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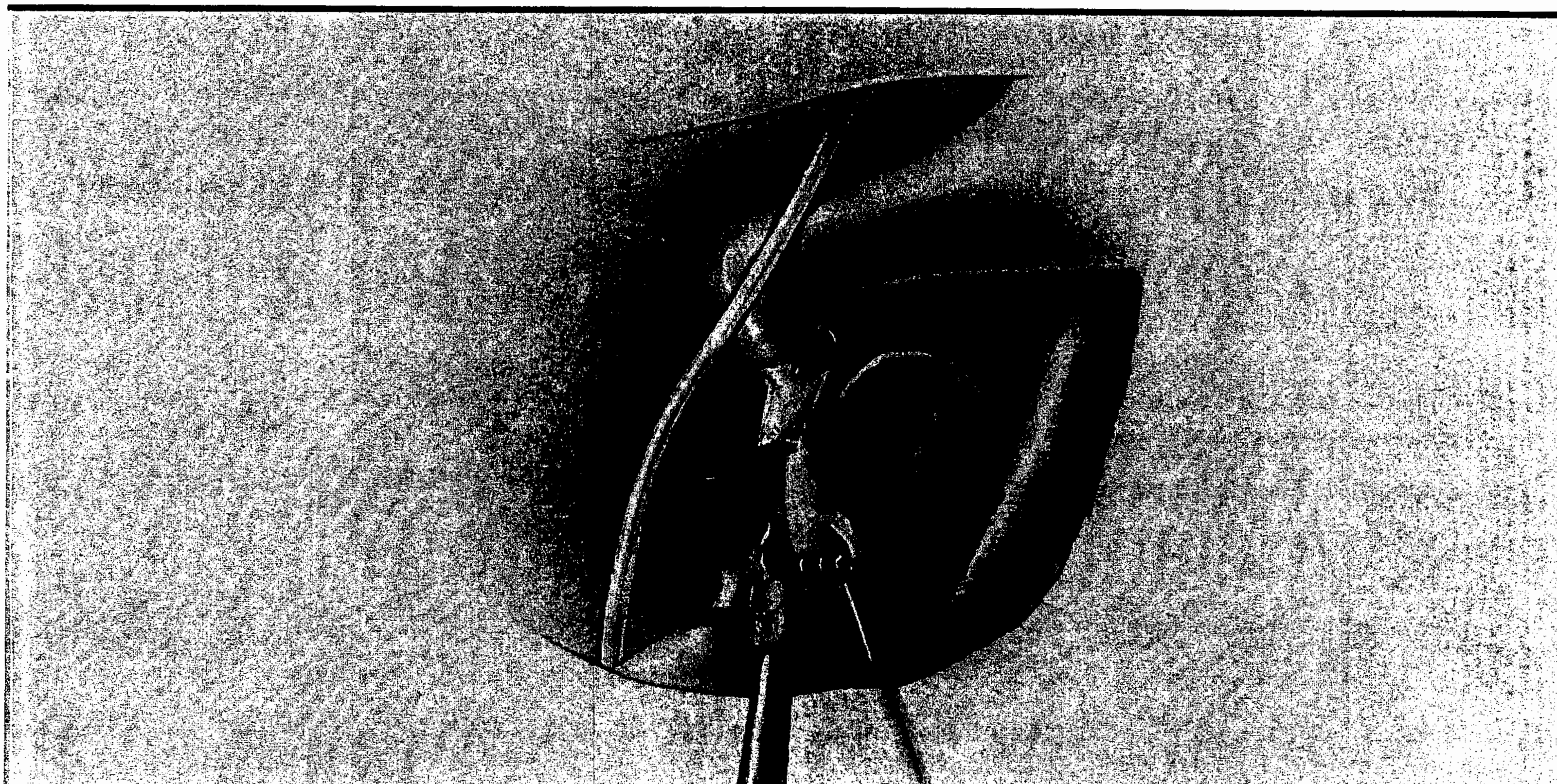

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CHAPTER 21

Benign Paroxysmal Positional Vertigo

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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is a very common entity representing 15 to 40% of patients presenting to a dizziness clinic. BPPV often has a self-limited course, but can be chronic and recurrent, possibly being the most common cause of recurrent vertigo. The pathophysiology involves dislocated otoconia, from the utricle, typically migrating into the posterior semicircular canal causing abnormal stimulation. The classic syndrome is described as short-lived bursts of vertigo brought on by certain positional changes. It is less commonly known that this disorder can also be the cause of chronic, ill-defined disequilibrium. It is also frequently found in association with other inner ear disorders (Ménière's disease, vestibular neuritis, and labyrinthine concussion). Although, this has been traditionally thought of as a "benign" and self-limiting disorder, it can cause significant impairment in the quality of life and can persist for many years. The diagnosis can be made on physical exam with the Dix-Hallpike maneuver. Approximately 80 to 85% of patients can be successfully treated with a single office procedure (canalith repositioning) that results in almost immediate relief of symptoms. The remaining 15 to 20% of patients may require a second repeat procedure, treatment of associated pathology, or surgery. Cases refractory to these simple in-office procedures have been successfully treated with surgical occlusion of the posterior semicircular canal. A newer approach has come with the institution of the Epley Omniax multiaxial repositioning device.

