

Benign paroxysmal positional vertigo

Part I: Background and clinical presentation

Gabrielle M van der Velde, BSc, DC, FCCS(C)*

Purpose: To review recent theories regarding the aetiology and pathophysiology of benign paroxysmal positional vertigo (BPPV), including its epidemiology, clinical presentation, diagnosis, and differential diagnosis.

Data sources: Relevant studies were identified by searching MEDLINE from 1966 – March, 1997.

Study selection: A total of 35 studies were selected on the basis of their relevance to Part I of this review.

Data extraction: The findings and results of relevant studies and their subsequent theories and conclusions are discussed and compiled into a general overview of BPPV.

Results of data synthesis: BPPV is considered the most common cause of vertigo of peripheral origin. A potential causal association has been observed with numerous apparent aetiological factors, all of which may lead to peripheral vestibular trauma. Findings of densities within the posterior semi-circular canal have given rise to the most recent theories regarding the pathophysiology for BPPV, canalithiasis and cupulolithiasis.

Conclusions: BPPV is a multiaetiological peripheral vestibular disease whose underlying cause remains an enigma. The existing evidence supports two recent pathophysiological theories, cupulolithiasis and canalithiasis. Two conditions of special concern to the chiropractor, vertebrobasilar insufficiency and cervicogenic vertigo, closely resemble BPPV, and can be differentiated by certain identifying features. BPPV may be diagnosed clinically, after ruling out conditions in which vertigo is a central feature. A review of the

But : L'étude vise à passer en revue les théories récentes sur l'étiologie et la physiopathologie du vertige positionnel paroxystique bénin (VPPB), ainsi que sur l'épidémiologie, le tableau clinique, le diagnostic et le diagnostic différentiel de l'affection.

Sources des données : Des études pertinents ont été relevées dans la base de données MEDLINE, depuis 1966 jusqu'à mars 1997.

Choix des études : Trente-cinq études, au total, ont été retenues en raison de leur pertinence avec la première partie du présent examen.

Extraction des données : Les observations et les résultats tirés des études choisies ainsi que les théories et les conclusions auxquelles ils ont donné lieu font l'objet de discussion et ont été regroupés dans un exposé sommaire sur le VPPB.

Résultats de la synthèse des données : Le VPPB est considéré comme la cause la plus courante de vertige de'origine périphérique. Un lien causal possible a été observé entre le VPPB et de nombreux facteurs étiologiques apparents, qui semblent tous indiquer un traumatisme vestibulaire périphérique. Les résultats des examens de la densité du canal semi-circulaire postérieur ont donné lieu à l'ébauche de théories sur la physiopathologie du VPPB, la canaloithiase et la cupulolithiase.

Conclusion : Le VPPB est une maladie vestibulaire périphérique multifactorielle dont la cause sous-jacente demeure inconnue. Les données actuelles étayent deux théories récentes sur la physiopathologie du VPPB, soit la cupuloithiase et la canalolithiase. Deux affections doivent retenir l'attention des chiropraticiens :

* Division of Graduate Studies and Research, Canadian Memorial Chiropractic College, Toronto, Ontario, Canada.

Correspondence: Dr. Gabrielle M. van der Velde, Division of Graduate Studies and Research, Canadian Memorial Chiropractic College, 1900 Bayview Avenue, Toronto, Ontario, Canada M4G 3E6.

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treatment for BPPV, focusing on recent physical treatments will be discussed in Part II of this paper. (JCCA 1999; 43(1):31–40)

KEY WORDS: dizziness, vertigo.

Introduction

Vertigo is the cardinal subjective manifestation of vestibular disease, and may be experienced as a sensation of spinning (subjective vertigo) or a sensation of the world spinning about one's self (objective vertigo).¹ A vertiginous patient will likely complain of "dizziness", a non-specific term used to describe a number of symptoms, including unsteadiness, giddiness, light-headedness, dysequilibrium, and vertigo. Symptoms of imbalance, light-headedness, or syncope are not suggestive of vestibular system involvement and must be differentiated from true vertigo.

Benign paroxysmal positional vertigo (BPPV) is a clinical syndrome characterized by recurrent, brief episodes of severe vertigo and rotary nystagmus, precipitated by specific positions of the head relative to gravity.² This type of positional vertigo was first described by Barany in 1921, who believed it resulted from a peripheral vestibular system disorder.³ The clinical features of BPPV were further described in 1952 by the originators of the Dix-Hallpike manoeuvre.⁴ This provocative positioning manoeuvre induces the pathognomonic burst of rotary nystagmus with associated vertigo through a rapid change of head position from sitting to head-hanging positions. Dix and Hallpike perceived the lowermost ear in this manoeuvre as the source of BPPV. Decades later, BPPV is still viewed as a peripheral vestibular system disorder, despite the lack of a demonstrable lesion at this level.

