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Posterior canal occlusion for benign paroxysmal positional vertigo ☆

Peng You, MD, Ryan Instrum, MD, Sumit K. Agrawal, MD, FRCSC, Lorne S. Parnes, MD, FRCSC

From the Department of Otolaryngology, Head and Neck Surgery, Schulich School of Medicine and Dentistry, Western University, London, Ontario, Canada

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Benign paroxysmal positional vertigo is the most common etiology of peripheral vertigo and is caused by the hydrodynamic influence from free-floating canaliths. Any of the 3 semicircular canals can be involved, but posterior canal BPPV is the predominant subtype. The condition is diagnosed clinically, and the preponderance of cases resolve spontaneously or are amenable to particle-repositioning maneuvers. A small subset of patients experience intractable symptoms for which surgical intervention can be considered. Transmastoid posterior canal occlusion surgery has been demonstrated to be a safe and curative procedure insofar as it can eradicate vertiginous symptoms while preserving auditory function.

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular end organ disorder and a prevalent cause of episodic vertigo.[1] The pathophysiologic mechanism associated with the condition chiefly involves altered semicircular canal endolymph hydrodynamics via the presence of free-floating calcium carbonate crystals (canalithiasis) or adherence of this particulate matter to the cupula with resulting cupular loading (cupulolithiasis).[2,3]

These particles, known as canaliths, are normally embedded in and fixed to the otolithic membrane of the utricle. If these canaliths become detached from their native substrate, by way of traumatic or degenerative forces, they are free to travel passively throughout the vestibular labyrinth.[4,5] Since they are denser than their surrounding endolymph, their migration is influenced by gravity.

The canalithiasis theory is supported by scanning electron microscopy images of intracanalicular particles extracted during posterior canal occlusion procedures (Figure 1). The images show displaced canaliths of varying degrees of degeneration with attached otolithic membrane fragments.[6] The canaliths typically migrate to the posterior semicircular canal because it is the most gravity-dependent portion of the apparatus. In contrast, should the canaliths migrate into the superior or horizontal semicircular canals, these canals' orientation allows for particles to return to the utricle fairly quickly with natural head movements.

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Address reprint requests and correspondence: Lorne S. Parnes, MD, FRCSC, Department of Otolaryngology Head and Neck Surgery, Schulich School of Medicine and Dentistry, Western University, London Health Sciences Centre, University Hospital, 339 Windermere Road, PO Box 5339, London, Ontario N6A 5A5, Canada.

E-mail address: Lorne.Parnes@lhsc.on.ca

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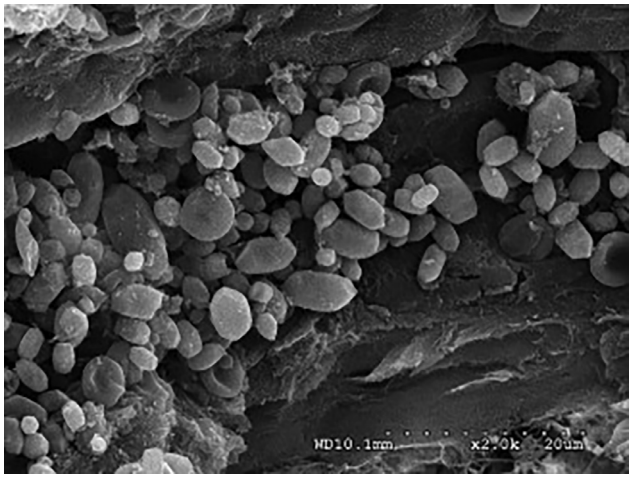

Figure 1 Scanning electron micrograph image of canaliths within the posterior canal endolymphatic duct removed from a patient with intractable BPPV. Image shown at 20k magnification. Scale bar 20 μm. *Reproduced with permission from Kao, Parnes, and Chole. Otoconia and otolithic membrane fragments within the posterior semicircular canal in benign paroxysmal positional vertigo. Laryngoscope 90:709-714 (2016).*

Nonsurgical management of BPPV focuses on returning these particles to the utricle through a specific series of particle repositioning maneuvers which are successful in most cases.[7] Numerous variations of particle repositioning maneuvers exist in the literature and are derivations of the original maneuvers described by Epley and Semont.[8,9] For intractable cases that cannot be treated conservatively, surgical occlusion of the posterior canal can be considered.