PHYSIOLOGY AND PATHOPHYSIOLOGY

The vestibular system senses angular and linear acceleration by means of the semicircular canals and otolithic organ sensors. The semicircular canals are oriented in 3 perpendicular planes with each canal having an enlarged or ampullated end. The nerve receptor area is found in the ampulla. The cupula, a gelatinous mass, sits atop the sensory cells in the ampulla and acts as an impermeable barrier across the lumen of the semicircular canal. The cupula acts essentially weightless in that it is of the same specific gravity of the surrounding endolymph. Consequently, movement of fluid within the semicircular canal deflects the cupula and causes stimulation or inhibition of the end organ. Deflection of the cupula in the direction of the ampulla (ampullopetal) causes inhibition and away from the ampulla (ampullofugal) causes stimulation of the posterior and superior canals whereas the reverse is true of the horizontal canal. The otolithic organs, which include the saccule and utricle, have a similar gelatinous mass as the cupulae of the semicircular canals but also contain calcium carbonate crystals. These crystals detect gravity and linear acceleration.

The proposed pathophysiology of BPPV may be caused by dislocated otolithic crystals floating in the semicircular canals (canalithiasis) or adhering to one of the semicircular canal cupulae (cupulolithiasis). The cupulolithiasis theory was first proposed by Schuknecht in 1962 with the identification of basophilic particles attached to the posterior canal cupula noted on temporal bone histology specimens of patients with BPPV.[1] He theorized that the particles caused the cupula to be sensitive to the force of gravity resulting in the abnormal stimulation. The canalithiasis theory (free-floating particles within the semicircular canal) was first reported by Hall in 1979.[2] This was later confirmed in vivo by Parnes and McClure in 1992.[3] The free-floating particles typically migrate to the posterior semicircular canal (although migration to the other canals is possible) as it is the most dependent portion of the labyrinth and get trapped in the canal due to the cupula acting as a barrier at the ampullated end of the canal.

These canalith are thought to cause minimal problems until a critical mass is reached. At that point the movement of the mass of canaliths causes a hydrodynamic drag of the endolymph in the posterior semicircular canal. The abnormal stimulation by either abnormal endolymphatic flow or abnormal weighting of the semicircular canal cupulae results in the aberrant stimulation of the vestibular system resulting in the clinical symptoms of BPPV. Whereas cupulolithiasis generally causes immediate stimulation upon placing a patient in the provocative position, canalithiasis requires a short time delay, or latency, before the onset of stimulation. Although pure forms of cupulolithiasis and canalithiasis occur, both can also occur concurrently rendering a more atypical clinical presentation. Similarly, although in the vast majority of cases the posterior semicircular canal is affected, the horizontal semicircular canal has been estimated to be involved in 5 to 30% of the time. Obviously, studies of the prevalence of horizontal BPPV are greatly affected by selection bias of the individual clinics involved. Anterior canal involvement has been reported to be rare, but the author notes this is more common than reported due to selection bias. Anterior canal BPPV is not adequately assessed via the traditional Dix-Hallpike maneuver. A prone, head forward maneuver is more apt to elicit responses of anterior canal involvement, but is simply not performed routinely in clinical practice.

Cupulolithiasis is felt to be the predominate pathophysiology for horizontal canal BPPV and canalithiasis the predominate pathophysiology for posterior canal and anterior canal BPPV. Multiple canal involvement has also been reported and results in atypical findings during exam.

BPPV affecting the posterior canal, as mentioned, is by far the most common variant of BPPV. This is likely due to the dependent nature of the posterior canal, especially while lying supine sleeping at night. Very likely, free-floating crystals migrate into the posterior canal due to the effects of gravity; particularly at night while the upper limb of the posterior canal most easily can be accessed. Once in the posterior canal and the patient arises, the crystals are likely to continue their migration down the posterior canal and become trapped by the cupula at the ampullated end of the canal. At this point the crystals can either migrate further and become attached to the cupula causing cupulolithiasis or once a critical mass of crystals have formed in the posterior canal, canalithiasis may predominate. With canalithiasis, this critical mass of crystals will move cephalad within the canal when placed in the most provocative position (Dix-Hallpike position). However, there is a short time delay before this migration occurs resulting in the latency before symptoms appear. Once movement occurs, there is a hydrodynamic drag, which causes an ampullofugal deflection of the posterior canal cupula, which is excitatory. This results in the symptoms of vertigo and a torsional-type nystagmus in the plane of the posterior canal. The nystagmus would beat clockwise in the left Dix-Hallpike position for a left ear BPPV and counterclockwise in the right Dix-Hallpike position for a right ear BPPV.

