatment regimens for transtympanic gentamicin using different concentrations and treatment intervals.

The trend has been from more frequent dosing which increases the objective ablation of vestibular function, but also increases the risk of SNHL. Toth and Parnes compared multiple daily and single weekly gentamicin dosing regimens. They concluded that weekly dosing offers equivalent efficacy for control of vertigo and considerably less risk to hearing. Rauch and Oas found that more than four injections of 40 mg/mL in a single week produced profound SNHL in 2 of 4 patients. In general the goal of chemical labyrinthectomy has been complete ablation of the caloric vestibular response with preservation of hearing. However, recently partial chemical labyrinthectomy has been considered. Harner et al. recently prospectively studied patients who received one transtympanic treatment with follow-up in one month, patients with remaining symptoms were then given additional injections no more frequently than monthly. Of 43 patients with all but one being function level 3-5, 36 patients were function level 1 or 2 and 40/43 were level 1-3. Only 15 patients showed unilateral weakness of 61% or greater. There was no change in audiometric data from pre to post injection. There conclusion was that partial chemical labyrinthectomy produced improvement in symptoms with little or no risk to hearing.

The procedure is carried out in the office setting. Anesthesia of the tympanic membrane may be carried out with injectable lidocaine, topical lidocaine, or phenol. Several techniques have been described using tympanostomy tubes or wicks, however, for weekly or less frequent injections 25 gauge needle insertion through the tympanic membrane is used on a tuberculin syringe. The puncture can be made anterior inferior or posterior inferior and approximately .5 to .75 ml of gentamicin 40mg/mL or some other concentration usually diluted with bicarbonate is injected into the middle ear cleft. The amount seems unimportant as long as the round window is submerged. The patient is kept supine with the treatment ear up from 30-45 minutes and asked not to swallow to prevent egression of the solution down the eustachian tube.

Overall, transtympanic gentamicin therapy for Meniere's disease adds a significant new approach to the armamentarium of the treating physician. More studies need to be carried out to optimize the treatment regimens to optimize control of vertigo while not placing residual hearing at risk.

Endolymphatic Sac Procedures

Portmann originally described the opening of the endolymphatic sac to relieve pressure in the treatment of Meniere's disease. It was later that histiologic evidence confirmed dilation of the endolymphatic spaces in cadaver specimens supporting Portmann's theory of endolymphatic hypertension. Later histopathologic studies showed intralabyrinthine membrane ruptures, fibrosis, or obstruction of the endolymphatic, saccular and utricular ducts. These findings seemed to be contrary to the successful theory of endolymphatic sac opening, stenting, or shunting. The proposed causes of endolymphatic hydrops have been equally as
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The proposed causes of hydrops include infection, autoimmune dysfunction, vascular insufficiency, and altered endolymph production and/or resorption. Additionally, the mechanism of sac surgery is debated as well as its efficacy. Multiple variations on Portmann's technique have been described including endolymphatic-subarachnoid shunting, sac decompression, sac excision, and the use of endolymphatic-mastoid stents. Summarized data from a number of reports make the observation that the mean success rate is near 75% regardless of technique.1,5,7

There are no controlled studies to document the efficacy of sac surgery. This is mostly due to the difficulty in providing the appropriate control group. This is compounded by the unpredictability and fluctuations of symptoms during the natural course of the disease. Several studies however deserve mention. Bretlau, Thomsen et. al. in 1981 performed a prospective, blinded comparison of 15 patients who underwent simple mastoidectomy, control group, with 15 patients who underwent an active mastoid shunt procedure. They concluded no difference in control of vertigo between the two groups at yearly intervals with up to 9 years of follow-up.12 This same data was then reviewed by Welling and Nagaraja and published in 2000 with quite different conclusions. Welling concluded that there was a statistically significant difference between the active and the control group in vertigo symptoms as well as several other symptoms.13 Thomsen then published a follow-up study comparing endolymphatic shunt with typanostomy tube insertion. They found no difference in vertigo reduction at one year between the two procedures.14 Silverstein et. al. compared in a retrospective fashion three groups of patients eligible for surgery to control vertigo from Meniere's disease. Each patient was offered endolymphatic sac surgery or vestibular nerve section, those that declined surgery functioned as controls and presumably demonstrated the natural history of Meniere's disease. Control patients experienced elimination of vertigo in 57% of cases at 2-year follow-up and 71% at last follow-up, averaging 8.3 years. Patients undergoing sac surgery had 40% complete control at 2 years and 70% at last follow-up, averaging 8.7 years. Vestibular neurectomy patients had complete control in 93% of cases at 2 years.5,15 It would appear from these studies that the benefit of sac surgery is limited in duration and perhaps follows the natural history of the disease. Given the availability of other procedures, which are less invasive, perhaps they should be considered over sac surgery.