BPPV has been estimated to account for 17–30% of all cases of vertigo presenting to vestibular clinics,^{5,6,7,8} making it an important differential diagnosis for this com-

l'insuffisance vertébro-basilaire et le vertige d'origine cervicale, que ressemblent beaucoup au VPPB mais dont certains signes caractéristiques permettent de les différencier de celui-ci. Le diagnostic de VPPB peut être posé sur le plan clinique après élimination des causes ayant comme principale manifestation le vertige. Un examen des moyens permettant de traiter le VPPB, notamment les nouveaux traitements physiques, est présenté dans la deuxième partie du présent article. (JACC 1999; 43(1):31–40)

MOTS CLÉS : étourdissement, vertige.

plaint. Chiropractors should therefore be familiar with the clinical presentation of BPPV, as well as alert to conditions which closely resemble it, when attempting to diagnose the cause of provoked vertigo in a patient. This paper discusses current aetiological and pathophysiological theories for BPPV and reviews its clinical presentation, diagnosis, and differential diagnosis.

Methods

To find background information about BPPV, a search of MEDLINE from 1966 – March 1997 was conducted. Textword searches were used, in which the term *benign positional paroxysmal vertigo* was combined with the following textwords: *epidemiology, natural history, etiology, pathophysiology, pathological mechanism, differential diagnosis, diagnosis, canalithiasis, and cupulolithiasis*. Reference lists of the retrieved studies were hand searched to identify additional relevant studies. Studies were restricted to French and English-language publications.

Results

A total of 65 studies were identified. Of these, 35 were accepted for the purposes of this review. The criteria for acceptance were based on relevance to this review and included landmark papers on BPPV, epidemiologic studies, retrospective chart reviews, clinical trials, histologic studies, and descriptive observational studies. Studies not included included introductory papers on BPPV and single case reports. The results of the retrieved studies are summarized in the discussion below.

Discussion

Epidemiology: frequency of BPPV

The annual incidence rate for BPPV in the general population has been estimated at 64 per 100,000 in the United States, and 10.7 per 100,000 in Japan.^{9,10} Overall prevalence of BPPV in the general population has been estimated in Japan at 17.7 per 100,000.¹⁰ BPPV is the most commonly observed peripheral vestibular disorder, accounting for 17 to 30% of new patients in vestibular clinics. It is considered the most common cause of vertigo of peripheral origin.^{5,6,7,8,12} BPPV is generally seen in individuals aged 40 years and older, with the highest age distribution between 50 to 70 years.^{5,9,10,11,13,14} It rarely appears before the age of 20. A higher prevalence has been reported in women.^{9,10,11,13,14,15}

Natural history

Paroxysmal positional vertigo is generally regarded as a self-limiting disorder and therefore considered benign.^{9,16} In most cases, spontaneous recovery is expected within weeks to months.^{9,17} In 20–30% of patients, BPPV persists or recurs for years when untreated.^{14,18,19,20} In recurrent cases, attacks are episodic and often occur in clusters over a period of weeks with remissions of months or years.^{20,21}

Pathophysiology

The signs and symptoms of BPPV are generally believed to originate from the peripheral vestibular system, although a conclusive, demonstrable lesion at this level has not been confirmed.^{5,14}

Positional vertigo has been reported as a central feature in conditions where ischemic lesions were found in the central nervous system (CNS).¹⁴ In one case series, 14 of the 240 BPPV patients had symptoms or signs indicative of associated CNS involvement.²¹ However, as the majority of patients suffering from positional vertigo do not exhibit associated abnormalities of the central nervous system, many authors view reports of CNS abnormalities associated with BPPV as coincidental, explaining these BPPV patients were likely suffering from a concurrent disorder of the CNS.^{14,22} Often, reports of positional vertigo of central origin fall short of demonstrating the complete diagnostic criteria for BPPV or a satisfactory description of the clinical signs was not provided.