A right Dix-Hallpike maneuver should result in no symptoms or nystagmus for a left ear BPPV and the converse is true for a right ear BPPV. The nystagmus ceases once the mass of crystals stops moving and the cupula returns to a neutral position, which is typically 15 to 30 seconds duration. When the patient is brought back to an upright position, the canaliths will move in the opposite direction back down the limb of the posterior canal resulting in an ampullopetal deflection of the cupula. The nystagmus associated with this is in the reverse of the direction associated with the Dix-Hallpike maneuver and is of a lesser magnitude since this is an inhibitory stimulation of the posterior canal. After repeated maneuvers the nystagmus response will fade and is said to be fatigable. This is likely due to the eventual dispersion of the crystals and loss of the critical mass of canaliths.

The horizontal canal is less commonly involved in BPPV likely due to its up-sloping anatomy with the cupula at the upper end of the canal as opposed to the inferior limb like the posterior canal. Hence, canaliths most likely will not enter the horizontal canal and, if they do enter, will migrate out of the horizontal canal during normal daily head movement. Consequently, cupulolithiasis is a more frequently proposed entity with horizontal canal BPPV. In horizontal canal BPPV, a lateral head position is the most provocative position and, unlike the case of posterior canal BPPV, this will cause stimulation for either the right or left ear-down position with just one ear involvement. Cupulolithiasis of the right ear will cause ampullopetal deflection (inhibitory stimulation) in the right-ear-down position and ampullofugal deflection (excitatory stimulation) in the left-ear-down position. This results in an ageotropic (away from ground) horizontal nystagmus, which is stronger in the left-ear-down position (excitatory position). Conversely, canalithiasis of the right ear will cause an opposite pattern. Migration of the canalith mass in the right-ear-down position will cause ampullofugal deflection (excitatory stimulation) and ampullopetal deflection (inhibitory stimulation) in the left-ear-down position. This results in a geotropic (toward ground) horizontal nystagmus, which is stronger in the right-ear-down position (excitatory position). When viewed from the clinician's standpoint, ageotropic horizontal nystagmus is viewed as cupulolithiasis and geotropic horizontal nystagmus is viewed as canalithiasis. The stronger beating nystagmus is noted on the same side as the affected ear in canalithiasis and in the contralateral ear in cupulolithiasis (Table 21-1). One way of remembering which ear is affected in horizontal canal BPPV is that the stronger nystagmus points to the affected ear, regardless of whether it is cupulolithiasis or canalithiasis. So, if the stronger beating nystagmus is going to the left, then the left ear is the pathologic ear.

Table 21-1. Horizontal Canal BPPV Etiology and Location According to Nystagmus Associated with Lateral Ear Position

Strongest Intensity of Nystagmus	Geotropic Nystagmus	Ageotropic Nystagmus
Left	Left Canalithiasis	Right Cupulolithiasis
Right	Right Canalithiasis	Left Cupulolithiasis

ETIOLOGY

BPPV is considered to be either primary or secondary. Primary BPPV is more commonly seen in the elderly population where one study identified unrecognized BPPV in 9% of a large inner city geriatric cohort.[4] Primary BPPV is the most commonly reported type, representing as much as 50 to 70% of BPPV cases. Secondary BPPV can be divided among the differing antecedent conditions and concomitant pathologies. The most commonly recognized of these is head trauma and whiplash injuries. However, many other commonly recognized conditions associated with BPPV have been noted Table 21-2.

It seems much more common to find BPPV among patients who have another pathology of the same inner ear.

DEMOGRAPHICS

BPPV has been reported in virtually all age groups but is more common with increasing age and is especially common among the elderly. There is a gender predilection for afflicting females overall, but the gender distribution appears to be essentially balanced

Table 21-2. Possible Causes of BPPV

Head trauma, whiplash
Viral labyrinthitis, vestibular neuritis
Ménière's disease
Migraine
Ear surgery and nonear surgery
Chronic otitis media
Otosclerosis
Dental manipulation
Acoustic neuroma and other posterior fossa tumors
Viral URI

among younger and post-traumatic patients. There is limited information regarding racial distribution/affinity, but BPPV appears to be common among all ethnic/racial groups.

SYMPTOMS/HISTORY

There are essentially two categories of presentation for BPPV: classic and nonclassic. Additionally, a mixture of these two presentations is not uncommon.