Prior to the 1980s, invasive inner ear surgery in normal hearing ears was considered ill-advised due to the perceived risk of enduring sensorineural hearing loss secondary to cochlear insult.[10] A paradigm shift occurred when Parnes and McClure demonstrated that posterior canal occlusion did not result in permanent hearing loss in an animal model, and subsequently in humans with intractable BPPV.[11] The premise of the posterior canal occlusion is that endolymph movement can be reduced by compressing the membranous labyrinth, effectively stiffening the cupula. In doing so, the high-frequency acceleration vestibulo-ocular reflex response in the plane of the canal is muted while the low-frequency response of canalithiasis or cupulolithiasis is completely eliminated.[12] Posterior canal plugging is a safe procedure and is essentially curative for BPPV given appropriate case selection.[13]

Signs and symptoms

Patients with BPPV typically present with severe and abrupt onset of rotary vertigo precipitated by certain head movements.[14] Common provocative motions include rolling toward the affected ear while supine, as well as neck extension. These vertiginous episodes typically last less than 30 seconds, although patients may be prone to overestimation due to the lingering disequilibrium and nausea.[15] While BPPV carries a favourable prognosis, with nearly 25% of patients having a spontaneous resolution by 1 month and up to 50% at 3 months, recurrence is common.[16,17]

While the preponderance of BPPV cases is of the primary or idiopathic variety, a thorough history should be taken to exclude secondary causes.[5] These may include head trauma, Ménière's disease, migraine, viral labyrinthitis, vestibular neuritis, or previous otologic or nonotologic surgery.[18-23] Moreover, vertigo originating in the central nervous system such as with multiple sclerosis, neoplastic processes, and cerebrovascular accidents must be ruled out so as not to delay the implementation of appropriate treatment for these more sinister causes.[24-28]

Diagnostic tests

Posterior canal BPPV is diagnosed with the Dix-Hallpike maneuver. The maneuver begins with the patient seated and head turned 45° to the side being tested. This isolates and vertically orients the ipsilateral posterior canal.[3] The patient is then laid back quickly into a supine position with the tested ear down. Traditional teaching has the head hanging back over the edge of the bed, but this is not necessary for the Dix-Hallpike test alone and can elicit a false positive response from the contralateral side.

In a patient with posterior canal BPPV, the nystagmus elicited with the Dix-Hallpike maneuver is typically preceded by a brief latent period (2-5 seconds). The nystagmus usually lasts no more than 20 seconds and consists of both an up-beating and torsional component with the top pole of the eye beating toward the affected ear. The nystagmus reverses direction as the patient is returned to the upright position and the response to the maneuver will weaken with repeated testing. The intensity of the nystagmus usually correlates with the severity of the patient's vertigo during the test.[29]

Patients with a compelling history and characteristic nystagmus on provocative maneuvers are readily diagnosed with BPPV. Advanced electrophysiologic vestibular and auditory tests are rarely indicated for isolated BPPV.[30,31] If concurrent symptoms or atypical findings raise suspicion for other inner ear or posterior fossa abnormalities, further investigations should be arranged accordingly.

Patient selection

Indications

The majority of BPPV cases resolve either spontaneously or with appropriate repositioning maneuvers.[16] Operative intervention is reserved for patients with intractable BPPV whose severe symptoms interfere with their occupation or daily life.[13] Surgical candidates must have failed conservative management including vestibular rehabilitation and/or multiple particle repositioning maneuvers performed by an experienced clinician. Sometimes the

procedure is done for patients with very frequent and severe recurrences.

Contraindications

As with every otologic surgery, there are possible risks to the auditory and vestibular system.[32] With BPPV being a benign condition, patient selection is essential in minimizing the risks. To this end, the procedure is not recommended in an ear possessing the only vestibular function or the only or significantly better hearing ear. Furthermore, local anatomical concerns may preclude patients from surgery if they exhibit abnormal temporal bone anatomy that renders the posterior canal inaccessible via a transmastoid approach.[29] Ongoing otomastoiditis or other local infections would also prevent surgical intervention until such time that the infection has fully resolved.

