

Benign Paroxysmal Positional Vertigo: Diagnosis and Treatment in the Emergency Department—A Review of the Literature and Discussion of Canalith-Repositioning Maneuvers

From the Department of Emergency Medicine, University of Mississippi Medical Center, Jackson, MS,* and the University of Tennessee College of Medicine, Memphis, TN.†

Received for publication March 16, 2000. Revision received September 11, 2000. Accepted for publication October 2, 2000.

Address for reprints: Paul Koelliker, MD, 466 Arundel Drive, Brandon, MS 39047; 601-992-7673, fax 601-984-5583; E-mail pkoelliker@aol.com.

Copyright © 2001 by the American College of Emergency Physicians.

0196-0644/2001/\$35.00 + 0

47/1/112250

doi:10.1067/mem.2001.112250

Paul Koelliker, MD*
Richard L. Summers, MD*
Brian Hawkins, BS†

Dizziness is a frequent presenting complaint in emergency department patients. Although seen in patients of all ages, it is more prevalent in patients older than 50 years of age. Vertigo represents a subset of dizziness and is defined as an illusion of movement, usually rotational, of the patient or the patient's surroundings. The illusion of motion may be of oneself (subjective vertigo) or of external objects (objective vertigo). The emergency physician should consider a large differential in the evaluation of vertigo with special attention to whether the vertigo is central or peripheral in origin.

[Koelliker P, Summers RL, Hawkins B. Benign paroxysmal positional vertigo: diagnosis and treatment in the emergency department—a review of the literature and discussion of canalith-repositioning maneuvers. *Ann Emerg Med.* April 2001;37:392-398.]

INTRODUCTION

Dizziness is a frequent presenting complaint in emergency department patients. In a study of 1,000 outpatients, dizziness was the third most common complaint.¹ Although seen in patients of all ages, it is more prevalent in patients older than 50 years of age.² Vertigo represents a subset of dizziness and is defined as an illusion of movement, usually rotational, of the patient or the patient's surroundings.³ The illusion of motion may be of oneself (subjective vertigo) or of external objects (objective vertigo).⁴ A large number of entities cause vertigo, ranging from benign and self-limited causes such as vestibular neuritis and benign paroxysmal positional vertigo (BPPV), to immediately life-threatening causes such as cerebellar infarction or hemorrhage.⁵

The emergency physician should consider a large differential in the evaluation of vertigo with special attention to whether the vertigo is central or peripheral in origin.³ The current textbooks in emergency medicine^{6,7} mention diagnostic maneuvers for BPPV, but do not illus-

trate the therapeutic maneuvers found in the neurology and otorhinolaryngology literature. BPPV frequently occurs, can be reliably diagnosed, and can often be treated with a high success rate by using a repositioning or liberatory maneuver in the outpatient setting. These maneuvers can be performed quickly at the bedside with rapid results, often providing much satisfaction to both patient and physician.

Adler⁸ first described BPPV in 1897 and Bárány⁹ in 1921. The condition has often been referred to as Bárány vertigo, and the positioning used to elicit the vertigo the Bárány test or the Bárány-Nylen test. Dix and Hallpike¹⁰ first described this test in 1952, and they coined the term "benign paroxysmal positional vertigo." It is now believed that the provocative test should be called the Hallpike-Dix test and not the Bárány-Nylen test. In 1973, Schuknecht and Ruby¹¹ hypothesized that heavy debris that had settled on the cupula transformed the cupula from an organ of angular acceleration into one of linear acceleration, which caused the symptoms of BPPV. Since the work of Parnes and McClure¹² in 1992, it has become generally accepted that free-floating debris in the posterior semicircular canal causes the symptoms of BPPV. Semont et al¹³ first described a liberatory maneuver in 1988, and another was described by Epley¹⁴ in 1992. Brandt¹⁵ has suggested that the condition be called benign paroxysmal positioning vertigo because it is the act of positioning the patient, not the position of the patient, that elicits the symptoms.