Classic History/Symptoms

In the classic presentation of BPPV, the patient will describe a sudden onset of whirling vertigo spells that are induced by very specific head positions or movements. The most common provocative position is with the offending ear in a more dependent position such as when rolling over in bed to one side. This position places the posterior canal in the vertical position, which will allow for the largest magnitude of cupular deflection. Other provocative positions include looking up ("top shelf syndrome"), bending over, and arising from a supine position. The patients will also note that putting themselves in the provocative position does not always produce vertigo; this is due to fatigability. The severity of the vertigo can vary significantly but is often very severe and violent. The duration of the vertigo is typically less than 30 seconds but may be described by patients as lasting much longer. This is probably due to an overestimation of the time because it is such a noxious stimuli. In patients with classic BPPV, they do not have any symptoms of vertigo or dizziness in between these discrete spells. Occasionally, a patient will report constant vertigo, but often when they are questioned very closely, they relate that whenever they move their head they feel vertiginous, but as long as their head is still they have no symptom.

Nonclassic History/Symptoms

Many patients will complain of other symptoms that have not traditionally been attributed to BPPV and not complain of the classic symptoms. It is felt that the severe classic symptoms of BPPV are often avoided by the patient, either consciously or unconsciously avoiding the provocative position. When questioning patients with nonclassic symptoms they may not report vertigo but you may also find that they never put their head back. By avoiding these certain positions, the severe rotary vertigo is avoided, but the patients will continue to note other symptoms associated with abnormal (but lesser magnitude) stimulation of the posterior semicircular canal. This abnormal stimulation is brought on by linear movements, which should not normally provoke any stimulation of the semicircular canals. The nonclassic symptoms include dizziness associated with:

1. *Arising.* The posterior semicircular canal is stimulated by an abnormal weight (misplaced otoconia) on the cupula that would otherwise not be there. Consequently, the posterior semicircular canal is stimulated when arising. This results in confusing angular input with linear input. These patients can often be confused with patients suffering with postural hypotension. The two main distinctions are normal blood pressure and pulse, and the presence of a spinning sensation. Note that postural hypotension can make a patient feel woozy and lightheaded, but it will not make them have a sensation of spinning.
2. *Leaning forward.* This, too, causes input from an angular accelerometer (posterior semicircular canal) to be confused with linear acceleration (gravity) input. In essence, leaning forward is the inverse of the provocative position (Dix-Hallpike position).
3. *Linear acceleration/deceleration.* Although this motion does not cause maximal stimulation, the effects of gravity and acceleration of the canaliths or cupuloliths will cause mild abnormal stimulation. Instead of the anticipated linear acceleration input, the vestibular organ presents a combination of linear and angular acceleration input.
4. *Vertical oscillation.* Similarly, the effects of gravity on the crystals will cause inappropriate sensation of gravity by the semicircular canal.
5. *Morning dizziness.* Typically, patients will describe mornings to be the worst time of the day for their symptoms. It has been theorized that through-

out the night the canaliths gather into and form a critical mass in the posterior canal while the patient is supine, relatively still and sleeping. Upon arising in the morning, the motions of the canaliths cause the dizziness. With repeated head movements throughout the day, the canaliths disperse and do not cause significant stimulation and hence symptoms lessen.

6. *Head rotation.* Dizziness with head rotation, especially fast head movement, is felt to be secondary to either the inertia of the canaliths or due to their gravitary pull resulting in aberrant stimulation.

PHYSICAL EXAM

The physical examination of the patient with BPPV should be focused on identifying any comorbid ear pathology although frequently the physical exam is unremarkable with the exception of the Dix-Hallpike maneuver. The Dix-Hallpike maneuver is the diagnostic test for posterior canal BPPV. The Dix-Hallpike maneuver is accomplished by placing the patient in a sitting position on the examining table and, with one movement, turning their head 45 degrees to one side and lowering the patient to a supine position. The head is extended 30 degrees below the plane of the examining table if neck mobility allows. It is probably prudent to check for neck mobility and to auscultate the carotid arteries in at risk patients prior to the Dix-Hallpike maneuver. When the head is in the down position, the eyes are examined for nystagmus and the patient s questioned for symptoms of vertigo. Nystagmus and vertigo should coincide.

Examination of the eyes could be done with simple visual inspection but due to overriding visual fixation, lesser degrees of BPPV would be missed. Consequently, a means of eliminating visual fixation is recommended, such as with Frenzel lenses or, even better, infrared video goggles. These methods enhance the sensitivity of the Dix-Hallpike maneuver. Relying on ENG or VNG tracing for evaluation will lead to many missed diagnoses of BPPV as the torsional nature of the nystagmus is not captured on these tracings. Although the Dix-Hallpike maneuver is very specific for the diagnosis of posterior canal BPPV, the sensitivity is somewhat lacking due to the fatigable nature of BPPV. In other words, a negative Dix-Hallpike maneuver does not necessarily mean that the patient does not have BPPV.

