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The Diagnosis and Treatment of Benign Paroxysmal Positional Vertigo

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THE DIAGNOSIS AND TREATMENT
OF BENIGN PAROXYSMAL POSITIONAL VERTIGO

by

Jennifer Anderson
Bachelor of Science in Physical Therapy
University of North Dakota, 1999

An Independent Study
Submitted to the Graduate Faculty of the
Department of Physical Therapy
School of Medicine
University of North Dakota
in partial fulfillment of the requirements
for the degree of
Master of Physical Therapy

Grand Forks, North Dakota
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2000
This Independent Study, submitted by Jennifer Anderson in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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(Chairperson, Physical Therapy)
PERMISSION

Title Diagnosis and Treatment of Benign Paroxysmal Positional Vertigo

Department Physical Therapy

Degree Master of Physical Therapy

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ABSTRACT

Benign paroxysmal positional vertigo (BPPV) is a disorder of the peripheral vestibular system presenting with nystagmus and vertigo. Other symptoms experienced include nausea, vomiting, and balance disturbances. The purpose of this paper is to review diagnostic and rehabilitation techniques currently being utilized. First, the anatomy and physiology of the peripheral vestibular system will be explained. Next, the diagnostic procedures performed by a physician as well as a physical therapist will be discussed. Following, current rehabilitative techniques will be explored. In conclusion is the author’s opinion of the diagnostic and therapeutic techniques.
CHAPTER I

INTRODUCTION

Dizziness is the primary complaint in 2.5% of all primary care visits, accounting for over 8 million visits per year. After age seventy, it becomes the number one reason for seeking medical attention.\(^1\)\(^2\) Vertigo is defined as the sensation of moving around in space or of having objects move about the person.\(^4\) It may result from metabolic imbalances, otologic, cardiovascular, psychological, or neurologic conditions. Meniere’s disease, a chronic inner ear ailment, is characterized by progressive deafness, tinnitus, and a sensation of fullness or pressure in the ear.\(^3\)\(^4\) Labyrinthitis is another common otologic disorder, presenting with vertigo, vomiting, and nystagmus. Presyncope refers to loss of consciousness or fainting, resulting from decreased blood flow to the brain, leading to a shortage of oxygen and glucose.\(^4\)

The vestibular system is composed of three components: a peripheral sensory apparatus, a central processor, and motor output. Motion sensors of the peripheral portion consists of motion sensors which send information to the central nervous system about angular velocity, linear acceleration, and orientation of the head. The central nervous system processes these signals, combines them with other sensory information to estimate the orientation of the head. The output of the vestibular system goes to the ocular muscles and spinal cord. The vestibulo-ocular reflex generates eye movements
enabling clear vision while the head is moving. The vestibulo-spinal reflex generates compensatory body movements to maintain head and postural stability.\textsuperscript{11

In order for a diagnosis of BPPV to be made, a thorough examination is imperative. During the evaluation, the following areas are assessed: physical status, gaze stabilization, balance control, and vertiginous positions. The illness is considered benign because of its spontaneous recovery within several weeks or months. However, if left untreated, 20\% to 30\% of cases persist or reoccur after variable periods of time.\textsuperscript{5} Thus, after a diagnosis has been confirmed, the appropriate rehabilitation program can be sought. Various exercises are aimed at treatment of disequilibrium and dizziness associated with vestibular pathology.

Benign paroxysmal positional vertigo was described by Barany in 1921 as brief attacks of vertigo and linear-rotatory nystagmus precipitated by rapid head extension and lateral head tilt toward the affected ear.\textsuperscript{5-7,9} Other symptoms experienced include unsteadiness in gait and postural balance, and nausea.\textsuperscript{5} In 1952, Dix and Hallpike described a maneuver to elicit the classic symptoms and confirm a diagnosis of BPPV. With the patient sitting on a plinth, the physical therapist rapidly moves the individual into a supine position, extending the head $30^\circ$ below the exam table, while rotating the head approximately $40^\circ$ to one side. The patient’s eyes are observed for nystagmus. The procedure is then repeated to the other side to determine which ear is affected.\textsuperscript{5,6} The procedure is performed with the patient wearing magnified frenzel glasses. The lenses prevent suppression of symptoms by fixation, which could lead to inaccurate test results.\textsuperscript{5} Two other diagnostic techniques include recording of eye movements through
electronystagmographic testing. Bithermal caloric testing involves irrigating the ear with water of varying temperatures, which will illicit abnormal vestibular functioning.8

Benign paroxysmal positional vertigo exhibits clinical manifestations that separate it from other conditions. Symptoms are induced when the patient is moved into the Hallpike-Dix position. The nystagmus is latent, with onset between 1 and 40 seconds. It ceases within one minute once in the provoking pose. In all cases of BPPV, the pattern of nystagmus is identical. The beating occurs to the undermost and affected ear with a rotatory component; clockwise in left ear lesions and counterclockwise in right ear lesions. Constant repetition of the maneuver results in lessening of symptoms. This process is referred to as fatigability.5-7,10

Benign paroxysmal positional vertigo may be caused by a number of conditions including head trauma, vertebro-basilar ischemia, or prolonged bedrest. However, the origin to a majority of cases remains an enigma; with women predominating in a two to one ratio.