Cervical spine or vascular conditions, such as basilar vertebral insufficiency secondary to osteophytic compression of the vertebral artery, have been reported in cases of BPPV, suggesting that lesions at these sites may also be responsible for provoked vertigo of the benign paroxysmal type. As with the apparent cases of BPPV of central origin, many authors reject these reports of BPPV of cervical or vascular origin as coincidental, since the presence of true rotary nystagmus of limited duration was not established.^{7,23} Most conclude there is no good evidence supporting a role of cervical spine or vascular disease in the aetiology of BPPV.¹⁶

Several features of BPPV suggest involvement of the posterior semicircular canal (PSC). The vertigo and nystagmus associated with BPPV are typically most intense with changes in head position in the plane of one of the PSCs.^{18,24} Singular neurectomy or partial posterior canal occlusion to the involved posterior canal relieves BPPV symptoms.^{24,25} The associated nystagmus is primarily rotary, and can be evoked by stimulation of the PSC, implying ampullofugal deflection of the cupula in the PSC.²⁴ Cases of BPPV apparently involving the horizontal semicircular canal (HSC) have also been reported, based on observations of horizontal-type nystagmus, and other signs consistent with stimulation of the HSC.²⁵

Aetiology

The exact aetiology of BPPV is still under debate. Case series and chart reviews, attempting to formulate hypotheses on the aetiological origins of BPPV, have observed an apparent association with BPPV and certain conditions. These conditions include head (labyrinthine) trauma, stapes surgery, viral neurolabyrinthitis, chronic suppurative otitis media, mastoiditis, and vestibular neuritis.^{13,14,15,26} All these conditions constitute mechanisms which could theoretically lead to peripheral vestibular trauma. By far the most frequently reported cases of BPPV are idiopathic, where no association was found. These cases of idiopathic BPPV account for over 50% of all reported cases.^{8,13,14} In perimenopausal women, idiopathic BPPV prevails, whereas in young men, a post-traumatic aetiology is more common.²⁷

Diagnosis

The specific clinical characteristics of BPPV include: 1) acute onset of vertigo and nystagmus induced by pro-

vocative positioning of the head with the affected ear down, 2) vertigo and nystagmus having a brief latent onset period (1–30 seconds), 3) vertigo and nystagmus of limited duration (15–30 seconds), 4) characteristic rotary nystagmus in head hanging position, 5) reversal of nystagmus on upright sitting position of shorter duration, and 6) fatiguability of the response to the Dix-Hallpike (Barany-Nylan) manoeuvre with repeated positioning.^{17,19} A diagnosis of BPPV can be made by correlating historical data and clinical findings.^{13,25,28} Patients typically complain of episodes of severe dizziness of sudden onset, precipitated by changes in head position, lasting less than a minute. Attacks may be provoked by lying down or rising out of bed, rolling over, suddenly turning the head to one side, bending forward and straightening up, or by throwing the head back and to one side as when reaching for something on a shelf. Nausea and vomiting rarely accompany the attacks.²⁹

Clinical diagnosis is dependent on an accurate history and a functional evaluation which includes the demonstration of a paroxysmal positioning nystagmus (observed best under Frenzel's glasses or electronystagmography (ENG)), accompanied by vertigo of short duration occurring after a brief latent period (1–5 seconds), with the Dix-Hallpike manoeuvre.³⁰ The nystagmus is predominantly rotary with a linear component dependent on the direction of the gaze.^{13,28} When the gaze is directed to the lowermost ear, the fast phase of the nystagmus beats downward. When the gaze is directed to the uppermost ear, the nystagmus beat upwards, towards the forehead.^{18,20} While maintaining the precipitating head position, the complete cycle of nystagmus subsides quickly after 10 to 40 seconds and the accompanying provoked vertigo will cease. Immediately after, when returned to the upright sitting position, the patient may experience another, less severe and shorter attack of vertigo, along with a reversal in the direction of the nystagmus.²⁰ If the Dix-Hallpike manoeuvre is immediately repeated, a more brief and less severe attack of nystagmus and vertigo may result. With repeated positioning, the nystagmus and vertigo diminishes. This less marked or absent response to the Dix-Hallpike manoeuvre with repeated positioning demonstrates the fatiguability of the response. Audiological findings and caloric-induced response are generally within normal limits.²⁴

Not all cases of BPPV are classic in their presenta-

tion.^{28,30} Atypical histories may be associated with positive Dix-Hallpike manoeuvres, and histories strongly suggestive of provoked vertigo are either not accompanied by a positive Dix-Hallpike manoeuvre or nystagmus cannot be detected. In the latter cases, many authors caution on relying solely on the Dix-Hallpike manoeuvre, urging the use of other provocative manoeuvres, such as the vestibular habituation training test battery, to elicit paroxysmal positioning nystagmus, or the use of ENG to identify undetected nystagmus.^{28,30,31} The reader should note that estimates of the specificity and sensitivity of the Dix-Hallpike manoeuvre have apparently not been reported, as a Medline search (January 1963 – August 1998), using the keywords “sensitivity”, “specificity”, “Dix maneuver”, and “Dix-Hallpike maneuver”, failed to identify manuscripts examining these issues.