Preoperative planning

While the diagnosis of BPPV is made through clinical history and provocative maneuvers, additional preoperative investigations are required to confirm the diagnosis, understand surgical risk, and assist with surgical planning. Preoperative evaluation includes a routine audiogram for postoperative comparison. Videonystagmography or video head impulse testing is used to document normal vestibular response in the contralateral ear. A high-resolution computed tomography (CT) scan is used to define the temporal bone anatomy, in addition to affirming that a transmastoid approach can be used to access the posterior canal. Preoperative magnetic resonance imaging or CT is used to rule out the rare central lesion that mimics the symptoms of intractable BPPV.[25,26]

Surgical technique

The surgical set up includes perioperative antibiotics, corticosteroids, and intraoperative monitoring. The use of perioperative broad-spectrum antibiotic coverage, although not required, is common. Antibiotic prophylaxis should be strongly considered for ears with a history of otitis media. An intraoperative dose of IV corticosteroids may be used to reduce nausea related to surgery and has the potential to decrease the duration of transient postoperative vestibular and cochlear dysfunction. Intraoperative facial nerve monitoring is standard, but auditory nerve monitoring is not required.

The procedure is performed under general anesthesia and should take no longer than 1 to 2 hours for an experienced otologic surgeon. The patient is placed in a supine position with the head turned 45° toward the contralateral side. The incision line designed 1 cm behind the postauricular sulcus is infiltrated with long-acting local anesthetic with epinephrine. Surgical site preparation and draping are performed in a routine fashion.

The procedure begins with a limited mastoidectomy. Typically, adequate exposure requires a 5-6 cm postauricular incision. Fluted ball drill and suction irrigation are used to open up the antrum, as is routine for mastoid surgery. This allows identification of the lateral semicircular canal. Some dry mastoid cortex bone chips are collected and set aside for later use.

Adequate exposure is often possible without having to define the digastric ridge or the tegmen. First, the antrum and lateral semicircular canal are identified. The bony sigmoid sinus prominence is identified next, but it is usually not necessary to bare the sigmoid sinus dura. Next, bone removal proceeds anteriorly from the sigmoid sinus along the cerebellar plate toward the posterior semicircular canal. When the posterior canal otic capsule is defined, the bone is "blue-lined" with a 2 mm diamond burr and adequate irrigation. This entails thinning the otic capsule bone so much that the microscope light is absorbed (not reflected) through the thin bone into the underlying perilymph, thus creating a dark appearance or so-called "blue-line" (Figure 2).

The target zone for the occlusion is the area at, or just inferior to, the Donaldson's line (an imaginary line extending posteriorly from the course of the lateral semicircular canal). Sometimes there are abundant air cells between the posterior canal otic capsule bone and the dura, and sometimes the bony canal directly contacts the dura. Hence, this step should be approached with caution, and the review of the preoperative CT scan is critical.

In order to breach the perilymph, the bony covering must first be removed. A 1 mm diamond burr is used to skeletonize a 3 mm in length segment of the canal 180° around the outer circumference down to endosteum (Figure 2). This creates a 1×3 mm endosteal island. Bone removal should proceed evenly so that there would be no further drilling when the endosteum is violated, and the perilymph is exposed.

A fine 90° pick is used to remove the endosteal island, which creates a fenestra and exposes the perilymph (Figure 3). At this stage, the exact outline and limits of the membranous labyrinth may not be easily discernible. Perilymph should be gently "wicked" away with a Cottonoid, or gently suctioned with a No. 24 suction with the thumb off the hole to expose the membranous labyrinth, at which time the membranous duct can be seen to collapse. It is essential not to suction directly on the perilymph and especially the membranous labyrinth for fear of disrupting it. Some perilymph needs to be removed to provide space for the ensuing plug.

Various materials have been used to occlude the canals, including bone dust alone, bone wax, fascia or other tissue, and bone pâté made from either bone dust and blood or bone dust and fibrin glue. Our approach is to mix dry bone chips gathered from the mastoidectomy with a few drops of 2-component, fast-acting human fibrinogen glue. Once set (about 30 seconds), it forms an easily workable, but malleable plug with a firm consistency.