BPPV is a commonly encountered problem with an incidence reported to be 64 per 100,000 population per year.¹⁶ As many as 20% of patients presenting with dizziness may have BPPV,¹⁷ and it accounts for a fifth of referrals to clinics that specialize in dizziness.¹⁸ Female patients outnumber males 1.6 to 1, and it is most common in the sixth decade, but episodes may occur in childhood. Many cases occur after vestibular neuritis or head trauma (which may be minor), whereas other cases are idiopathic. Many cases of BPPV associated with vestibular neuritis occur after a viral upper respiratory tract infection. BPPV is also common in patients with Ménière's disease.¹⁹ Posterior canal BPPV accounts for the large majority of cases, but 5% to 10% may have BPPV of the horizontal canal in which the vertigo is elicited by different positional changes.²⁰

ANATOMY AND PATHOPHYSIOLOGY

The functioning portion of the peripheral vestibular system is located in the membranous labyrinth of the ear. It

consists of 3 semicircular canals, a utricle, and a saccule. The cristae ampullaris in the semicircular canals detect angular acceleration, and the maculae of the utricle and saccule detect linear acceleration and changes in position of the head with respect to gravity. The maculae are the sensory organs in the utricle and saccule. In the maculae are complexes consisting of a gelatinous layer in which calcium carbonate crystals or otoliths are embedded. Hair cells project cilia into this complex, which are bent when the complex moves. The bending of the hair cells causes changes in the rate of impulses sent to the central nervous system (CNS). It is these otoliths that can become displaced into the endolymph of the semicircular canals and cause the symptoms of BPPV. The otoliths are often dislodged after head trauma, after a viral illness, in patients with vertebral basilar insufficiency, or in patients with Ménière's disease.^{19,21} Clotted blood in the posterior canal has also been implicated as a cause.¹⁵

The 3 semicircular canals are arranged in right angles to represent all 3 planes in space. They are filled with a viscous fluid called endolymph. At one end of each of the canals is an enlarged portion called the ampulla. The ampulla houses the crista ampullaris, which is a collection of hair cells, covered with a gelatinous mass called the cupula. The semicircular canals move with the head, and the viscous endolymph remains relatively stationary. This creates a deflection of the cupula opposite to head motion, which bends hair cells and sends signals to the CNS.²²

Inappropriate particles in the endolymph of the semicircular canals move as a result of gravitation forces when head position changes. This movement of the particles causes the endolymph to deflect the cupula and stimulate the hair cells. The CNS interprets these signals as angular acceleration of the head when none exists. This faulty stimulation causes the sensation of vertigo in BPPV. Signals from the CNS cause the eyes to deviate in the opposite direction of the perceived acceleration, which causes the slow component of the nystagmus associated with BPPV. The fast component of the nystagmus originates in the frontal lobe as a corrective action.²¹ It is the fast phase by convention that determines the direction of the nystagmus.¹⁷

In the past, BPPV has been speculated to have arisen from cupulolithiasis or debris adherent to the membrane of the cupula. The cupula was suspected to become "heavy" relative to the endolymph and respond to changes in position relative to gravity. It is now thought that classic symptoms of BPPV are unlikely to be caused by this theory, and it is generally believed that free-floating debris is responsi-

ble. As in canalithiasis, a “heavy cupula” should cause nystagmus brought on by a particular head position. This nystagmus, however, should not have a latency period, should not fatigue with repeat positioning, and should be sustained for long periods of time. Some maintain that this variant of BPPV exists and accounts for BPPV with atypical nystagmus or that is resistant to treatment.^{14,15}

BPPV of the posterior semicircular canal

BPPV of the posterior semicircular canal classically is accompanied by a history of brief attacks of severe rotational vertigo precipitated by changes in position. The patient may complain of vertigo when turning over in bed, arising from a supine position, or extending the head backward to look upward.³ Placing objects on a tall shelf or bending over to tie shoes may precipitate attacks of vertigo.²³ Between attacks of vertigo, patients may complain of nonspecific dizziness, nausea, or a feeling of imbalance.¹⁹ Patients with long-standing BPPV may learn to avoid provocative positions and complain only of disequilibrium.²³

Dix and Hallpike¹⁰ first described characteristic symptoms and a provocative test. Typical nystagmus is elicited in a position with the affected ear down. The nystagmus is predominantly torsional with the fast phase toward the undermost ear. The inappropriately stimulated canal drives the eyes toward the opposite side, and in response the frontal lobe sends corrective signals to the eyes, causing them to quickly return to their original position. There is a latency period, after the patient has been positioned, before the nystagmus begins of 5 to 15 seconds. The nystagmus is transient, usually lasting only 20 seconds, but may last up to a minute (extinguishes spontaneously). The nystagmus reverses when the head is returned to the upright position. This reverse nystagmus is usually less severe than that experienced in the head-down position. The severity of the nystagmus temporarily fatigues with repetition of the provocative test. If nystagmus is not elicited with the Hallpike-Dix test, the test should be repeated with the opposite ear down. The side of the ear in the downward position during the Hallpike-Dix test that elicits the greater nystagmus is the affected ear. In some cases, nystagmus and vertigo only occur with the affected ear down.^{24,25}