Dix and Hallpike describe the classic signs of BPPV[5]:

1. *A critical provocative position.* The episodes of rotary vertigo are often produced when the patient places their head with a particular ear directed downward.
2. *Characteristic nystagmus.* During the vertiginous attack, the eyes of the subject can be seen to produce a very specific nystagmus pattern. For BPPV affecting the right ear, the nystagmus would be a torsional movement with the fast phase in the counterclockwise direction. For BPPV affecting the left ear, the nystagmus is characterized by a torsional movement with the fast phase in the clockwise direction
3. *Latency.* This is typically 2 to 5 seconds in canalithiasis but can be delayed by as much as 30 seconds. Latency may be absent in cupulolithiasis.
4. *Limited duration.* Duration is typically less than 30 seconds in canalithiasis but may be persistent in cupulolithiasis.
5. *Reversal nystagmus.* Reversal of the characteristic nystagmus is frequently noted when bringing the patient back to the upright position after a Dix-Hallpike maneuver. However, this is usually much less in magnitude and is not always discernible.
6. *Fatigability.* Repeat testing results in diminished or absent response.

Evaluation for horizontal canal BPPV is accomplished by having the patient lie supine. While tucking the chin in slightly in order to bring the horizontal canal into a vertical plane (approximately 30 degrees ventrally), the head is quickly turned toward the test side. The eyes are examined for nystagmus and the patient questioned about vertigo. The nystagmus of horizontal canal BPPV has a shorter or absent latency and is horizontal in nature. In unilateral horizontal canal BPPV, nystagmus will typically be present with testing on either side. As detailed in Table 21–1, the direction and magnitude of the nystagmus can elucidate the mechanism of BPPV and the affected ear.

EVALUATION

The Dix-Hallpike test is the gold standard for diagnosis of posterior canal BPPV. Virtually no other pathology will mimic the characteristic nystagmus and clinical symptoms of posterior canal BPPV. Beyond the Dix-Hallpike and the positional testing for horizontal canal involvement, there are no required tests for diagnosing BPPV. However, concomitant pathology of the inner ear or skull base (such as cerebellopontine angle masses or Ménière's disease) may dictate further evaluation.

The indications for these additional studies would be based on clinical findings that do not fit under the classic presentation of BPPV. Most ancillary vestibular tests do not demonstrate any abnormality with straightforward BPPV (with the exception of the Dix-Hallpike test and positional testing). However, caloric testing, vestibular autorotation testing and posturography may show abnormalities with routine BPPV cases. Some have reported abnormal caloric results in horizontal canal BPPV speculating that the canaliths interfered with normal thermally induced endolymphatic flow or cupular deflection during caloric testing. Similarly, vertical VOR testing in vestibular autorotation testing has been shown to result in phase abnormalities in BPPV. This is likely due to a similar mechanism but due to involvement of the posterior canal. Computerized dynamic posturography, although typically normal in BPPV cases, has been shown to produce reduced stability in SOT conditions 5 and 6, the classic vestibular deficit pattern, particularly in the elderly. This has been noted to improve after successful treatment of BPPV.

DIFFERENTIAL DIAGNOSIS

Very few disorders can replicate the findings of BPPV, but there are several that can produce positional nystagmus and dizziness. Although these disorders can produce positional vertigo and nystagmus, they do not typically produce the characteristic nystagmus found with BPPV. Some of these include posterior fossa masses, craniovertebral junction abnormalities, cervical vertigo, vascular loop compression syndrome, alcoholic intoxication, and vertebrobasilar vascular insufficiency. BPPV has been implicated as a secondary pathology to a number of entities including those listed here. However, BPPV is so prevalent that coincidental occurrence of BPPV with other disorders can also be expected.

TREATMENT

Traditionally, the treatment of BPPV has been benign neglect since most cases are suspected to resolve spontaneously. Consequently a period of deferred treatment seems reasonable if the symptoms have recently occurred. Medical treatment for BPPV has been largely ineffectual, with limited response to vestibular suppressants and antiemetics. Canalith repositioning maneuvers (CRM) have been the mainstay of treatment for BPPV. These have included the Epley and Semont maneuvers for posterior canal BPPV. For horizontal canal BPPV, forced prolonged positioning and barrel roll maneuvers typically have been favored. CRM are based on the idea of moving the canalith through the offending semicircular canal and back into the area of the utricle in order to eliminate the aberrant stimulus.