The purpose of this paper is to review the peripheral vestibular system and rehabilitative procedures available for those suffering from benign paroxysmal positional vertigo. Chapter II will focus on the anatomy and physiology of the inner ear. The pathology of the vestibular system will be discussed next. The diagnostic requirements and techniques will be explained, followed by current rehabilitation options which are available to the patient. In the conclusion will be the author’s view of the diagnostic and rehabilitation techniques.
CHAPTER II
ANATOMY AND PHYSIOLOGY

In order to understand BPPV, one must have a thorough grasp of the anatomy and physiology of the vestibular system. The peripheral vestibular system is located in the inner ear. It contains the bony and membranous labyrinths and hair cells, the motion sensors of the system.\textsuperscript{12,13-16}

The osseous labyrinth consists of three semicircular canals and a central chamber, the vestibule, which houses the utricle and saccule (figure 1). The labyrinth is filled with perilymphatic fluid. It is similar in consistency to cerebrospinal fluid (CSF), a high sodium (Na\textsuperscript{+}) to potassium (K\textsuperscript{+}) ratio. This liquid communicates with CSF through the cochlear aqueduct and subarachnoid space. The membranous portion of the labyrinth is suspended within the corresponding bony canals by supportive connective tissue and endolymph. It contains five sensory and two otolith organs, the utricle and saccule. Endolymphatic fluid resembles intracellular fluid, a high K\textsuperscript{+} and low Na\textsuperscript{+} concentration. No communication exists between the two inner ear fluids.\textsuperscript{13}

The semicircular canals are small ring-like structures comprising approximately two-thirds of a circle. One end of each canal is dilated to form an ampulla. The semicircular canals open into the posterior wall of the utricle through five openings. They are arranged at right angles to each other, representing the three planes of space.\textsuperscript{12-14}
Figure 1. The bony and membranous labyrinth. Reprinted with permission from Herdman SJ. *Vestibular Rehabilitation*. Philadelphia, PA: F. A. Davis Company; 1994: 5.
The six individual semicircular canals of the two inner ears become three coplanar pairs:
1) right and left lateral, 2) left anterior and right posterior, 3) left posterior and right anterior.

Located in the ampulla of the semicircular canals is the receptor area, the cristae. The cristae sense angular motion of the head. The sensory epithelium is covered by a gelatinous mass, the cupula. The utricle and saccule contain specialized epithelium known as the macula. On each macula is a statoconial membrane in which is embedded small calcium carbonate crystals, the otoconia. Unlike the cupula, the maculae are sensitive to gravity due to the increase in mass from the otoconia. The utricle and saccule are responsible for sensing linear acceleration of the head.16

Each hair cell is innervated by an afferent neuron. The main function is to convert displacement due to head motion into neural firing. Hair cells are suspended in each ampulla and otolith organ. In the ampulla, they rest on blood vessels, nerve fibers, and supporting tissue called the crista ampullaris. Hair cells are found on the medial wall of the saccule and the floor of the utricle. In each cell unit, there is one single thick and longer kinocilium and 50–110 thin stereocilia (figure 2). The stereocilia are arranged in a step-like fashion toward the single kinocilium. Hair cells of the ampulla, utricle, and saccule are deflected by endolymphatic flow. Displacement of the stereocilia towards the kinocilia causes depolarization of the hair cell. An increased discharge rate in adjacent afferent nerve fibers occurs and the vestibular nerve (cranial nerve VIII) is excited. If displacement occurs in the opposite direction, the impulses are inhibited or hyperpolarized.13-16
There is a difference in directional sensitivity of the horizontal and vertical canals. The kinocilia of the sensory cells in the horizontal canal are oriented towards the utricle, while those of the vertical canals are oriented away from the utricle. This phenomenon is described in Edwald's second and third laws; afferent nerve fibers of the horizontal canals are stimulated by endolymphatic movement towards the utricle (ampullopedal). Ampullofugal (away from the utricle) endolymph flow stimulates those of the vertical canal.\textsuperscript{13}

The impulses received by the hair cells are sent to the central nervous system via the vestibular nerve. Input from the primary afferents is directed at the vestibular nucleus complex and the cerebellum. The vestibular nucleus complex, consisting of four nuclei, is the primary processor of vestibular input, implementing direct fast connections between incoming afferent information and motor output neurons. The afferent neurons from the cristae of the semicircular canals end in the superior and medial vestibular nuclei, while the afferents of the lateral and descending nuclei arise from the utricular and saccular maculae.\textsuperscript{16}