The surgery is performed via a post-auricular incision. A complete mastoidectomy is performed with the retrofacial aircell tract widely opened with the boundaries of dissection being the jugular bulb anteroinferiorly, the facial nerve laterally, and the PSCC superiorly. All the bone is removed from the posterior fossa dura anterior to the sigmoid sinus. The dura will appear thickened in the region of the sac, as these two structures overlap. Once identified the sac is opened, stented, or excised according to the surgeon's preference. The surgery is carried out on an outpatient basis, and unlike other procedures for Meniere's the patient is usually not vertiginous following the operation. Complications although rare include, SNHL, CHL due to bone dust entering the middle ear, facial nerve injury, cerebrospinal fluid leak, or bleeding from the sigmoid sinus or jugular bulb.3

Selective Vestibular Nerve Section

Sectioning of the eight cranial nerve for vertigo was described initially around the turn of the 20th century. The high incidence of facial nerve paralysis and hearing loss associated with the procedure limited its popularity. Selective vestibular nerve section techniques via the middle fossa approach described by House in 1961 and the retrolabyrinthine approach described by Brackmann and Hitselberger and popularized by Silverstein in 1978 renewed interest in this procedure for the control of vertigo and preservation of hearing in cases of incapacitating vertigo. More recently the retrosigmoid and the retrosigmoid-IAC approaches have been described and compared to the other two procedures.

The routine use of perioperative antibiotics and intraoperative facial nerve monitoring and cochlear nerve monitoring is strongly suggested. Initially the patients are monitored in the ICU with particular attention to neurologic status and control of hypertension. Vestibular symptoms are managed with intravenous droperidol. The patient may be transferred to the regular nursing floor on post-operative day number one or two. The patient is observed for potential CSF leak and meningitis. Early ambulation is encouraged and the patient may be discharged home when able to ambulate without assistance and tolerate a regular diet.5,16
Middle fossa VNS proceeds via a 4X4 cm temporal craniotomy centered slightly anterior to the external auditory canal. The middle fossa dura is elevated from posterior to anterior along the medial border of the petrous ridge. Temporal lobe elevation is maintained with the House middle fossa retractor. Several methods may be employed for identification of the internal auditory canal (IAC), utilizing the greater superficial petrosal nerve, malleus head, and superior semicircular canal as the initial landmarks. Bone over the superior aspect of the IAC is removed allowing approximately 180 degrees of exposure. The dura is incised posteriorly to reduce risk of facial nerve injury. The superior and inferior vestibular nerves are sectioned laterally in the IAC. Care must be taken to include the singular nerve. The facial and cochlear nerves lie anteriorly. Once neurectomy is complete, a piece of muscle or fat is used to plug the dural opening. The temporal lobe is allowed to return to its normal position. Tacking sutures from the edges of the craniotomy to the dura limits the possibility of epidural hematoma.\(^5\)

Retrosigmoid and retrolabyrinthine operations commence with a postauricular incision placed much further posteriorly than that used in routine mastoid surgery. The retrosigmoid craniotomy is placed just posterior to the sigmoid sinus and inferior to the transverse sinus, measuring 4X5 cm. Retrolabyrinthine craniotomy requires a complete mastoidectomy and removal of bone posterior to the PSCC to a point 1 to 2 cm posterior to the sigmoid sinus. Following dural incisions and release of CSF, the cerebellum may be gently displaced posteriorly, often without the need for retraction. The sigmoid sinus may be retracted (anteriorly in retrosigmoid, posteriorly in retrolabyrinthine) to improve the exposure of the porus acusticus. In this location, the vestibular portion of the eighth nerve is superior to the cochlear portion. A cleavage plane is visible in approximately 75% of patients, allowing separation of the two portions. Inability to define the cleavage plane necessitates drilling away bone from the medial lip of the porus to expose the nerves further laterally. An abdominal fat graft is used to obliterate the mastoid and prevent CSF leak in the retrolabyrinthine approach. The dura is more easily closed in the retrosigmoid approach, and a fat graft is unnecessary. A layered skin closure is performed followed by placement of a pressure dressing.\(^5\)