To perform the Hallpike-Dix test, the patient is seated on a stretcher in a manner to allow the patient to hang the head off of the bed when placed in the supine position. The examiner holds the sides of the patient's head and turns the head to the side of the ear being tested 45 degrees. The patient is then lowered to the supine position with

the head turned, and the head is angled backward off the end of the bed 45 degrees. The examiner should ask the patient to keep the eyes open and fixated on the examiner's face, so the examiner may appreciate the character and severity of the nystagmus. It is necessary to explain to the patient that the test may cause vertigo and nausea and encourage him or her to keep the eyes open.²⁶ A study of 95 patients examined with symptoms of BPPV showed that the presence of paroxysmal positional nystagmus is the most reliable finding in patients with this disorder.²⁷

Traditional treatment for BPPV has been antiemetics and referral to an otorhinolaryngologist for vestibular habituation training (Brandt-Daroff exercises).²⁸ Once diagnosed, BPPV can be easily treated with a canalith-repositioning procedure (CRP). Two CRPs can easily be performed at the bedside in 5 to 10 minutes.

The Epley maneuver. With the patient seated, the patient's head is turned 45 degrees toward the affected ear. The canalith is in the base or most distal portion of the canal at this point. The patient is then tilted backward to a head-hanging position with the head kept in the 45-degree rotation. An attack of vertigo is induced by the canalith moving toward the apex of the canal. The patient is held in this position (same as the Hallpike-Dix position) until the nystagmus and vertigo abate (some authors recommend 4 minutes in each position).²⁸ The head is then turned 90 degrees toward the unaffected ear. With the head remaining turned, the patient is rolled onto the side of the unaffected ear (patient is now looking at the floor). This causes the canalith to move toward the common crus of the 3 semicircular canals. This may again provoke a paroxysm of nystagmus and vertigo. The patient should again remain in this position for 3 minutes. The patient is then moved to the seated position. Finally, the head is tilted down 30 degrees, allowing the canalith to fall into the utricle (Figure 1).^{14,15} It has been reported that patients with nystagmus in the final CRP position have better results.²⁹ If patients have severe nausea and vomiting during the procedure, then they may need to be premedicated with an antiemetic before undergoing a CRP.³⁰

The Semont maneuver. With the patient seated, the head is turned 45 degrees horizontally toward the unaffected ear. In this position, the canalith is in the base of the posterior canal. The patient is tilted 105 degrees toward the affected ear (patient lying on side with head hanging and nose pointed upward). The patient should experience a paroxysm of nystagmus beating toward the undermost ear. This is caused by the canalith moving to the apex of the canal, which is now in a dependent position. The patient is left in this position for 3 minutes. The patient is

then quickly moved through the seated position to lie on the side of the affected ear (nose pointed toward the ground). The patient may again experience vertigo and nystagmus beating toward the uppermost ear as the canalith moves toward the exit of the canal. The patient is then slowly moved to the seated position, allowing the canalith to fall down the common crus into the utricle (Figure 2).^{15,31}