The efferents proceed to one of five systems. Efferent fibers from the descending nucleus pass to the cerebellum and reticular formation. The fibers from the lateral nucleus ascend into the vestibulo-spinal tract ending in the ventral column of the spinal cord. The fibers continue and innervate appropriate postural muscles of the trunk and limbs needed to maintain balance. The efferent fibers from the medial nucleus project to the medial longitudinal fasiculus (MLF) and the vestibulo-cerebellum. It is responsible for responses such as nausea and vomiting which occur when the vestibular system is
disrupted. Another ability is that of coordinating eye, head, and neck movements. The fibers connecting with the superior nucleus continue to the MLF, and finally terminate in the oculomotor, trochlear, and abducen nerves (cranial nerves III, IV, and VI, respectively) which are responsible for innervating extrinsic eye musculature.\textsuperscript{13}

The functional purpose of the vestibulo-ocular reflex is to maintain stable vision while the body and or the head is in motion.\textsuperscript{12} For example, as one moves one’s head to the right, the eyes move to the left, maintaining gaze on a specific object. The opposite occurs as the head is turned to the left. When the head rotates to the right in the plane of the horizontal plane, the endolymph shifts. The stereocilia in the cupula are bent in the opposite direction, the left. When the stereocilia are bent toward the kinocilia, they are depolarized. Hyperpolarization occurs as they are bent away. Thus the receptors in the right and left semicircular canals work in functional pairs; when one is excited, the other is inhibited. Signals travel to the MLF and cranial nerves III and VI, which causes contraction of the left lateral rectus and right medial rectus. Compensatory deviation of the left and right eye to the left occurs, maintaining vision on a fixed object.\textsuperscript{12-17}

When one or more elements of the vestibular system are not functioning properly, incorrect impulses are sent to the brain via cranial nerve VIII. Although the head is stationary, the brain perceives that it is in motion. This causes the cardinal signs of BPPV such as vertigo, nystagmus, and balance difficulties. In the following chapter, the pathology of the vestibular system will be discussed.
CHAPTER III
PATHOPHYSIOLOGY OF THE VESTIBULAR SYSTEM

The vestibular system is an internal reference, informing the brain of how the head is oriented in space. External references include vision and the somatosensory system, providing details about movement of the surrounding environment. It is the harmonious integration of these sensory systems that provide normal equilibrium.\(^1\)

In a normally functioning vestibular system, the cerebellum and vestibular nucleus complex receive symmetrical impulses from the vestibular end organs, the semicircular canals, utriculae, and saculae. As the head moves, the vestibulo-ocular reflex is initiated, allowing gaze stabilization. Simultaneously, postural muscles of the limbs and trunk are stimulated to maintain balance.\(^1\)

After a unilateral vestibular insult, there is persistent asymmetry in the vestibular nerve discharge rates leading to the incorrect sensation of vertigo.\(^1\) The vestibulo-ocular reflex is also disrupted resulting in loss of clear vision when the head is moved into the provoking position.\(^1\)

Lesions of the vestibular system can occur anywhere along the connecting pathways. The vestibular end organs, vestibular nerve or nucleus, and brainstem or cerebellum are several common anatomic sites of insult. The causes of these lesions are
vast. A few include bacterial or viral infection, vascular disease, neoplasm, and trauma.\textsuperscript{10,18}

The exact pathophysiology of BPPV is uncertain. The earliest assumption was proposed by Barany in 1921. He believed the lesion was in the otolith organ. His theory was supported by Dix and Hallpike in 1952.\textsuperscript{5}

In 1969, Schuknecht performed studies of the temporal bones of two individuals who previously had been diagnosed with BPPV. He found basophilic deposits adhered to the cupula of the posterior canal of the affected ear. These basophilic deposits were hypothesized to be loosened calcium carbonate crystals (otoconia) of the utricular membrane that had migrated to the cupula of the semicircular canal.\textsuperscript{7} This condition was known as cupulolithiasis. When the head is upright, the posterior canal resides directly under the utricle, and thus becomes a receptacle for the disjoined particles.\textsuperscript{5}

Studies have supported the theory of cupulolithiasis. Ampulofugal stimulation of the posterior semicircular canal resulted in excitation of the ipsilateral superior oblique and contralateral inferior rectus muscles. The resultant linear-rotatory nystagmus was apparent. Secondly, chronic unilateral BPPV was eliminated after surgical severance of the ipsilateral posterior ampullary nerve.\textsuperscript{5}

Normally the cupula and endolymph have the same specific gravity. As the otoconia adhere to the posterior canal, an imbalance is created. This results in the posterior semicircular canal becoming oversensitive to angular acceleration in the plane specific to the canal.\textsuperscript{7} The cupula deflects abnormally, inducing vertigo, nystagmus, and nausea.\textsuperscript{5-7,10}
Another causative theory was hypothesized by Eply, Parnes, and McClure. They suggested that the degenerative debris were not adherent to the cupula but free floating in the endolymph. They termed this phenomenon canalithiasis. When the head is moved into the provoking position, the endolymph is moved by the falling otoconia. The neurons are excited as the cupula is pulled by the endolymph.