Although it wasn't until 1992 that Epley[6] first published his report of the canalith repositioning procedure (CRP), he had been performing and teaching CRP in his private practice since 1979. Prior attempts to publish his findings met with rejection due to skepticism that such a simple maneuver would alleviate vertigo. In fact, skepticism ran so high that early in his career, complaints by fellow physicians were brought to the hospital committee in order prevent him from using CRP (J. Epley, personal communication, July 2008). The "Epley maneuver," or a variation thereof, appears to be the most popular maneuver by clinicians in treating posterior canal BPPV. The maneuver (Figure 21–1) based on the canalithiasis theory, is accomplished by: (1) Starting in a sitting position, (2) Placing the patient in the Dix-Hallpike position (of the offending ear) while observing for nystagmus, (3) Rotate the head 90 degrees to the contralateral Dix-Hallpike position, (4) Have the patient roll into a lateral decubitus position with the head 90 degrees further (with

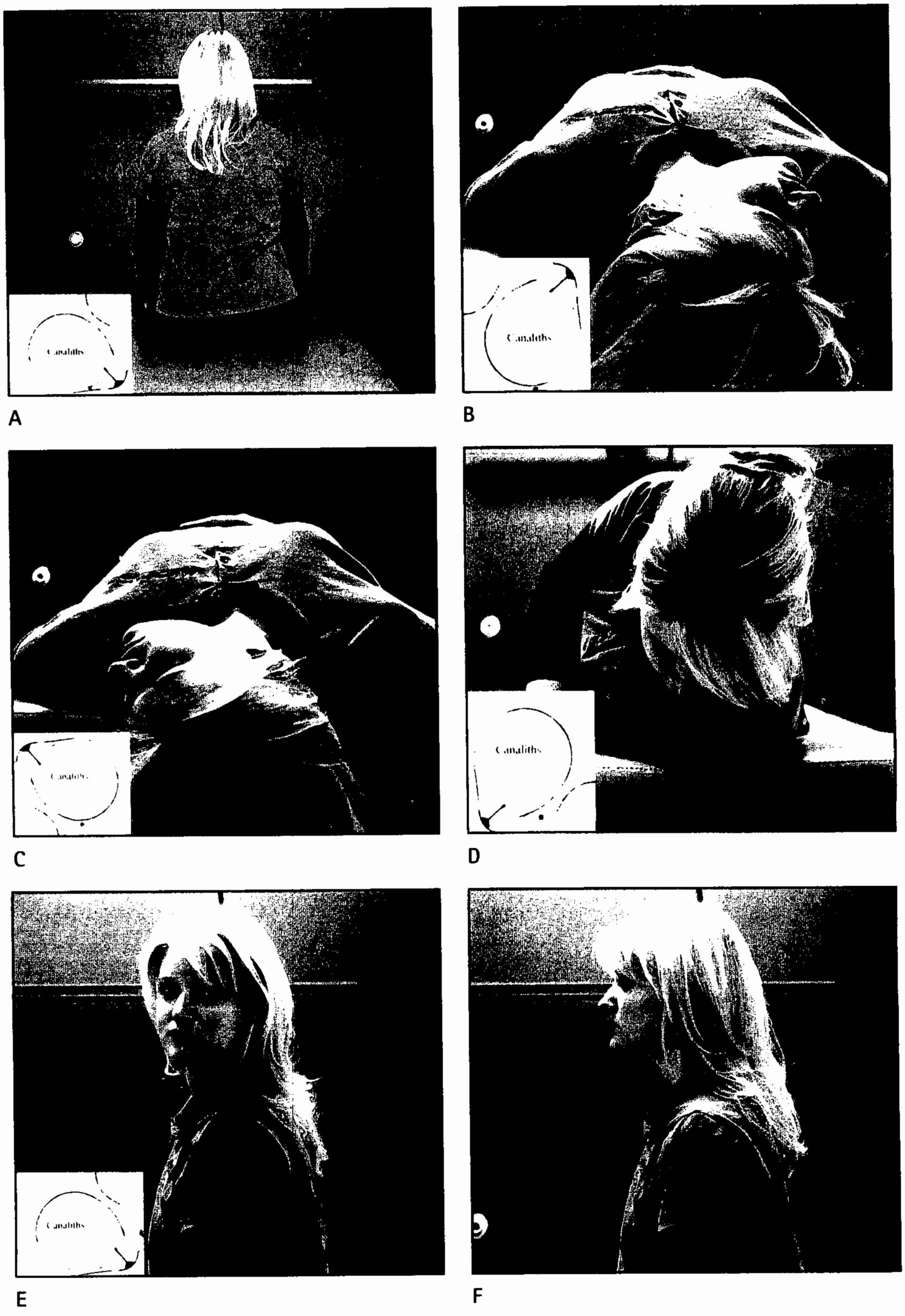


Figure 21–1. The canalith-repositioning maneuver (for right ear posterior canal BPPV) with corresponding posterior semicircular canal position and canalith position. **A.** Starting in a sitting position. **B.** Placing the patient in the Dix-Hallpike position (of the offending ear) while observing for nystagmus. **C.** Rotate the head 90 degrees to the contralateral Dix-Hallpike position. **D.** Have the patient roll into a lateral decubitus position with the head 90 degrees further (with the plane of the nose pointing 45 degrees below the horizon). **E.** Bring the patient to an upright sitting position with their head still turned away. **F.** While sitting turn the head back to midline.