Differential diagnoses

As numerous conditions present with vertigo, a comprehensive list of differential diagnoses must be considered (Table 1). The somewhat daunting task of ruling out differential diagnoses for BPPV can be rendered more manageable by categorizing the vertigo by site of origin, either central or peripheral, depending on the neuroanatomic location of the causative lesion.^{1,31}

Vertigo of central origin results from lesions of the central vestibular apparatus (including the brainstem vestibular nuclei and their central connections). Central causes of vertigo include demyelinating diseases (multiple sclerosis, post-infectious demyelination), arteriovenous malformations, intracranial and extracranial tumours of the brainstem and cerebellum (acoustic neuroma, extracranial meningioma, intracranial and extracranial metastatic tumor), cerebral haemorrhage, and brainstem vascular disease (Wallenberg's syndrome, transient vascular insufficiency in vertebrobasilar migraine, vertebrobasilar insufficiency).^{1,31,33} Of the central causes, vertebrobasilar insufficiency closely resembles BPPV.

Vertigo of peripheral origin includes conditions affecting the peripheral vestibular apparatus (including the internal ear labyrinth and the vestibular portion of the acoustic nerve) or the proprioceptive sense organs of the cervical spine. Peripheral causes of vertigo, localized to the peripheral vestibular apparatus, include Meniere's syndrome, Lermoyez syndrome (a variant of Meniere's syndrome), labyrinthitis, vestibular neuronitis, acoustic neuroma, di-

Table 1
Differential diagnosis for vertigo

Origin	Central	Peripheral
	arteriovenous malformations brainstem vascular disease <ul style="list-style-type: none"> – Wallenberg's syndrome – transient vascular insufficiency (in vertebrobasilar migraine) – vertebrobasilar insufficiency cerebral haemorrhage cerebromedullary junction malformations <ul style="list-style-type: none"> – Arnold-Chiari malformation demyelinating diseases <ul style="list-style-type: none"> – multiple sclerosis – post-infectious demyelination tumours of brainstem and cerebellum <ul style="list-style-type: none"> – acoustic neuroma – meningioma – metastatic tumour 	acoustic neuroma Barre-Lieou syndrome benign paroxysmal positional vertigo cervicogenic vertigo direct trauma to semicircular canals labyrinthitis Lermoyez syndrome Meniere's syndrome perilymphatic fistula semicircular canal infection semicircular canal water penetration vestibular neuronitis

rect trauma to or infection of the semicircular canals, vestibulotoxic pharmaceuticals or substances (furosemide, salicylates, quinine, barbituates, tranquilizers, diphenyl hydantoin, antihistamines, arsenic, mercury, lead), or irritation of the semicircular canals by water penetration.³⁰ Peripheral causes of vertigo affecting the proprioceptive afferents of the cervical spine include Barre-Lieou syndrome and cervicogenic vertigo.^{1,31,32,34} Of the peripheral causes, cervicogenic vertigo closely resembles BPPV.

Fortunately, a number of key features distinguish BPPV from other diagnoses. First, the vertigo seen in BPPV is provoked, present only when the eliciting movement is executed.¹ In contrast, all the conditions above exhibit a spontaneous type vertigo which occurs at rest. Cervicogenic vertigo and vertebrobasilar insufficiency represent the only exceptions to this rule.¹ While a sudden position change can occasionally induce vertigo in cases of central lesions or certain vestibular-labyrinthine disorders, such as labyrinthitis, the resultant vertigo is not characterized by the key features of the benign paroxysmal type, namely, latency, brevity, and fatiguability of response.^{1,35}

Second, vertigo of peripheral origin, as in BPPV, exhib-

its clinical characteristics distinguishing it from vertigo of central origin. Vertigo of peripheral origin is severe, with a brief latent response period of up to 40 seconds, a fatigable response, and habituation to repeated testing. In contrast, vertigo of central origin is relatively mild, there is no latent period in the response to provocative positioning, and there is no fatiguability or habituation to repeated provocative positioning.^{1,31} Third, BPPV exhibits isolated provoked vertigo and nystagmus, not associated with other clinical signs or symptoms. In contrast, every differential diagnosis for BPPV typically presents with additional signs and symptoms, such as tinnitus, hearing loss, motor, sensory or cerebellar deficits, nausea, and vomiting, in addition to vertigo and nystagmus. Cervicogenic vertigo is sometimes a rare exception to this rule, but it is believed that even this condition most often presents with associated neck pain and headaches.^{34,36}