BPPV is not confined to the posterior semicircular canal. Recently, involvement of the anterior and horizontal canals has been reported. To correctly identify which semicircular canal is involved, the direction of the nystagmus is observed when the individual is first moved into the provoking position.

The anterior semicircular canal projects to the ipsilateral superior rectus and contralateral inferior oblique muscles. The nystagmus is therefore downbeating and torsional.

The horizontal semicircular canal stimulates the ipsilateral medial and contralateral lateral rectus muscles. The nystagmus produced is horizontal in nature. The provoking position for involvement of the horizontal canal is sidelying due to the alignment of the horizontal canal with respect to the pull of gravity. This differs from the Dix-Hallpike position used for posterior or anterior semicircular canal involvement.

The name benign paroxysmal positional vertigo implies that it is a positional disorder. Brandt, however, disagreed. He believed BPPV rather to be a disorder of positioning. His rationale is that the nystagmus and vertigo occur only when the head is rapidly moved into the provoking position. The intensity of the symptoms can vary from mild to severe, depending on the velocity of the positioning maneuver. Also, BPPV
attacks can be avoided if the challenging position is assumed slowly, generally longer than six seconds.

A thorough examination is imperative for the diagnosis of BPPV to be made. There are several characteristics that differentiate BPPV from other disease processes that may manifest with vertigo, nystagmus, and nausea.

These signs are supported in the two causative theories, cupulolithiasis and canalithiasis. The brief delay before the onset of nystagmus and vertigo is explained by the time it takes to overcome the inertia of the cupula or the time for endolymph to begin moving. Fatigability refers to the diminishment of symptoms within 60 seconds. Two ideas exist as to the reasoning of this. First, the lessening of vertigo and nystagmus as the provoking position is maintained is due to the cessation of endolymph flow, or it is due to the dispersement of the otoconial particles into the endolymph. Symptoms, however, will recur once the dispersed deposits resettle in the posterior semicircular canal.6,10

The diagnostic criteria and testing procedures utilized in the clinic by physicians as well as physical therapists will be discussed in detail in the following chapter.
CHAPTER IV

DIAGNOSIS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO

A thorough examination is essential to design an appropriate and effective rehabilitation program. The initial examination is usually performed by a physician who then refers the patient to a physical therapist for an evaluation and treatment. Using specific diagnostic techniques and criteria, the physician’s primary concern is to determine whether the disorder is peripheral in origin or more specifically if BPPV is suspected.

As part of the physician’s evaluation, a detailed report of the patient’s history is first obtained. The patient is asked to describe the sensation he/she is feeling in his/her own words. Dizziness is a sensation of altered orientation in space. External as well as internal references provide information as to the position of the head in space; thus, dizziness can occur when either are impaired. Dizziness is divided into four categories: 1) vertigo, 2) unsteadiness, 3) lightheadedness, and 4) giddiness. If the pathology is related to the vestibular system, the individual will describe an illusion or rotation. This is known as vertigo. Additional information of importance is concerned with the time course, precipitating factors, associated symptoms, and predisposing factors related to vertigo.
The time course of dizziness is an important feature in distinguishing its cause. Vertigo associated with labyrinthine disorders is short in duration, lasting less than 60 seconds. The onset is abrupt followed by decreasing intensity as the symptoms subside. Continuous dizziness without fluctuation indicates possible insult of central origin or systemic involvement.

The physician will want to know what events occurred prior to the episode of vertigo. Rapid movement of the head usually elicits dizziness related to vestibular pathology. Attacks can be precipitated by actions such as looking up or turning over in bed. If vertigo follows periods of coughing or sneezing, a perilymph fistula is suspected. A patient presenting with endolymphatic hydrops will report that loud noises precede attacks.

The coexisting symptoms reported by the patient would assist the physician in determining the cause of vertigo. Nausea and vomiting typically accompany dizziness associated with BPPV. There are other differential diagnoses that should be ruled out. A patient complaining of hearing loss and tinnitus is describing Menieres disease. Sudden complete unilateral deafness and dizziness are symptoms of a viral or bacterial labyrinthitis, requiring immediate medical attention. Epilepsy is considered if dizziness is followed by loss of consciousness.