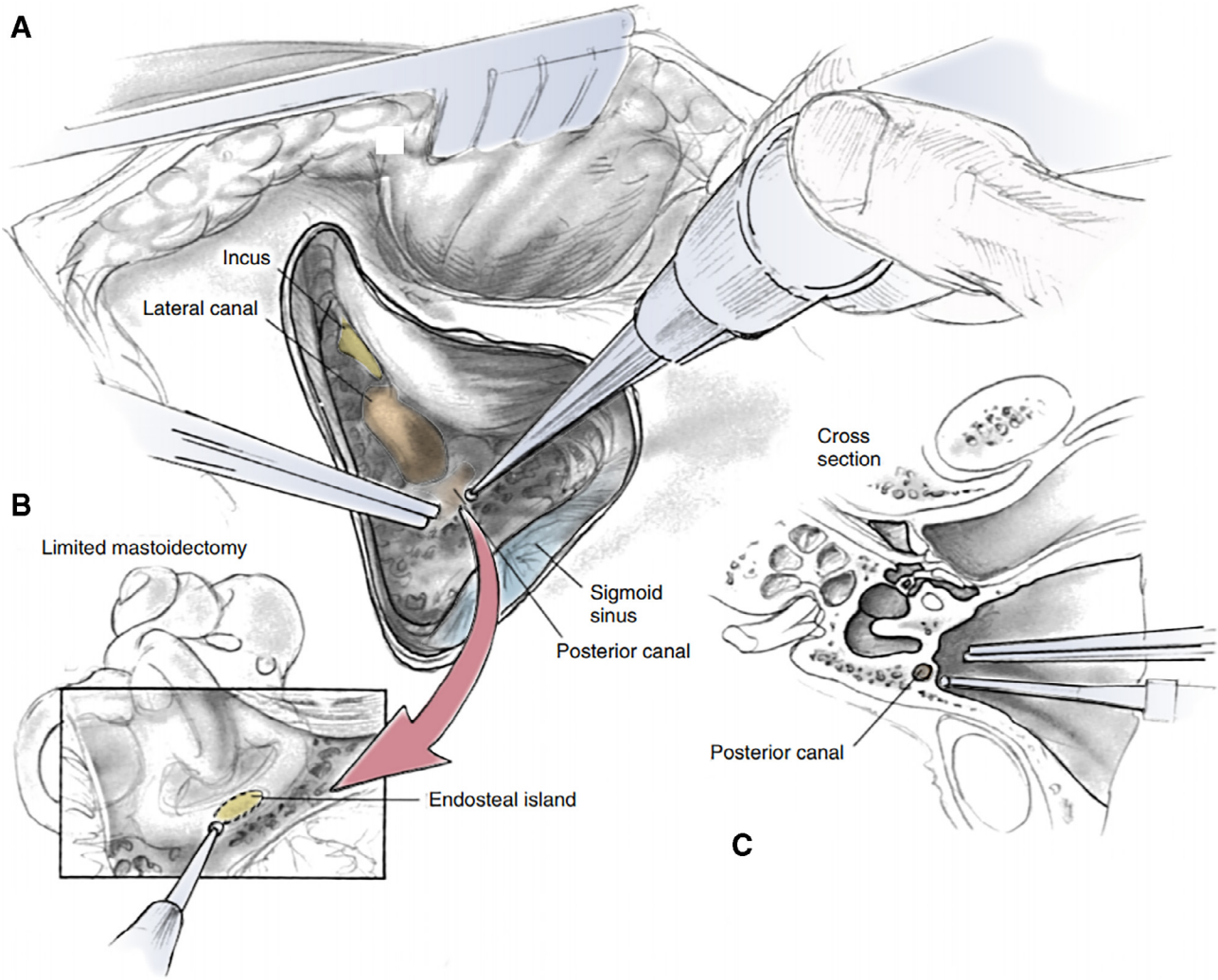

Figure 2 Surgical exposure of posterior semicircular canal. (A) Exposure of posterior semicircular canal otic capsule using limited mastoidectomy. (B) Creating 1 × 3 mm endosteal island with a small diamond burr. (C) Cross-section view. *Reproduced with permission. This figure was published in Otologic Surgery 4th edition by Brackmann, Shelton, and Arriaga.*[29]

A plug slightly larger than the fenestra is fashioned and then gently and firmly inserted through the fenestra with the intention of filling the canal lumen and compressing the membranous labyrinth closed. A blunt 45° probe is used to pack the plug tightly in the canal (Figure 4). With time, the bone chips within the plug lead to intracanal ossification resulting in complete permanent occlusion of the canal.

After completing the plug insertion, a tissue seal helps to further safeguard against a postoperative perilymph fistula. This is accomplished by covering the fenestra and surrounding bone with a piece of temporalis fascia, which is kept in place by several more drops of fibrinogen glue (Figure 5). The incision is closed in 2 layers, and a standard mastoid dressing may be applied. A drain is unnecessary.