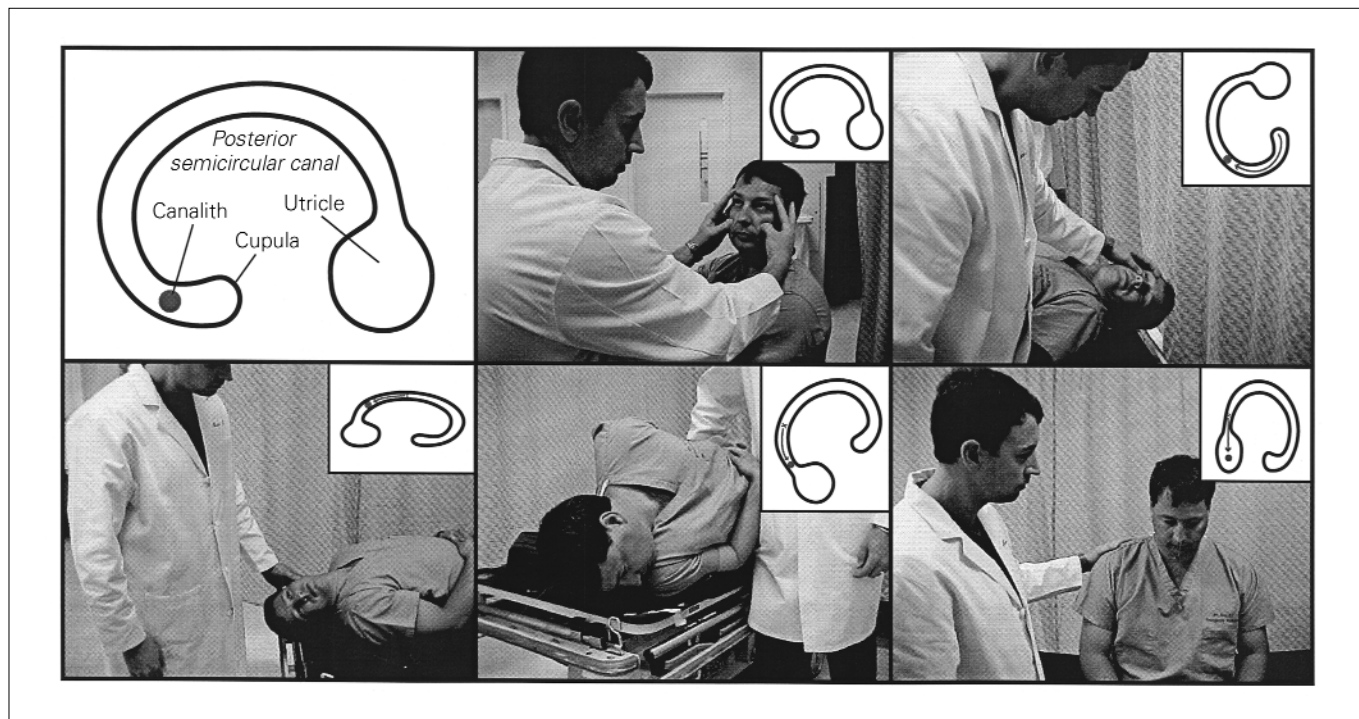
Contraindications to these maneuvers include severe disease of the cervical spine, unstable cardiac disease, and high-grade carotid stenosis.²³ Handheld vibrators are sometimes used to aid the movement of the particles, but this is contraindicated in patients with retinal detachment and perilymphatic fistulas. Conversions of transient nystagmus to a rapid form of persistent nystagmus that occurs irrespective of head position has been described. This phenomenon is attributed to a “canalith jam” by Epley.²⁴ All cases of “canalith jam” were successfully treated with

further repositioning with the aid of a low-frequency handheld vibrator. In 400 patients treated with the Epley maneuver, no significant long-term complications were encountered. Another possible complication is the conversion of posterior canal BPPV to anterior or horizontal canal BPPV after a CRP.¹³

Most authors recommend that the patient remain upright for 1 to 2 days and avoid bending over after a successful CRP.^{13,23} Even with a successful CRP, the patient may continue to feel off-balance for a few days and should not drive home from the ED.²⁸ Routine use of vestibular suppressants (meclizine and benzodiazepines) as primary therapy is discouraged because they do not reduce the frequency of attacks of vertigo. They may diminish the intensity of the attacks and prevent the patient from seeking treatment for a potentially curable disease, and they often worsen the patient’s imbalance. Short-term use on an as-needed basis in patients with severe symptoms

Figure 1.

*Epley maneuver. **Top** (left to right), The first window is a legend for the inset, which is a simplified representation of a posterior semicircular canal. This figure shows the Epley canalith-repositioning maneuver for the left semicircular canal. The patient’s head is turned 45 degrees toward the affected ear, with the patient holding the physician’s arm for support. The patient is then lowered to a supine position. The patient’s head remains turned 45 degrees and should hang off the end of the bed. **Bottom**, In the third position, the patient’s head is rotated to face the opposite shoulder. Next, the patient is rolled onto his or her side, taking care to keep the head rotated. The patient is now returned to a seated position with the head tilted forward.*



may be appropriate provided adequate follow-up with an otorhinolaryngologist or neurologist is obtained.²¹