the plane of the nose pointing 45 degrees below the horizon), (5) Bring the patient to an upright sitting position with their head still turned away, and (6) While sitting turn the head back to midline.

The "Semont maneuver" or liberatory maneuver was first described in 1988 and is based on the cupulolithiasis theory.[7] The patient is seated on the exam table with the head turned away from the affected side. Quickly, the patient is brought into a side-lying position on the affected side with the head turned upward. After 5 minutes, the patient is then rapidly brought to the contralateral side-lying position with the head facing downwards. After another 5 minutes, the patient is then brought back up to the sitting position. The maneuver is based on the idea that the rapid change in position would free the canaliths attached to the cupula and allow them to migrate out of the posterior canal. Although demonstrated to be an effective maneuver, the rapid changes in body position are difficult for some patients (especially the elderly and those with mobility problems) compared to the Epley maneuver.

A simple treatment of horizontal canal BPPV is forced prolonged positioning.[8] In this technique the patient is instructed to lie with the affected ear in the uppermost position for 12 hours, or nightly for several nights in a row. This has been reported to be effective for both canalithiasis and cupulolithiasis, but appears to be more effective with the former. Another treatment option is a barrel roll or barbeque roll maneuver. In the maneuver, the patient starts off supine with the head turned toward the affected ear. The patient is then rolled 360 degrees, in 90-degree increments, while keeping the horizontal canal perpendicular to the ground.

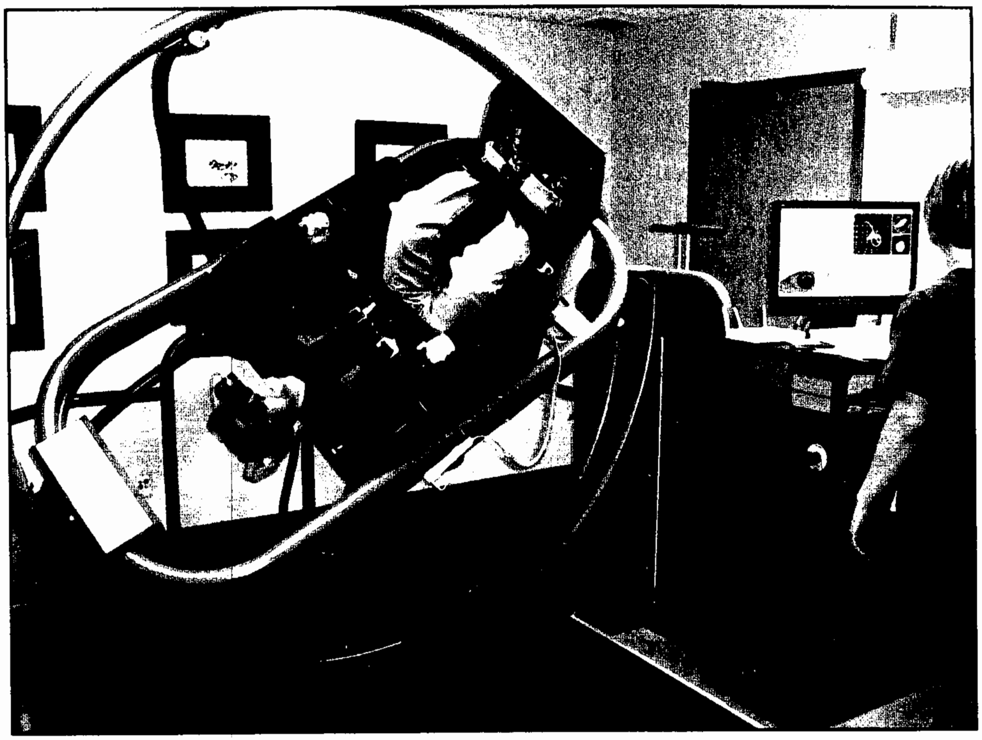
The use of mastoid vibration during CRM has been controversial. Adding mastoid vibration to CRM was first reported by Li[9] to significantly improve successful outcome, but additional studies have not replicated these findings. It may be that mastoid vibration makes the most impact in cases of cupulolithiasis. After CRM, many clinicians restrict the patient's head position for 24 to 72 hours postmaneuver. Studies evaluating postmaneuver restrictions have been mixed with most demonstrating no difference in treatment and control groups.