Postoperative care

Most patients are hospitalized for 1 to 3 days following the procedure. Posterior canal occlusion does not disturb the resting neuronal discharge from the occluded canal. Therefore, most patients do not have spontaneous postoperative vertigo or nystagmus at rest, unless complicated by other factors. The dynamic vestibular asymmetry caused by the canal occlusion commonly results in early postoperative disequilibrium and motion sensitivity.[29] Over time, central adaption relieves the initial postoperative imbalance. Compensation typically occurs within a few days to weeks, but vestibular physiotherapy can help expedite the process.[33] Vestibular suppressants are not used routinely as they may provide short-term relief at the expense of prolonging the overall recovery.

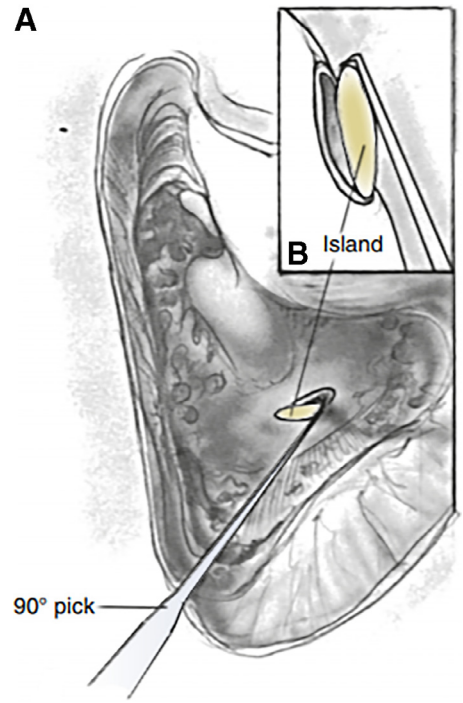

Figure 3 (A) Lifting out the endosteal island with a fine 90° pick. (B) Magnified lateral view. *Reproduced with permission. This figure was published in Otologic Surgery 4th edition by Brackmann, Shelton, and Arriaga.*[29]

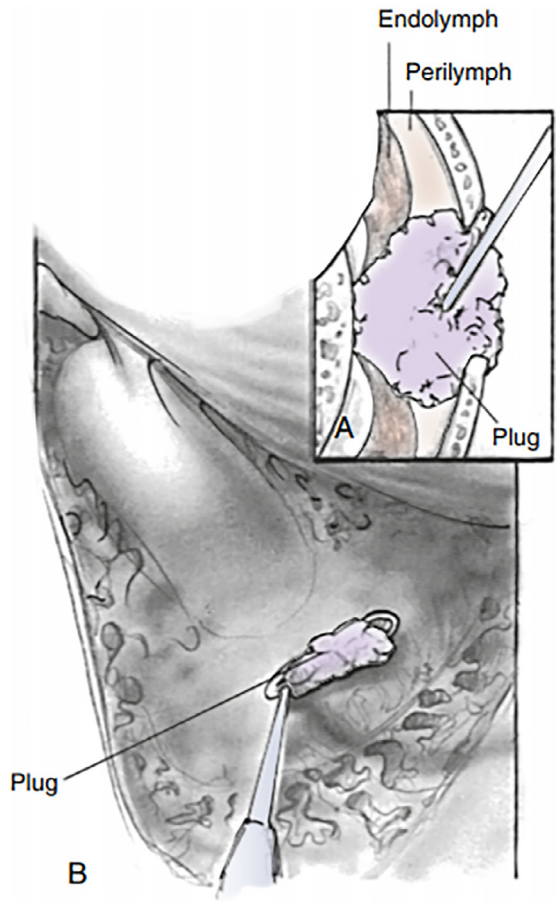

Figure 4 (A) Tamping plug through the fenestra into the canal. (B) A cross section of the canal shows the intact but occluded membranous canal. *Reproduced with permission. This figure was published in Otologic Surgery 4th edition by Brackmann, Shelton, and Arriaga.*[29]

Complications

In the early postoperative period, mixed hearing loss and vestibular symptoms are to be expected with few patients experiencing prolonged symptoms. Protracted vestibular symptoms generally return to near baseline over the course of a few months with diligent vestibular rehabilitation.[29] Most other potential complications can be averted with adequate preoperative surgical planning and sound technique. These include infection, facial nerve injury, dural tears, and hematoma, all of which are rare occurrences.