An understanding of the patient’s general health status prior to the onset of episodes is imperative for identification of predisposing factors. Previous ear disease or a head injury can lead to bouts of dizziness. Illnesses such as diabetes, syphilis, and atherosclerosis may also cause vestibular damage. A patient with systemic disease may
complain of dizziness, nausea, and vomiting. These symptoms are also associated with BPPV; thus, the physician must distinguish between the two. Medications can produce dizziness in an individual without vestibular pathology. Several of importance include antihypertensives, barbiturates, and antihistamines.\(^\text{20}\)

After a complete history of the patient is obtained, a general examination of the ears, nose, and throat is performed.\(^\text{19,20}\) The primary emphasis is placed on the evaluation of the ears where the more crucial information is obtained. First, the physician must remove excess earwax which may cause canal obstruction, leading to hearing loss and dizziness. Next, the patient’s hearing is assessed using audiometric facilities or the forced whisper or tuning fork tests. The external canal and tympanic membrane are examined to rule out possible pathology.

Inspection of the eyes is another vital portion of the evaluation. This should occur under good lighting and with the patient in an erect posture.\(^\text{20}\) The patient is asked to perform several movements to help determine the origin of pathology. First, the patient is instructed to demonstrate movements on command looking from the examiner’s finger to the examiner’s nose. The physician is checking for overshoot, undershoot, or an increase in symptoms.\(^\text{11}\) Next, the examiner observes the patient’s ability to move smoothly, without consistent jerky movements by instructing the patient to follow his/her finger. Having the patient follow the examiner’s finger to his/her nose tests convergence. Normally, objects should come within three to four centimeters of the nose without blurring of vision.\(^\text{11}\) To test the integrity of the vestibulo-ocular reflex, doll’s head movements are performed.\(^\text{11}\) The patient fixates on an object while quickly moving the
head from side to side. This is repeated having the patient move his/her head up and
down. Normally, when the head is moved to the right, the eyes deviate to the left. Eye
dominance is also noted. The patient is instructed to make a triangle with his/her hands
and place an object approximately ten feet away so it appears in the middle of the shape;
ask the patient to close one eye, then the other. The preferred eye keeps the object inside
the shape. Following gaze stabilization exercises, the patient is observed for
spontaneous nystagmus or nystagmus at rest. Nystagmus present within 30° of the
position of primary gaze is always pathological, while nystagmus occurring at the
extreme limits of gaze is not.

To confirm a diagnosis of BPPV, the Dix-Hallpike maneuver (figure 3) is
performed. With the patient sitting on an exam table or plinth, the physician rapidly
moves the individual into a supine position, extending the head 45° below the table,
while rotating the head approximately 40° to one side. The patient’s eyes are observed
for nystagmus. This procedure is performed with the patient wearing magnified frenzel
glasses. The lenses prevent suppression of symptoms by fixation, which could lead to
inaccurate results. The position is maintained for one minute as the patient describes any
symptoms that arise. The patient is then returned to an upright position and symptoms
are allowed to dissipate. Once the symptoms have vanished, the patient is again rapidly
moved into the supine position with the head extended without any rotation. The position
is maintained for one minute as the patient describes any symptoms that occur. Once
again, the individual is returned to a sitting posture while the symptoms decrease in
intensity. Finally, the first maneuver is repeated, with the head extended and the head
rotated to the opposite side. Performing the Dix-Hallpike to both sides will determine which side is affected. Nystagmus related to BPPV is similar in direction in all cases.\(^5\) It will beat towards the undermost and affected ear in a rotatory fashion. The direction of the rotatory component is clockwise when the head is extended and turned towards the left; counterclockwise when extended and rotated towards the right.

If symptoms arise secondary to the Dix-Hallpike maneuver, the examiner will notice the following cardinal signs of BPPV.\(^5,6\) First, the onset of vertigo is delayed 1 to 40 seconds. In addition, nystagmus of equal latency as the vertigo will be present. Second, the symptoms are of short duration with a decline in intensity, disappearing within 60 seconds. Finally, after the maneuver is performed repeatedly, the symptoms decrease in intensity. This is known as fatigability. These observations are unique to BPPV, differentiating it from other peripheral vestibular lesions.

Bithermal caloric stimulation and electronystagmography are two additional diagnostic techniques for vestibular pathology.\(^21-25\) A diagnosis cannot be made with the results of these tests alone. The information provided, however, is beneficial in the decision making process. The caloric test is the most widely used test of VOR function. The test allows each labyrinth to be tested separately without complex equipment.\(^21\) The procedure attempts to disrupt the balance of the right and left vestibular organs and induce nystagmus. In normal individuals, the nystagmus is symmetrical, lasting for approximately two minutes. If vestibular disfunction is present, shorter lasting nystagmus is created from the affected side.\(^23\)
Before the examination can be performed, excess earwax must be removed, and pathology of the tympanic membrane should be ruled out.\textsuperscript{19} The patient is then positioned supine on the exam table. The head is elevated 30° to bring the horizontal canals into the vertical position.\textsuperscript{22} Next, each ear is irrigated separately with water 7° above and 7° below body temperature. A minimal interval of five minutes should be allowed between irrigations.