These maneuvers have reported success rates for decreasing or alleviating symptoms of 66% to 100% in different reports.^{14,31-36} The maneuvers should be repeated several times if the symptoms do not resolve in the first attempt. Vertigo that does not respond to one maneuver may respond to the other. Thirty percent to 50% of patients experience a recurrence. The recurrences are usually easily treated with repeat maneuvers.^{14,15} Patients who do not respond to CRP may respond to vestibular habituation therapy (Brandt-Daroff exercises).³⁷ These exercises have similar success rates to CRPs but take much longer for results. They consist of a set of positions in which the patients place themselves to elicit, and eventually habituate them to, the vertigo. A significantly higher proportion of patients (75%) in an untreated control group continued to have vertigo compared with patients treated with either CRP or vestibular habituation.³⁶

BPPV of the horizontal canal

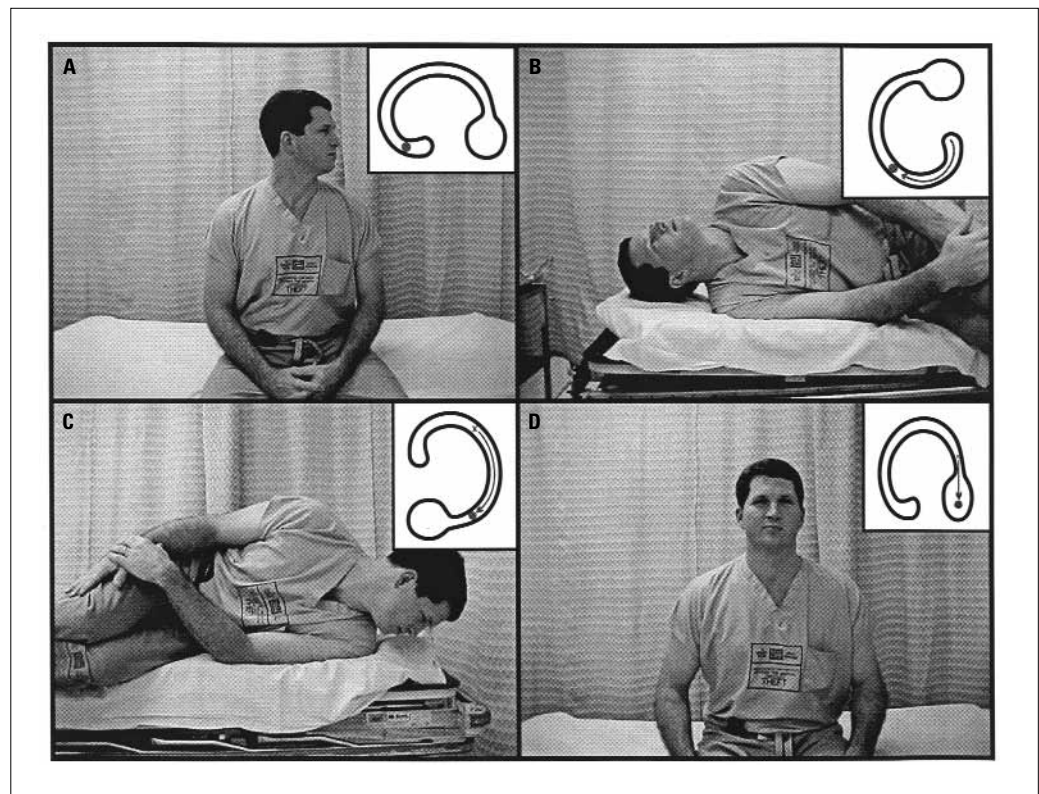
A variant of BPPV exists where debris in the horizontal canal stimulates positional vertigo and has been reported

to account for 5% to 22% of cases of BPPV.^{20,38} The symptoms of vertigo mainly occur when the patient is supine and rolls over in bed.³⁹ The nystagmus and vertigo both occur without latency and with head motion to either side, but is more intense with movements toward the affected ear.⁴⁰ Unlike BPPV of the posterior canal, episodes rarely occur with getting up from or lying down in bed. The intensity of the vertigo and nystagmus is worse with rapid turning of the head and does not fatigue with repeat movements. Results of neurologic examination should be normal, but many patients have imaging studies to rule out central causes because of the atypical features of this variant of BPPV.⁴¹ Trials of repositioning maneuvers have been described with variable success.³⁸⁻⁴² There are currently no large trials to support the efficacy of these maneuvers.