Differentiating BPPV from vertebrobasilar insufficiency and cervicogenic vertigo

Certain fundamental features of vertebrobasilar insufficiency and cervicogenic vertigo help to further identify them from BPPV (Table 2). First, the vertigo in BPPV is

provoked by movements or *change* in of the head position, as opposed to *maintained* by a particular head posture. This feature of BPPV has lead some to argue that it would be more accurate to label the type of vertigo seen in BPPV as a “positioning” rather than a “positional” type vertigo.^{1,28} True positional vertigo, they maintain, is observed

in vertebrobasilar insufficiency, where the signs and symptoms continue as long the head position is held (Unfortunately, this belief is not current with recent, incontestable evidence confirming the invalidness of vertebrobasilar insufficiency positioning manoeuvres.³⁷ This point should be kept in mind during the following discus-

Table 2
Features of BPPV, vertebrobasilar insufficiency (VBI), and cervicogenic vertigo

Condition	Type of vertigo	Associated signs & symptoms	Characteristics of nystagmus
BPPV	provoked positioning-type	nystagmus	short latency of onset period (1–5 sec), brief (<30 sec), fatiguable / habituable with repeated testing
VBI	provoked positional-type	nystagmus, nausea, numbness diplopia, drop attacks, dysphagia, dysarthria, ataxia	long latency period (< 50 sec), increased intensity of symptoms with maintenance of head position, not fatiguable/ habituable with repeated testing
Cervicogenic vertigo	provoked positioning-type	nystagmus, neck pain, cervical dysfunction, suboccipital headaches	no latency period, brief, fatiguable/ habituable to repeated testing

Table 3
Comparison of two recent pathophysiological theories for BPPV

Theory	Cupulolithiasis	Canalithiasis
Originator(s)	Schuknecht, 1969	Hall, Ruby, and McClure, 1979
Location of lesion	posterior semicircular canal (PSC)	posterior semicircular canal (PSC)
Proposed pathophysiology	cupuloliths (basophilic densities adhered to the PSC cupula) alter the specific gravity of the cupula making it sensitive to gravitational changes	canaliths (free-floating PSC endolymphatic densities) create a hydrodynamic drag which displaces and stimulates the cupula
Supportive evidence	1) histological observation of cupular basophilic densities 2) reports of positive responses to physical treatment inspired by this pathophysical theory	1) operative observation of free-floating endolymphatic densities 2) reports of positive responses to physical treatment inspired by this pathophysical theory

sion). In cervicogenic vertigo, there is neither change nor maintenance in head position, as vertigo and nystagmus are elicited by torsioning the neck, while keeping the head immobile.³⁴ Second, whilst the nystagmic response in vertebrobasilar insufficiency has a latency period as in BPPV, the latency has been reported to be far longer than that of BPPV, lasting up to 50 seconds.³⁶ In cervicogenic vertigo, the onset of nystagmus is reported to be immediate, having no latency of onset.³⁶ Moreover, the nystagmus in vertebrobasilar insufficiency tends to increase in intensity with maintenance in head position.³⁶ whereas in BPPV and cervicogenic vertigo there is decrease in nystagmus within seconds. Habituation to repeated testing in the provocative position does not occur in vertebrobasilar insufficiency as it does in BPPV and cervicogenic vertigo. Finally, vertebrobasilar insufficiency, on provocative positioning, tends to be associated with additional signs and symptoms, including disturbances in vision, diplopia, nausea, ataxia, impairment of trigeminal sensation, dysarthria, hemianaesthesia, and hemiplegia.³⁸ Cervicogenic vertigo is believed to present with neck pain, suboccipital and occipital headaches, and manual findings including segmental dysfunction and palpatory tenderness of the upper cervical musculature (The reader should note that these features have been established only on the basis of a few case series).^{34,35} Outside of vertigo and nystagmus, there are no associated signs and symptoms in BPPV.

Recent pathophysiological theories

Cupulolithiasis

Two theories have been put forth to explain the clinical characteristics of BPPV (Table 3). The first, known as cupulolithiasis or “heavy cupula” theory, was proposed by Shuknecht in 1969.³⁹ This theory is based on histological observations of basophilic densities, adhered to the cupulas of the PSCs, in post-mortem temporal bone specimens of three individuals known to have suffered from BPPV. He proposed that sedimentous material, possibly detached utricular otoconia of the maculae, could be released into the endolymph of the PSC as a result of either trauma (head blow, post-stapedectomy, chronic suppurative otitis media) or degenerative changes associated with aging. When the head is in the upright position, the sediment, which has a specific gravity greater than that of endolymph, would gravitate to and settle on the cupula of

the PSC. Normally, the cupula of the PSC has the same specific density as the endolymph and responds only to angular acceleration. In this theory, fixed deposits on the PSC cupula are proposed to create a “heavy cupula”, sensitive to gravitational force and therefore sensitive to head position¹⁷