Comparing the three general approaches, middle fossa, retrolabyrinthine, and retrosigmoid, there are some differences depending upon author and approach. Overall complete elimination of vertigo is achieved in 90% of patients utilizing the middle fossa approach. With the posterior approaches complete elimination of vertigo is achieved in over 80% and substantial improvement in vertigo symptoms are achieved in over 95%. McKenna et. al, and Glasscock et. al. compared the retrosigmoid and retrolabyrinthine approaches. McKenna concluded that the retrosigmoid-IAC approach resulted in slightly less recurrent postoperative vertigo than did the retrolabyrinthine approach.\(^15\) While Glasscock found no difference in control of vertigo they preferred the retrosigmoid approach because of better visualization of the neurovascular structures.\(^18\) Silverstein compared middle fossa approach with the other two and concluded that the retrosigmoid-IAC approach provides better and more complete denervation of the vestibular labyrinth than the retrolabyrinthine approach and is easier to master than the middle fossa approach.\(^19\) Another study by Silverstein interviewing 350 members of the American Otologic society and American Neurotology Society found that 92% of vestibular nerve sections were being performed through a posterior approach.\(^20\)

Complications most commonly associated with VNS include dysequilibrium, headache, hearing loss, and CSF leak. Dysequilibrium may occur in as many as 30% of patients and is a result of inadequate central compensation. Usually this does not limit activities, although rarely it may be debilitating. Hearing loss as a consequence of surgery is uncommon. Wound infection and facial paralysis occur in less than 5% of cases, and meningitis, intracranial hemorrhage, and stroke are even more rare and can be avoided by control of hypertensive crisis and timely recognition of altered mental status. Specific to approach, the middle fossa approach has been associated with greater incidence of facial nerve injury and total hearing loss thought to be due to injury to the labyrinthine artery. Memory disturbances have been reported and caution must be exercised in elderly patients to the adherence of the dura to the middle cranial fossa and the increased risk of subdural hematoma.\(^5\) Retrolabyrinthine approaches are associated with an increased risk of CSF leak, conductive hearing loss if bone dust enters the middle ear, and requires an abdominal fat graft. Additionally, this approach may have a lower success rate probably due to the lack of a cleavage plane at the porus acusticus. Headache is more common with the retrosigmoid approach and has been postulated to be due to the greater release of occipital musculature. However, one report noted that the incidence was much greater if the internal auditory canal had been drilled out.\(^5,16\)
Labyrinthectomy

Labyrinthectomy is the final surgical option for the control of vertigo. Described originally in 1904, it can be performed as a transcanal or transmastoid procedure. It is reserved for patients with non-serviceable hearing with general guidelines being a PTA of 70 db and a discrimination score of 20%. However, the patient is the ultimate determinate of what is serviceable.

Transcanal labyrinthectomy may be performed under local or general anesthesia. Surgery begins with creation of a typanomeatal flap. The incudostapedial joint is disarticulated, and the incus is removed. Next, the stapedius tendon is divided, and the stapes is removed. The vestibule is drained of perilymph, which may induce significant vertigo. The oval window is enlarged with a drill or a curette, allowing improved visibility and room to introduce additional instruments. The saccule may be removed easily under direct vision. The utricle lies superiorly in the oval window, medial to the facial nerve. It is avulsed using a sweeping motion with a 3 mm right-angled hook. The hook is then used to probe the ampullated ends of the semicircular canals. It is difficult to extract the canal ampullae, but severe damage should result from manipulation with the hook. Gelfoam soaked in an ototoxic medication may then be packed into the vestibule.

The surgical objective of transmastoid labyrinthectomy is to completely excise all five end-organs. The initial step is performance of a complete mastoidectomy via a postauricular approach. It is important to visualize the facial nerve in the tympanic segment and at the second genu to avoid injury. Perilabyrinthine air cells are exenterated to define the semicircular canals. The lateral canal is entered on its superior aspect to minimize chance of facial nerve injury. The superior canal is entered on its posterior aspect. the ampullae of these two canals are adjacent and are located superior to the vestibule. After these two structures are avulsed, the vestibule is enlarged medially and posteriorly, providing wide access for removal of the utricle and saccule. The lateral wall of the vestibule must be respected to avoid facial nerve injury. The vestibulotomy is carried further posteriorly, medial to the second genu to identify the ampullated end of the posterior canal. finally this structure is removed. The wound is closed in layers and a mastoid dressing is placed.