Surgery for BPPV is limited to only the rare cases that have frequent and multiply recurrent BPPV after CRM. The first surgical procedure directed to treatment of posterior canal BPPV was singular neurectomy (posterior ampullary nerve section), which was popularized by Gacek in the 1970s.[10] Although effective, the technically demanding nature and the high incidence of sensorineural hearing loss significantly reduced its popularity. Posterior canal occlusion was introduced by Parnes and McClure in 1990.[11] This procedure, done through a mastoidectomy approach, results in occlusion or blockage of endolymphatic movement in the posterior semicircular canal and effectively locks the cupula in place and eliminates posterior canal stimulation. Effective and technically easier than singular neurectomy, this has become the most common surgical treatment for BPPV refractory to CRM.

COMPLEX AND MIXED CANAL BPPV

Since the introduction of CRM, many clinicians have noted the presence of multiple canal involvement BPPV—mixed horizontal/posterior, bilateral BPPV, and anterior canal BPPV. In one study the incidence of multiple canal involvement was 20% and bilateral BPPV at over 10%. The nystagmus generated by multiple canal involvement can significantly complicate the diagnostic and treatment paradigm. The most prudent approach has been to treat the most suspicious ear or the posterior canal component first and then evaluate the patient 2 to 3 weeks later. At that point the clinical picture is often much clearer.

The Epley Omniax multiaxial positioning device (Figure 21–2) appears to be a very effective and useful alternative for both the diagnosis and treatment of complex/atypical BPPV.[12] Developed by Vesticon, the Omniax received FDA approval in 2008. With the Omniax, the patient is seated and harnessed in a chair that allows for 360-degree rotation in any direction while monitoring eye movement with infrared videography. The chair is controlled by the accompanying computer that records video, eye tracings and position. Although many standard maneuvers are programmed into the Omniax (eg, Epley, Barrel roll, Dix-Hallpike, etc), many less commonly used maneuvers and a free-style component are included.



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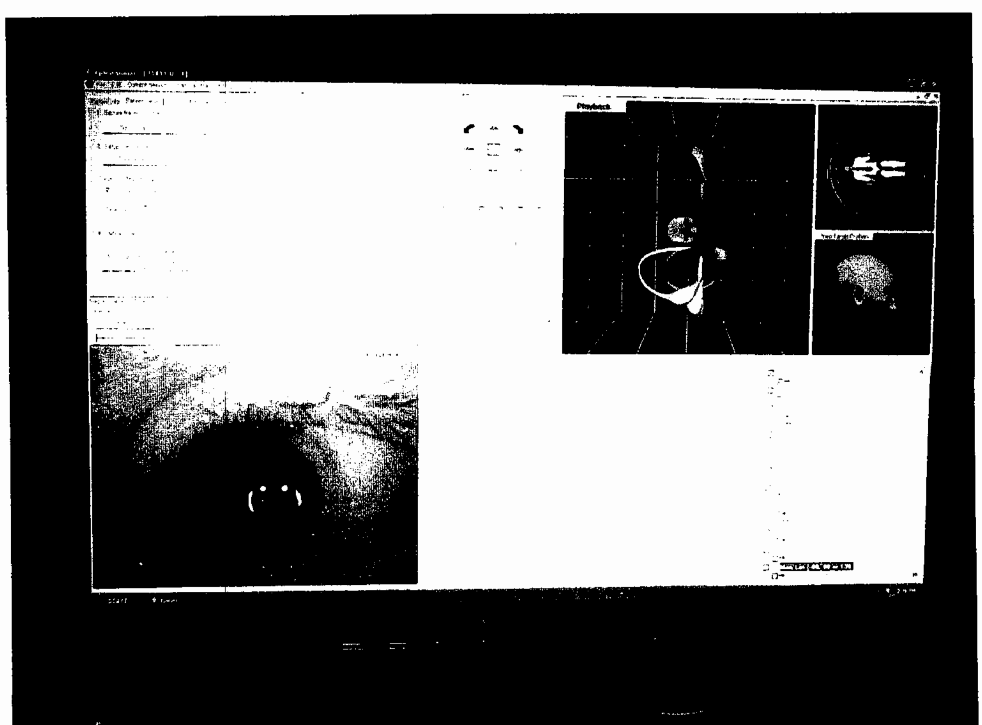

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Figure 21–2. Epley Omniax device. **A.** Epley Omniax multiaxial positioning device. **B.** Screenshot of Epley Omniax computer control module.

The Omniax has been shown effective in the treatment of routine case of BPPV but in the author's opinion, it is most useful in the tertiary care diagnosis and treatment of complicated BPPV.

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