Shuknecht and Ruby attempted to estimate the prevalence of cupular deposits by performing serial dissections of temporal bones in cadavres.¹⁷ Of 391 temporal bones examined, 149 had cupular deposits of varying sizes. The deposits were as likely to be found in normal temporal bones as in those which exhibited pathological changes. It was unknown whether the specimens were of individuals known to have suffered from BPPV, therefore the detection of cupular deposits could not be correlated with clinical manifestations of BPPV. In the 149 cases with cupular deposits, four had deposits that were as large as those identified in the three known BPPV sufferers. Based on their observational evidence, Shuknecht and Ruby renamed the type of positional vertigo originally described by Barany and by Dix and Hallpike as cupulolithiasis.

A recent serial dissection of 566 postmortem temporal bone specimens demonstrated cupular deposits in all three semicircular canals.⁴⁰ Cupular liths were observed in 216 of the 1031 semicircular canals dissected, with a higher percentage found in the posterior semicircular canal relative to the superior and lateral semicircular canals. Again, it was unknown whether any of the individuals from which the specimens were taken had been BPPV sufferers. These serial dissections suggest that cupular deposits are relatively common, but their significance has yet to be determined.

Canalithiasis

The second theory, canalithiasis, was introduced by Hall, Ruby and McClure in 1979.²² They proposed that the signs and symptoms observed in BPPV were the result of free-floating endolymphatic densities in the PSC. During positional testing, movement of the particles within the endolymph would create a current and the resultant hydrodynamic drag would then displace and stimulate the cupula.

The canalithiasis theory has been recently supported by direct observation of free-floating particles in the endolymph of PSCs, during operative procedure.¹⁶ This pathophysiological theory has gained more acceptance

since it best explains the clinical manifestations of BPPV.^{7,12,41,42,43,44} After the affected ear is placed in the dependent position, several seconds are necessary for the movement of the particles to overcome the inertia and resistance of the endolymph within the canal to create sufficient hydrodynamic drag to stimulate the cupula.⁴² This accounts for the brief latent period before the onset of nystagmus. Reversal of the nystagmus on return to sitting position can be explained by the movement of the particles filtering back into the inferior aspect of the PSC.⁴² Some suggest that the fatiguability of the response to repeated positional testing is most likely due to the dissolution of the clumped particulate material in the endolymph, which reduces the hydrodynamic drag on the cupula.⁴³ The ability of canalithiasis to explain the clinical manifestations of BPPV cases apparently involving the horizontal semicircular canals further accounts for the growing acceptance of this theory.²⁵

Obtaining conclusive evidence regarding the underlying pathophysiology for BPPV has been complicated by a number of factors, notwithstanding the difficulty in observing in vivo what is likely a microscopic lesion or mechanism at the peripheral vestibular system level. This accounts for the greatest shortcoming of the existing evidence: the inadequate correlation of histological findings in cadaveric specimens to known BPPV sufferers. Nonetheless, cupulolithiasis and canalithiasis are convincing theories as they incorporate the available evidence and explain the clinical manifestations of BPPV.

Some authors have suggested that cupulolithiasis and canalithiasis represent two stages of a pathological process which begins with production of endolymphatic sediments resulting from insult to the semicircular canals. Perhaps the process of canalithiasis and cupulolithiasis occurs simultaneously in some cases. All presumed aetiological factors for BPPV may lead to endolymphatic densities, either free-floating or adhered to the cupula, which mechanically stimulate the PSC cupula. White blood cells, phagocytes, and endothelial fragments might be found following infections such as viral labyrinthitis or chronic suppurative otitis media. Red blood cells might be found following stapedectomy or head trauma. Idiopathic BPPV suggests degenerative changes associated with aging, leading to degenerated cellular debris or otoconia released into the endolymph.^{16,17}

Treatment

Treatment for BPPV includes expectant observation (relying on the generally self-limiting natural history of BPPV), medication, operative procedures for intractable cases, and physical treatment.^{6,45,46,47,48} A more comprehensive review of treatment for BPPV, focusing on recent physical treatments inspired by the theory of canalithiasis, will be presented in Part II of this paper.