During this process, the patient is instructed to fixate on a target straight-ahead. As water enters the external auditory canal, a temperature gradient is established between the external auditory canal and the inner ear through conduction. The specific gravity is altered, resulting in endolymphatic flow in the semicircular canals. The direction of flow depends upon the temperature of water used for irrigation of the ears. A warm stimulation causes endolymph to rise because of its decreased density.\textsuperscript{22,23} This results in deviation of the cupula toward the utricle (ampullopetal flow). Horizontal nystagmus is induced with the fast beating component directed toward the stimulated ear. Cold water produces the opposite effect.\textsuperscript{22,23} The endolymph flows away from the utricle (ampullofugal flow) and the nystagmus is directed away from the stimulated ear. The mnemonic "COWS" (cold opposite, warm same) is helpful in remembering which direction the nystagmus should beat.

Electronystagmography (ENG) is another technique utilized in the evaluation of vestibular function.\textsuperscript{21,22,24,25} This procedure is widely accepted due to its ease of application and because eye movements can be performed with the eyes open or closed in complete darkness. This attribute prevents fixation without the use of frenzel glasses.
ENG provides a permanent record of the patient’s eye movements, determines whether the pathology is of peripheral or central origin, and which eye is affected.\textsuperscript{25,26} The principle of electronystagmography is based upon the potential difference between the cornea and retina.\textsuperscript{22} Recordings are produced with three electrodes; two active electrodes lie lateral to each eye, while a ground electrode is placed on the patient’s forehead. As the eye rotates toward one direction, the electrode on that side becomes more positive and the opposite electrode becomes less positive. The electrodes are attached to a pen recorder that moves according to each eye movement. The recommended ENG test battery includes evaluation of visual-ocular control and vestibular reflex function as well as recording function for pathologic nystagmus.\textsuperscript{22,25}

Three visual-ocular systems contribute to production of eye movements. Saccadic movements and smooth pursuit are determined by following the examiner’s finger. With a unilateral peripheral lesion, the saccades should be normal while transient contralateral impairment is expected with smooth pursuit.\textsuperscript{25} The optokinetic nystagmus test consists of vertical strip moved across the patient’s visual field in each direction at a constant velocity. This test enables identification of absent or asymmetrical optokinetic nystagmus. This should be normal in a patient presenting with labyrinth lesion.\textsuperscript{22}

The function of the vestibulo-ocular reflex is determined with the caloric test combined with ENG for more precise interpretation. Acutely, the vestibulo-ocular reflex will be decreased contralaterally with insult to the labyrinth. Finally, pathologic nystagmus is identified. It is recommended this be performed with the eyes open or closed in complete darkness to avoid fixation. Positional tests are performed in sitting
and in supine with the head moved in various directions. The Dix-Hallpike test is performed using ENG to allow more precise interpretation of the nystagmus.\textsuperscript{25} In BPPV, the nystagmus presents with a three to ten second latency before onset and dissipates within approximately 40 seconds. Both linear and torsional components will be observed by the clinician.\textsuperscript{22}

Once the physician has completed the medical examination and determined a diagnosis, the patient is referred to a physical therapist for vestibular rehabilitation. At the beginning of the evaluation, the diagnostic test result, the patient’s past medical history, and current medications are obtained.\textsuperscript{11,26} This information helps the therapist complete a thorough examination. The subjective history of the patient is imperative for the physical therapist to understand the vestibular pathology. The patient is asked to describe his/her attack in detail. The physical therapist will ask what precedes an episode and what movements or positions intensify symptoms. Knowledge of the frequency, duration, and intensity of symptoms assists the clinician in determining the appropriate rehabilitation protocol. Functional limitations due to the condition should also be noted. The objective portion of the evaluation includes assessment of active and passive range of motion followed by manual muscle testing of the upper and lower extremities. These should all be normal in a patient presenting with BPPV.\textsuperscript{26} Following, sensation, proprioception, and coordination deficits are ruled out. The gaze stabilization exercises performed with the physician are repeated. Static balance is assessed with the Romberg, sharpened Romberg, and single leg stance with the eyes open, then closed.\textsuperscript{11,26} Dynamic balance is evaluated by having the patient demonstrate hip, ankle, and step strategies.
These again should be normal in a patient with BPPV because symptoms only occur when moved into the provoking positions.\textsuperscript{11,26} A gait evaluation is also an important aspect of the evaluation.\textsuperscript{11,26} The patient is instructed to walk turning the head to the right and continue walking for 50 feet then turn to the left and walk an additional 50 feet. Then the patient is instructed to quickly turn his/her head from the right to the left repeatedly while ambulating. The patient is asked to repeat the test looking up and down. Gait deviations, loss of balance, and any increase in symptoms are noted. Once these tests are completed, the patient is instructed to move into various positions. Symptoms provoked are rated as mild, moderate, or severe. The Dix-Hallpike position is performed with the use of frenzel glasses to avoid fixation, which could obscure results. This movement will likely provoke the classic signs and symptoms of BPPV and a diagnosis can be confirmed.\textsuperscript{5,6,26}

