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Diagnostic and Rehabilitation Techniques for Benign Paroxysmal Positional Vertigo

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DIAGNOSTIC AND REHABILITATION TECHNIQUES FOR
BENIGN PAROXYSMAL POSITIONAL VERTIGO

by

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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

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1997
This Independent Study, submitted by Aaron E. Reinhardt 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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Title               Diagnostic and Rehabilitation Techniques for 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 that results in vertigo, nystagmus, and often nausea and vomiting. BPPV has been treated in the past by a variety of rehabilitation approaches. Today, physical therapists are becoming an integral part of the rehabilitation of these patients. The purpose of this study is to review the diagnostic and treatment techniques currently being used to rehabilitate patients diagnosed with BPPV. This will be accomplished by thoroughly examining the anatomical and physiological processes involved in BPPV. Once these are discussed, the diagnostic process will be explored. Finally, all current therapeutic techniques will be discussed including their efficacy. An in-depth discussion on how this author feels the problem of BPPV is being managed and recommendations for future research and treatment options will conclude this independent study.
CHAPTER I
INTRODUCTION

Dizziness is one of the most common complaints for which medical help is sought.\textsuperscript{1} Annually, dizziness accounts for two percent of all office visits.\textsuperscript{2} Although a large number of patients with dizziness are seen each year, it continues to be a difficult symptom to evaluate. This is mainly due to the numerous disorders that can cause dizziness. Such medical specialties as cardiology, psychiatry, neurology, otology, and ophthalmology can be involved in evaluating and treating this problem. The complaint of dizziness can be divided into four general categories.\textsuperscript{1,2}

First, a patient may describe an "illusion of movement" which may refer to either the patient himself moving or the surroundings moving around him. This is referred to as vertigo and is a product of improper functioning of the vestibular system. Such words as spinning, rotating, and swaying are used to describe the feeling. In the case of vertigo, there is often accompanying nystagmus, nausea, and vomiting.

Secondly, syncope or presyncope refer to a sense of "impending loss of consciousness or fainting." This is a result of a decreased blood flow to the brain and causes a shortage of oxygen and glucose. Third, the term
disequilibrium describes a sense of unsteadiness or imbalance. This occurs without vertigo and is caused by neurologic disorders. Such problems as sensory deficits, cerebellar dysfunctions, and drug intoxications are categorized in this grouping.

Finally, the fourth category includes "ill-defined dizziness." This can result from psychiatric disorders and lead to such underlying problems as hyperventilation, anxiety neurosis, and depression. This is described as a light-headedness or giddiness that is not a result of the three previously mentioned categories. It should be noted that with any type of dizziness there may be a resultant loss of balance or trouble with walking.

Of the four categories just described, it is vertigo that is of most relevance to physical therapists. This is due to the ability of therapists to manage the symptoms of this problem through specific exercises as an alternative to surgery or the use of medications.\textsuperscript{3-5} Vertigo can be caused by such pathologies as vestibular neuronitis, trauma to the inner ear, positioning of the head, Meniere's disease, labyrinthitis, and brainstem ischemia.\textsuperscript{1} The positioning of the head is the most common cause of vertigo and is the most treatable by therapists. The specific name for this problem is benign paroxysmal positional vertigo (BPPV).

BPPV can be a crippling disorder of the inner ear that is brought about when the head moves rapidly in certain directions or is placed in a certain position.\textsuperscript{6} Examples of this would be looking over your shoulder to back up a car, rolling over in bed, or even simply turning your head quickly.
By defining the terms in benign paroxysmal positional vertigo, one can gain a better understanding of the disorder. Benign means that the problem is not progressive or recurrent but is self-limiting. Paroxysmal refers to a sudden attack or recurrence of the symptoms of vertigo. The term positional describes the actions that usually precede the occurrence of symptoms. The term vertigo has already been defined as a spinning sensation felt by the person. By combining these four definitions, one can contrive a rather good explanation of the disorder: By positioning the head, a person may have a sudden, non-progressive attack of a spinning sensation that will soon dissipate.

In diagnosing a patient with BPPV, the examiner needs to be familiar with the concurrent nystagmus that is always present. Nystagmus is defined as involuntary movement of the eyes in a constant, rotatory pattern. This is due to the sending of incorrect messages from the peripheral vestibular system to the eye musculature responsible for movement. The pattern of nystagmus is similar in direction and pattern in all cases of BPPV and is one of the most important aspects of its diagnosis. A more detailed description of this process will be covered in the next chapter.

In 1921, Barany initially described BPPV and its attacks of vertigo and nystagmus. He discovered that if the head is rapidly extended and laterally tilted towards the affected ear, the symptoms would appear. Barany’s idea was expanded in 1952 by Hallpike and Dix. They are responsible for producing the Dix-Hallpike maneuver which is a provocative test used to diagnose BPPV.
The Dix-Hallpike maneuver is performed with the patient sitting on a table or bed. The therapist rapidly moves the patient to a supine position allowing the head to extend approximately 30° below the level of the table. As the head is extending, the therapist rotates the head 30° to 45° to one side. The patient is then observed for vertigo and nystagmus. This procedure is then repeated to the other side to help determine which ear is causing the symptoms. This test is performed with the patient wearing Frenzel's glasses which help the clinician view the nystagmus more clearly. The glasses contain lenses of higher magnification and have an internal light source that shines into the eyes. The light keeps the eyes from fixating which would inhibit the observable nystagmus.