Conclusion

BPPV is an important differential diagnosis for the complaint of dizziness as it is believed to represent the most common cause of vertigo of peripheral origin. BPPV closely resembles two conditions of particular interest to chiropractors: vertebrobasilar insufficiency and cervicogenic vertigo. Knowledge of the clinical features of these three conditions may help to differentiate them, however, as there is no valid clinical test for vertebrobasilar insufficiency and the clinical features of cervicogenic vertigo have not been conclusively established, this endeavour will be difficult. Further, estimates of the sensitivity and specificity of the Dix-Hallpike manoeuvre have seemingly not been determined, necessitating studies in this area.

Since BPPV was first described, many aspects of this condition remain an enigma, largely because its underlying pathophysiology is still unknown. The existing evidence implicates a number of aetiologies, all of which may lead to one consequence: the mechanical stimulation of the cupula in the posterior semicircular canal. The pathophysiological theories based on this evidence, cupulolithiasis and canalithiasis, have inspired promising physical treatments for BPPV. The most recently developed physical treatment based on the theory of canalithiasis, the *particle repositioning procedure*, will be presented and discussed in Part II of this paper.

References

- 1 Tierney LM, McPhee SJ, Papadakis MA. eds. Current Medical Diagnosis and Treatment. 36th ed. 1997, Stamford, CT: Appleton & Lange, 210–212.
- 2 Katsarkas A, Outerbridge JS. Nystagmus of paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol* 1983; 92:146–150.
- 3 Barany R. Diagnose von Krankheitserscheinungen im Bereiche des Otolithenapparates. *Acta Otolaryngol* (Stockh) 1921; 2:434–437.

- 4 Dix MR, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med* 1952; 45:341–354.
- 5 McClure J, Lycett P, Rounthwaite J. Vestibular dysfunction associated with benign paroxysmal vertigo. *Laryngoscope* 1977; 87:1434–1442.
- 6 Herdman SJ, Tusa RJ, Zee DS, Proctor LR, Mattox DE. Single treatment approaches to benign paroxysmal positional vertigo. *Arch Otolaryngol Head Neck Surg* 1993; 119:450–454.
- 7 Parnes LS, Price-Jones RG. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Ann Otol Rhino Laryngol* 1993; 102:325–331.
- 8 Dumas G, Charachon R, Ghozali S. Vertige positionnel paroxystique benin. A propos de 51 observations. *Ann Oto-laryngol Chir Cervicofac* 1994; 111:301–313.
- 9 Froehling DA, Silverstein MD, Mohr DN, Beatty CW, Offord KP, Ballard DJ. Benign positional vertigo: incidence and prognosis in a population-based study in Olmstead County, Minnesota. *Mayo Clin Proc* 1991; 66:596–601.
- 10 Mizukoshi K, Watanabe Y, Shojaku H, Okubo J, Watanabe I. Epidemiological studies on benign paroxysmal positional vertigo in Japan. *Acta Otolaryngol (Stockh)* 1988; Suppl.447:67–72.
- 11 Dumas G, Charachon R, Gozali S. Vertige positionnel paroxystique benin. A propos de 51 observations. *Ann Oto-laryngol Chir Cervicofac* 1994; 111:301–313.
- 12 Massoud E, Ireland D. Post-treatment intrusions in nonsurgical management of benign paroxysmal positional vertigo. *J of Otolaryngol* 1996; 25(2):121–125.
- 13 Katsarkas A, Kirkham TH. Paroxysmal positional vertigo—a study of 255 cases. *J Otolaryngol* 1978; 7(4):320–330.
- 14 Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology* 1987; 37:371–378.
- 15 Bourgeois PM, Dehaene I. Benign paroxysmal positional vertigo (BPPV): clinical features in 34 cases and review of literature. *Acta Neuro Belg* 1988; 88:65–74.
- 16 Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. *Laryngoscope* 1992; 102:988–992.
- 17 Schuknecht HF, Ruby RRF. Cupulolithiasis *Adv Oto Rhino Laryng* 1973; 20:434–443.
- 18 Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol* 1980; 106:484–485.
- 19 Brandt T. Positional and positioning vertigo and nystagmus. *J Neurol Sci* 1990; 95:2–28.
- 20 Brandt T. Benign paroxysmal positioning vertigo (BPPV). In *Vertigo: its multisensory syndromes*. 1st ed. London: Springer-Verlag, 1991:139–151.
- 21 Harbert F. Benign paroxysmal positional nystagmus *Arch Ophthal* 1970; 84:298–302.
- 22 Hall SF, Ruby RRF, McClure JA. The mechanics of benign paroxysmal positional vertigo. *J Otolaryngol* 1979; 8(2):151–158.
- 23 Cawthorne T. The physiological basis for head exercises. *J Chart Soc Physiother* 1944; 30:106–107.
- 24 Gacek RR. Pathophysiology and management of cupulolithiasis. *Am J Otolaryngol* 1985; 6(2):66–74.
- 25 McClure JA. Horizontal canal benign paroxysmal vertigo. *J Otolaryngol* 1985; 14(1):30–35.
- 26 Gordon N. Post-traumatic vertigo with special reference to positional nystagmus. *Lancet* 1954; 266:1216–1218.
- 27 Blessing R, Strutz J, Beck C. Epidemiologie des benignen Paroxysmalen Lagerungsschwindels. *Laryng Rhinol Otol* 1986; 65:455–458.
- 28 Norre, ME. Diagnostic problems in patients with benign paroxysmal positional vertigo. *Laryngoscope* 1994; 104(11):1385–1388.
- 29 Lempert T, Gresty MA, Bronstein AM. Benign positional vertigo: recognition and treatment. *BMJ* 1995; 311:489–491.
- 30 Norre ME. Reliability of examination data in the diagnosis of benign paroxysmal positional vertigo. *Am J Otology* 1995; 16(6):806–810.
- 31 Kumar A, Petchenik L. The diagnosis and management of vertigo. *Comprehensive Therapy* 1990; 16(12):56–66.
- 32 Mumenthaler M. *Neurological differential diagnosis*. 2nd ed. New York: Thieme Medical Publishers Inc., 1992: 68–79.
- 33 Troost BT. Dizziness and vertigo in vertebrobasilar disease. Part 2: Central causes and vertebrobasilar disease. *Stroke* 1980; 11(4):413–415.
- 34 Troost BT. Dizziness and vertigo in vertebrobasilar disease. Part 1: Peripheral and systemic causes of dizziness. *Stroke* 1980; 11(3):301–303.
- 35 Cote P, Mior SA, Fitz-Ritson D. Cervicogenic vertigo: a report of three cases. *JCCA* 1991; 35(2):89–94.
- 36 Adams RD, Victor M. *Principles of Neurology*. 5th ed. New York: McGraw-Hill, 1993:247–269.
- 37 Côté P, Kreitz BG, Cassidy JD. The validity of the extension-rotation test as a clinical screening procedure before neck manipulation: a secondary analysis. *JMPT* 1996; 19(3):159–164.
- 38 Hulse M. Disequilibrium caused by a functional disturbance of the upper cervical spine, clinical aspects and differential diagnosis. *Manual Medicine* 1983; 1(1):18–23.
- 39 Henderson DJ, Cassidy JD. Vertebrobasilar vascular accidents associated with cervical manipulation. In: Vernon H. ed. *Upper cervical syndrome*. Baltimore: Williams and Wilkins. 1988:194–206.
- 40 Schuknecht HF. Cupulolithiasis. *Arch Otolaryng* 1969; 90:765–778.