The information obtained from the medical and physical therapy evaluations are utilized to determine the appropriate rehabilitation protocol. The following chapter will review several rehabilitative techniques utilized in physical therapy clinics.
CHAPTER V

TREATMENT OF BENIGN PAROXYSMAL POSITIONAL VERTIGO

The first exercise regime designed to treat BPPV was developed in 1946, more than 20 years after the condition was initially described.\textsuperscript{6,17} Cawthorne and Cooksey noted that individuals who performed rapid head movements following a unilateral vestibular insult progressed quicker than those who did not perform the movements. There are many treatment options available to those who suffer from BPPV including exercise therapy, anti-vertigo medications, and surgical intervention.\textsuperscript{5,6,27}

Vestibular rehabilitation is an alternative form of treatment involving specific exercises designed to decrease dizziness, improve balance, and increase general activity levels.\textsuperscript{17} Successful resolution of symptoms is dependent on determining what positional changes provoke the patient’s vertigo. Norre\textsuperscript{27} developed 19 exercises that typically illicit vertigo and nystagmus associated with BPPV. These maneuvers were used as a test battery as well as a method of rehabilitation. As the patient performs each of the 19 positional changes, the clinician records whether vertigo and/or nystagmus occur. The exercise protocol is fabricated utilizing the maneuvers that elicited both vertigo and nystagmus. Thus, treatment is tailored specific for each individual.\textsuperscript{10}

Tangerman and Wheeler\textsuperscript{10} described three phases of habituation training. The first phase consists of having the patient move repeatedly into the Dix-Hallpike position.
The following two phases include a variety of balance exercises that incorporate eye and head movements. These exercises begin in supine and then progress to sitting. Finally, they are performed during functional activities. The patient is instructed to perform the exercises five times successively, two to three times per day until symptoms dissipate. The balance activities, often excluded from other treatment procedures, are beneficial for those presenting with postural instability.

The goal of habituation training is that the individual will be able to assume the provoking positions symptom free. The theory behind this mechanism of rehabilitation is adaptation. Through repeated exposure to the stimulus, the CNS will attenuate the responses.

Brandt and Daroff disagree with the central adaptation theory behind habituation training because many patients recover abruptly. Instead, they focused on the otolithic matter believed to be the source of symptoms. Brandt-Daroff exercises (figure 4) were developed in theory that vertigo and nystagmus associated with BPPV are due to cupulolithiasis. Through rapid head movements, the adhered otolithic particles will be dislodged from the cupula of the posterior canal and redistributed, eliminating symptoms.

The maneuver consists of three positions. Beginning in supine, the patient is rapidly moved into the provoking sidelying position with the lateral occiput on the plinth to ensure stimulation of the posterior canal. The individual remains in this position until symptoms subside, then returns to a sitting posture. Moving to sitting will frequently result in vertigo of lesser intensity and shorter duration. This phenomenon is known as
Figure 4. Brandt- Daroff habituation exercises. Adapted from Brandt, T, Daroff, RB: Physical therapy for benign paroxysmal positional vertigo. Arch Otolaryngol. 1980; 106: 484.
the "rebound effect." The patient remains in the upright position for 30 seconds and then is quickly moved into the mirror image position for an additional 30 seconds before returning to sitting. At each session, this sequence is performed until symptoms dissipate. The patient also carries out the maneuver every three waking hours until two consecutive vertigo-free days are achieved.

Semont\(^{10}\) developed a single-treatment approach based on cupulolithiasis and canalithiasis (figure 5). The Semont or Liberatory maneuver loosens debris from the cupula of the posterior canal. It may also cause debris to move through the posterior canal, into the common crus, and into the vestibule, relieving symptoms.