In summary, patients with vertigo can be a dilemma for the emergency physician. There are multitudes of causes of vertigo, including several medical emergencies such as cerebellar stroke or posterior fossa hemorrhage. Care should be taken in eliciting a history of stroke risk factors, and performing a thorough neurologic examination with emphasis on tests of cerebellar function. Vertigo in patients

Figure 2.

Semont maneuver. This figure shows the Semont canalith-repositioning maneuver for BPPV of the right posterior semicircular canal. A, Initially, the patient's head is rotated to face the left shoulder (contralateral to the affected ear). B, While holding the head in this position, the patient is lowered onto his or her right side. C, In the next position, the patient is quickly moved through the seated position and lowered to rest onto his or her left side (the head should remain rotated toward the left shoulder throughout this movement). D, Finally, the patient is returned to a seated position.



with neurologic findings on examination should not be attributed to benign causes without a comprehensive evaluation to rule out central causes for their symptoms.

BPPV is not uncommon, and most emergency physicians see several cases per year. Although it is not a life-threatening condition, BPPV causes debilitating nausea and vertigo, and patients are usually quite distressed by their symptoms. The condition can be reliably diagnosed in the majority of cases using the test and symptom complex described by Dix and Hallpike.¹⁰ Most patients experience relief from their symptoms with the liberatory maneuvers. Up to half of these patients have a recurrence of their symptoms that usually responds to repeat repositioning. The liberatory maneuvers require no special equipment and can be performed both quickly and easily at the bedside. Patients often have instantaneous relief from their symptoms, providing satisfaction to both patient and physician. Patients without all of the classic symptoms of BPPV may respond to the repositioning maneuvers. If a patient has complete resolution of symptoms with repositioning, other causes for the vertigo become less likely.

A working knowledge of BPPV and its treatment is important to an emergency physician even if the repositioning maneuvers are not attempted or are unsuccessful in the ED. An accurate diagnosis and prompt referral to an otorhinolaryngologist for treatment will provide the patient with reassurance and prevent the prolongation of the condition by the use of vestibular suppressants alone.

This condition and its treatment have been discussed in the otorhinolaryngology, neurology, and occasionally primary care literature in the past. Patients with BPPV often initially present to the ED, and with a good working knowledge the emergency physician can reliably diagnose and effectively treat this disorder.