- 41 Moriarty B, Rutka J, Hawke M. The incidence and distribution of cupular deposits in the labyrinth. *Laryngoscope* 1992; 102:56–59.
- 42 Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992; 107(3):399–404.
- 43 Epley JM. Positional vertigo related to semicircular canalithiasis. *Otolaryngol Head Neck Surg* 1995; 112(1): 154–161.
- 44 Welling DB, Barnes DE. Particle repositioning maneuver for benign paroxysmal vertigo. *Laryngoscope* 1994; 104:946–949.
- 45 Agus G, Puxeddu R, Demontis GP, Puxeddu P. Atypical “reversed” paroxysmal positioning nystagmus in benign paroxysmal positional vertigo. *Acta Otolaryngol (Stockh)* 1995; Suppl 520:143–147.
- 46 McClure JA, Willett JM. Lorazepam and diazepam in the treatment of benign paroxysmal vertigo. *J Otolaryngol* 1980; 9(6):472–477.
- 47 Herdman SJ. Treatment of benign paroxysmal positional vertigo. *Phys Ther* 1990; 70(6):381–387.
- 48 Parnes LS, McClure JA. Posterior semicircular canal occlusion in the normal hearing ear. *Otolaryngol Head and Neck Surg* 1991; 104(1):52–57.

CCA Researcher of the Year Award Nominations

The CCA Research Committee invites nominations and supporting documents for the Researcher of the Year Award. The recipient will receive the Award at the President’s cocktail reception during the semi-annual meeting of the Canadian Chiropractic Association in Toronto.

Deadline for submissions: June 1, 1999

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Dr. Grayden Bridge (Chair)	Dr. Zoltan Szaraz	Dr. David Peterson
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Please forward submissions to:

Dr. Allan Gotlib, Research Programs Co-ordinator,
Canadian Chiropractic Association,
1396 Eglinton Avenue West,
Toronto, Ontario M6C 2E4