Once the affected side is identified, the patient is quickly moved into the provoking sidelying position with the head in the plane of the posterior canal. After two to three minutes, the patient’s head and neck are grasped by the therapist with both hands and the patient is rapidly moved through the sitting position and down into the opposite sidelying position. The face should be angled toward the plinth. The nystagmus is now directed towards the upper ear. The patient remains in this position for five minutes before slowly returning to sitting. Following treatment, the patient is instructed to avoid the provoking position for one week and maintain the head in a vertical position for 48 hours.\(^{6,10,28}\) The individual is told not to bend his/ her head forward or backward. No form of exercise is permitted. The patient may not go to the hairdresser or dentist. While sleeping, pillows are utilized to maintain the body at a 30° to 45° angle. Something should be placed at the end of the bed to prevent sliding down during the night.
Another single-treatment approach was described by Eply. Based on canalithiasis, the canalith repositioning procedure attempts to move free floating debris in the posterior canal through the common crus and into the vestibule, relieving symptoms.

The procedure is carried out as follows. With the head turned $45^\circ$ toward the affected side, the patient is rapidly moved into the Dix-Hallpike position. After three to four minutes, the patient’s head is slowly rotated through extension and turned into the opposite Dix-Hallpike position. Next, the patient is rolled onto his/her side with the head downwardly rotated $45^\circ$. The patient remains in this position for an additional three to four minutes then slowly returns to sitting. Vibration can be applied to the mastoid process of the affected side throughout the maneuver to improve results. Following treatment, the individual is fitted with a cervical collar and instructed to remain in an upright position for 48 hours, avoiding bending forward, looking up or down with the head, and lying down.

As an exercise program is implemented, the patient should be aware of the following symptoms. If they do occur, treatment should be stopped and the physician should be notified.

1. A sudden change or fluctuation in hearing.
2. The onset of pressure or feelings of fullness in your ears, to the point of pain.
3. Ringing in the ears.
4. Fluid discharge from the ears.
5. Pain in the neck or back associated with performing the exercises.
Before a treatment protocol is selected, several factors must be considered. Elderly patients tend to move cautiously due to conditions such as arthritis and thus would be a less tolerant of the liberatory maneuver compared to other protocols. Second, the liberatory and Semont maneuvers may not be appropriate for patients who will find it difficult to remain in an upright position for 48 hours such as parents with small children or those who are required to perform bending activities at work. The success of habituation training is dependent on compliance. Single treatment approaches prove to be more beneficial to those who, for various reasons, cannot follow a daily home exercise program.

There are various physical therapy approaches to treating BPPV. For the rare population that does not respond, surgical intervention is considered. The posterior ampullary nerve supplies the ampulla of the peripheral vestibular system. After this nerve is cut, the debilitating symptoms of BPPV are abolished because the CNS no longer receives the incorrect impulses concerning head movement. A possible complication of this procedure is sensorineural hearing loss.

A third approach is drug therapy. Anti-vertigo medications reduce the nausea and vomiting experienced by many, however have not been proven to be effective in the treatment of BPPV (Gacek, 1984). Medications used for inner ear disorders have potential sedating side effects that limit the patient’s ability to carry out activities of daily living.

In the last few years, significant advances have been made in the rehabilitation of those suffering from BPPV. The exercises have become more sophisticated, reflecting
an increased knowledge of the vestibular system and the mechanisms of recovery following an insult. Physical therapy should be the first approach taken. If this method of treatment is unsuccessful, then more invasive measures are instigated.⁵
CHAPTER VI

CONCLUSION

Traditional treatment methods for vestibular pathology have relied on medication designed to suppress vestibular function. More recently, physical therapy has emerged as a successful alternative for those with motion intolerance.

In a study of 67 patients with BPPV of two days to eight months duration, Brandt and Daroff reported that 98% of the patients were symptom free within three to fourteen days. Ninety percent of 60 patients treated with the Semont Maneuver were asymptomatic after a single treatment. Habituation training has also proved to be a successful method of rehabilitation. Of the 28 subject included in the study, 32% were free of vertigo within one week. The remaining patients, however, did report a decrease in symptoms. By six weeks, all subjects showed no evidence of vertigo.

Vestibular rehabilitation can be a terrifying experience for many people. The treatment methods will bring on symptoms during each session. Patients will often scream, clutching to the clinician. It is important to explain to the patient that this is necessary, and their symptoms will soon dissipate. The fear and constant feelings of dizziness and nausea may lead to noncompliance with a home exercise program. Single treatment protocols may be more beneficial to those who cannot tolerate repeated head movements. Another option to assure successful treatment is the administration of anti-
vertigo medications prior to treatment sessions to reduce the severity of symptoms. The patient will then be more willing to participate in the exercises.

Those with BPPV often have secondary symptoms that cannot go unnoticed. These include decreased strength, loss of range of motion, balance disturbances, and increased tension in the cervical and shoulder region leading to muscle fatigue and chronic headaches. Once the vertigo is resolved, therapy should be directed toward increasing strength, range of motion, and coordination and balance to allow full return to daily and social activities.
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