REFERENCES

- Gizzi M, Rosenburg M. The diagnostic approach to the dizzy patient. *Neurologist*. 1998;4:138-147.
- Kroenke K, Mangelsdorff AD. Common symptoms in ambulatory care: incidence, evaluation, therapy, and outcome. *Am J Med*. 1989;86:262-266.
- Baloh R. Vertigo. *Lancet*. 1998;352:1841-1846.
- Disher M, Telian S, Kemink J. Evaluation of acute vertigo: unusual lesions imitating vestibular neuritis. *Am J Otol*. 1991;12:227-231.
- Hotsjon J, Baloh R. Acute vestibular syndrome. *N Engl J Med*. 1998;339:680-685.
- Olshaker JS. Vertigo. In: Rosen P, Barkin R, eds. *Emergency Medicine: Concepts and Clinical Practice*. 4th ed. St. Louis, MO: Mosby; 1998:2165-2173.
- Goldman B. Vertigo and dizziness. In: Tintinalli J, Kelen GD, Stapczynski JS, eds. *Emergency Medicine: A Comprehensive Study Guide*. 5th ed. New York, NY: McGraw-Hill; 2000:1452-1462.
- Adler D. Über den "einseitigen Drehschwindel." *Dtsch Z Nervenheilk*. 1897;358-375.
- Bárány R. Diagnose von Krankheitserscheinungen im Bereiche des Otolithenapparates. *Acta Otolaryngol (Stockh)*. 1921;2:334-437.
- Dix R, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Ann Otol Rhinol Laryngol*. 1952;6:987-1016.
- Schuknect HF, Ruby RRF. Cupulolithiasis. *Adv Otorhinolaryngol*. 1973;22:434-443.
- Parnes L, McClure J. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. *Laryngoscope*. 1992;102:988-992.
- Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. *Adv Otorhinolaryngol*. 1988;42:290-293.
- Epley J. The canalith repositioning procedure: for the treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1992;107:399-404.
- Brandt T. Benign paroxysmal positioning vertigo. *Adv Otorhinolaryngol*. 1999;55:169-194.
- Froehling D, Silverstein M, Mohn DN, et al. Benign positional vertigo: incidence and prognosis in a population-based study in Olmsted County, Minnesota. *Mayo Clin Proc*. 1991;66:596-601.
- Bernard M, Bachenburg T, Brey R. Benign positional vertigo: the canalith repositioning procedure. *Am Fam Physician*. 1996;53:2613-2616.
- Nezelski JM, Barber HO, McIlmoyle L. Diagnoses in a dizziness unit. *J Otolaryngol*. 1986;15:101-104.
- Baloh R, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology*. 1987;37:371-378.
- McClure JA. Horizontal canal BPV. *J Otolaryngol*. 1985;14:30-35.
- Brandt T, Steddin S. Current view of the mechanism of benign paroxysmal positioning vertigo: cupulolithiasis or canalolithiasis? *J Vestib Res*. 1993;3:373-382.
- Guyton A. *Basic Neuroscience: Anatomy and Physiology*. 2nd ed. Philadelphia, PA: WB Saunders; 1992:216-223.
- Furman J, Cass S. Benign paroxysmal positional vertigo. *N Engl J Med*. 1999;341:1590-1596.
- Epley J. Positional vertigo related to semicircular canalolithiasis. *Otolaryngol Head Neck Surg*. 1995;112:154-161.
- Epley J. New dimensions of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1980;88:599-605.
- Gordon N. Benign paroxysmal positional vertigo. *Br J Clin Pract*. 1996;50:208-210.
- Norré M. Reliability of examination data in the diagnosis of benign paroxysmal positional vertigo. *Am J Otol*. 1995;16:806-810.
- Herdman S, Tusa R, Zee DS, et al. Single treatment approaches of benign paroxysmal positional vertigo. *Arch Otolaryngol Head Neck Surg*. 1993;119:450-454.
- Katsarkas A. Paroxysmal positional vertigo: an overview and the deposits repositioning maneuver. *Am J Otol*. 1995;16:725-730.
- Lempert T, Gresty M, Bronstein A. Benign positional vertigo: recognition and treatment. *BMJ*. 1995;311:489-491.
- Serafini G, Palmieri A, Simoncelli C. Benign paroxysmal vertigo of posterior semicircular canal: results in 160 cases treated with Semont's maneuver. *Ann Otol Rhinol Laryngol*. 1996;105:770-775.
- Welling D, Barnes D. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Laryngoscope*. 1994;104:946-949.
- Weider D, Ryder C, Stram J. Benign paroxysmal positional vertigo: analysis of 44 cases treated by the canalith repositioning procedure of Epley. *Am J Otol*. 1994;15:321-326.
- Parnes L, Price-Jones G. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol*. 1993;102:325-331.
- Harvey S, Hain T, Adamiec L. Modified liberatory maneuver: effective treatment for benign paroxysmal positional vertigo. *Laryngoscope*. 1994;104:1206-1212.
- Steenerson R, Cronin G. Comparison of the canalith repositioning procedure and vestibular habituation training in forty patients with benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1996;114:61-64.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

Koelliker, Summers & Hawkins

-
37. Brandt T, Steddin S, Eng D, et al. Therapy for benign paroxysmal positional vertigo, revisited. *Neurology*. 1994;44:796-800.
 38. De la Meilleure G, Depondt M, Damman W, et al. Benign paroxysmal vertigo of the horizontal canal. *J Neurol Neurosurg Psychiatry*. 1996;60:68-71.
 39. Nuti D, Vannucchi P, Pagnini P. Benign paroxysmal positional vertigo of the horizontal canal: a form of canalolithiasis with variable clinical features. *J Vestib Res*. 1996;6:173-184.
 40. Lempert T, Tiel-Wick K. A positional maneuver for treatment of horizontal canal benign positional vertigo. *Laryngoscope*. 1996;106:476-478.
 31. Baloh R, Jacobson K, Honrubia V. Horizontal canal variant or benign positional vertigo. *Neurology*. 1993;43:2542-2549.
 42. Steddin S, Ing D, Brandt T. Horizontal canal benign paroxysmal positional vertigo (h-BPPV): transition of canalolithiasis to cupulolithiasis. *Ann Neurol*. 1996;40:918-922.