Of note, in our experience, 1 patient demonstrated a perilymph gusher from the fenestrated posterior canal. Performing the occlusion in the standard fashion stopped the perilymph leak, and there were no extraordinary postoperative issues.

Discussion

Posterior semicircular canal occlusion has been shown to be both a safe and effective surgical therapy for intractable posterior canal BPPV insofar as it is essentially curative and is associated with mild side-effects that are generally self-limiting.[13] The procedure is comparatively straightforward relative to other techniques, and serious complications are uncommon given sufficient preparation.[34]

As of 2018, our group at London Health Sciences Centre has performed 82 posterior semicircular canal occlusions.[13,29] These cases involved 76 patients, 6 of whom underwent staged occlusions for bilateral disease. Each of the 82 procedures yielded a complete symptom resolution, with only 1 patient experiencing atypical positional vertiginous symptoms many years after her occlusion surgery. In the immediate postoperative period, all patients experienced a mixed hearing loss but subsequently made full recoveries to their baseline. Only 1 patient had sustained deficits, and this occurred 3 months postoperatively. An intense 2-day headache preceded sudden, profound, permanent sensorineural hearing loss along with 7 days of concurrent vertiginous symptoms. Labyrinthitis was the working diagnosis. This patient had previously undergone 2 unsuccessful attempted singular neurectomies in the same ear by another surgeon. To have undergone a third procedure illustrated that her symptoms clearly incapacitated her. Though we were disheartened by her "dead" ear even though the direct causality was never proven, she was pleased that her positional vertigo was finally cured.

The principles of canal occlusion discussed here can also be applied to any of the semicircular canals (as

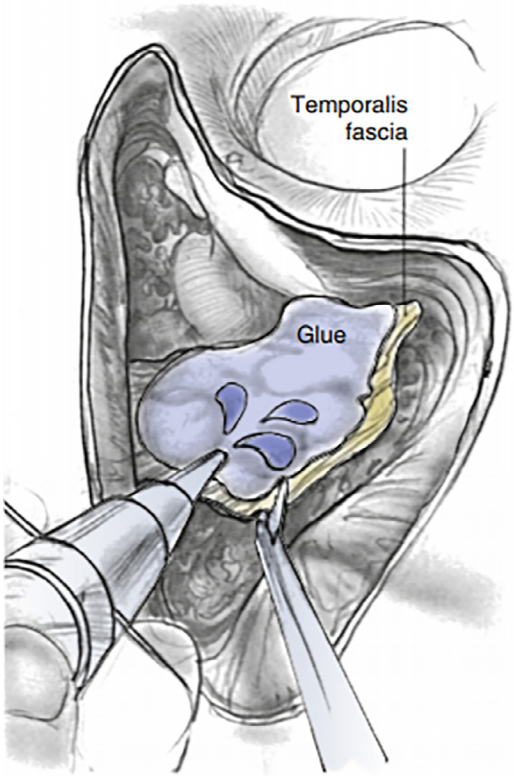

Figure 5 Covering the fenestra and surrounding bone with fascia and glue. *Reproduced with permission. This figure was published in Otologic Surgery 4th edition by Brackmann, Shelton, and Arriaga.*[29]

well as bilateral disease). Reports of canal occlusion in the horizontal and superior canals have shown comparable efficacy.[35-38] This underscores the significance of diagnostic accuracy in the identification of BPPV subtype when considering definitive surgical intervention. Other published adaptations to the technique include the use of lasers to produce fibrous occlusions within the canal.[39,40]

Summary

BPPV is the most common etiology of peripheral vertigo and is caused by the hydrodynamic influence of free-floating canaliths. Any of the 3 semicircular canals can be involved, but posterior BPPV is the predominant subtype. The condition is diagnosed clinically, and the majority of cases resolve spontaneously or are amenable to particle-repositioning maneuvers. A small subset of patients experience intractable symptoms for which surgical intervention can be considered. Transmastoid posterior canal occlusion surgery has been demonstrated to be a safe and curative procedure. The surgery can eradicate vertiginous symptoms while preserving auditory function. Moreover, occlusive interventions in the superior and horizontal semicircular canals, as well as in those with bilateral disease, have shown similar efficacy.

Disclosure

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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