The postoperative course of labyrinthectomy is independent of the operative approach. Horizontal nystagmus accompanied by vertigo may be severe, and the patient may need anti-emetics. Once again early ambulation is important for compensation. A brief hospitalization is required with discharge dependent on ambulation and oral intake.

The reported results of labyrinthectomy are complete elimination of vertigo in 85% of patients regardless of technique as long as they are performed correctly. Labyrinthectomy combined with vestibular nerve section has been postulated to provide even greater control of vertigo, but a study by Langman and Lindeman showed no difference to conventional labyrinthectomy. Complications are rare and include wound infection, hemorrhage and facial nerve injury. If translabyrinthine-VNS is contemplated the additional risk of CSF lead and meningitis must be considered. The incidence of post-operative dysequilibrium is 30% and is usually mild to moderate.

Superior Canal Dehiscence Syndrome

Minor recently described a syndrome of sound and/or pressure induced vertigo due to dehiscence of bone overlying the superior semicircular canal. The patients complain of vertigo induced by loud noises (tullio phenomenon) or maneuvers that change intracranial pressure such as sneezing, coughing, valsalva maneuvers, lifting, and autoinsufflation. These patients develop vertical-torsional eye movements (closely aligned with the plane of the superior semicircular canal on the affected side) with the fast phase toward the affected ear in response to positive pressure or loud noise. They may also experience constant and often disabling dysequilibrium and unsteadiness. The mechanism is such that the dehiscence of bone overlying the superior canal creates a third mobile window into the inner ear. Motion of endolymph occurs as a result of this third mobile window and results in deflection of the cupula of the superior canal. Positive pressure in the external canal causes excitatory stimulus with the fast phase toward the affected side and
with maneuvers that increase intracranial pressure develop inhibitory stimulus with fast phase to the opposite ear. The diagnosis can be confirmed with high resolution temporal bone CT scanning.22

Carey conducted a microscopic study of 1000 temporal bones from 596 adults and found that the incidence of complete dehiscence of the superior canal was identified in 5 specimens (0.5%). One was at the middle fossa floor and four were where the superior petrosal sinus was in contact with the canal. In 14 other specimens (1.4%), the bone at the middle fossa floor (n=8) or superior petrosal sinus (n=6) was no thicker than 0.1 mm, significantly less than values measured in the control specimens, and thin enough that it might appear dehiscent on ultra-high-resolution CT scanning. Abnormalities were typically bilateral. Additionally, specimens from infants demonstrated uniformly thin bone over the superior canal in the middle fossa at birth, with gradual thickening until 3 years of age. This lead to the theory that these abnormalities may arise from failure of post-natal bone development.23

Symptomatic patients are counseled to avoid the offending stimuli, which Minor found affective in 10/17 patients. Debilitating symptoms not amenable to avoidance are offered surgical therapy. The surgery is performed via a middle cranial fossa approach as previously described. The dura over the arcuate eminence must be performed carefully to avoid damage to the membranous labyrinth. The canal defect is repaired either by resurfacing the canal with fascia and bone dust or sculpted cortical bone or the lumen is occluded with bone dust or other material. The optimal surgical procedure for this disorder is yet to be determined.5,23

Conclusion

Both rehabilitative and surgical management of vestibular disorders require correct diagnosis. Medical and/or rehabilitative options should be attempted prior to considering surgical options. Diagnosis such as BPPV have relatively straight forward surgical management. Additionally, peripheral disorders other than Meniere's disease have essentially two surgical options, vestibular nerve section or labyrinthectomy, dependent on hearing. Meniere's disease creates more of a controversy both secondary to the lack of understanding of its causes and physiology, but also because of the lack of ability to produce conclusive studies of surgical outcomes due to the natural history of the disease. Sac surgery continues to be the primary choice by some, while others would condemn the technique. Vestibular nerve section then becomes the next surgical option followed by labyrinthectomy for non-serviceable hearing. Chemical labyrinthectomy may soon play a greater role in this therapy for both Meniere's and non-Meniere's syndromes and could even conceivably be used for non-serviceable hearing patients. Lastly, new discoveries continue to be made and as the causes and physiology of disease continues to be elucidated medical and surgical therapy will continue to evolve.

References