Along with the Dix-Hallpike maneuver, there are two other diagnostic tests that can be performed to measure nystagmus. Electronystagmography (ENG) is a process that uses electrodes placed around the eyes to record eye movements. The resultant movements are recorded on paper in such a way that the results can be saved for reference for future readings or to be compared with other patients.

The bithermal caloric test is another method of inducing the symptoms of vertigo. This test is performed by irrigating the ear with water of varying temperatures. By doing so, one is able to simulate irregular vestibular functioning and induce nystagmus. This test can be used in conjunction with ENG to obtain more precise results. Caloric stimulation has the advantage of
testing each ear separately without the need for complex equipment. These diagnostic tests will be examined in greater detail in Chapter V.

Certain characteristics of the observable symptoms help distinguish BPPV, a peripheral vestibular system lesion, from a central nervous system lesion also causing vertigo and nystagmus. The length of time it takes for the vertigo and nystagmus to appear in BPPV is between 1 and 40 seconds with an average of 7.8 seconds. This delay is referred to as latency. The duration of the symptoms is less than one minute, reducing gradually within 10 to 40 seconds while maintaining the head position. As the maneuver is repeated, their symptoms become less and less severe, eventually becoming absent. This process of “fatigability” is an important aspect of the therapy used for patients with BPPV. The direction and quality of nystagmus can be beneficial in locating the lesion. Nystagmus is primarily observed with the fast phase beating towards the undermost ear. Finally, nystagmus is presented in a clockwise rotation with lesions of the left ear and counterclockwise in right ear lesions.

In contrast, lesions of central peripheral system origin exhibit different characteristics. There is no latent period before the onset of nystagmus. The duration of the attacks is always longer than 30 seconds and may persist for periods of time. There is more than one head position that elicits the attacks as compared to the single position in BPPV. With repetitive testing, the symptoms do not fatigue. Finally, the direction of the nystagmus is inconsistent from one head position to another.
Once a correct diagnosis has been made, one needs to establish a course of rehabilitation. In the case of BPPV, the types of therapies available have been growing due to the increased knowledge of the disorder itself. Such procedures as exercise therapy, particle repositioning, and vestibular habituation training are commonly used by therapists today. These have been used as alternatives to surgery and medications. Each of these treatment procedures will be discussed in Chapter V.

The purpose of this study is to gain an understanding of the diagnostic and treatment techniques that are currently being used to rehabilitate a patient with benign paroxysmal positional vertigo. In the following chapters, the disorder of BPPV will be examined in great detail. The anatomy and physiology involved with the vestibular system will be discussed first. The focus will then shift to the pathophysiology and the resultant causes of BPPV. The fourth chapter will look at the diagnostic procedures used today by therapists and clinicians. The techniques and procedures used to treat BPPV will then be covered in the fifth chapter. An in-depth discussion on how this author feels the problem of BPPV is being managed and recommendations for future research and treatment options will conclude the study.
CHAPTER II

ANATOMY AND PHYSIOLOGY OF THE INNER EAR

The peripheral vestibular system is the area of the body that is associated with benign paroxysmal positional vertigo (BPPV).\(^8\) To fully understand the underlying mechanics of BPPV, one needs to be familiar with the anatomy involved in this area. The inner ear, which is located in the petrous portion of the temporal bone, houses the peripheral vestibular system.\(^{13-15}\) This system includes the bony and membranous labyrinths and the hair cells which are responsible for sensing motion.

The bony labyrinth contains perilymphatic fluid which helps suspend the membranous labyrinth (Figure 1). The membranous labyrinth in turn is filled with endolymphatic fluid. It is this fluid that is partially responsible for sensing movement of the head. The bony labyrinth consists of three semicircular canals and a small chamber called a vestibule that is formed at the base of the three canals. One end of each semicircular canal is enlarged forming an ampulla.

The membranous labyrinth, lying within the bony labyrinth, contains five sensory organs which help monitor the forces that are associated with linear and angular motion of the head. These are the receptor areas called crista located in the ampulla of each of the three semicircular canals, and two otolith organs.
named the saccule and utricle. The saccule and utricle both contain receptors known as the macula. To be more specific, the saccule and utricle sense linear acceleration while the crista of the three canals help sense angular acceleration of the head.

Hair cells are found in both the ampulla and the otolithic organs. The hair cells in the ampulla are found resting on the crista, while in the saccule and utricle they are located on the floors and walls respectively in the macula. The hair cells main function is to convert movements of the head into electrical impulses that are sent to the brain via afferent neurons. Each hair cell is innervated separately. These hair cells interpret the movement of the head by sensing changes in endolymphatic flow.

The protruding stereocilia do not lie directly in the endolymph. For example, in the semicircular canals, a gelatinous layer called a cupula covers each crista and is used as a connection between the endolymphatic flow and the resulting movement sensed by the hair cells. The change in flow causes the cupula to be moved or “deflected” and this in turn sends a signal to the hair cells to increase the afferent nerve firing rate. In the saccule and utricle, the macula have similar gelatinous membranes overlying them termed otolithic membranes in which are embedded calcium carbonate crystals. These crystals will be referred to later when the causes of BPPV are discussed.

The anatomical structure of the hair cell is important to understand how they actually work (Figure 2). The cell body, which is flask or globular-shaped,
has two different types of cilia that protrude from it. The stereocilia are thin and nonmobile while the kinocilium is much thicker and is the tallest. In each hair cell, the stereocilia number between 50 to 110 and are of varying height while there is only one kinocilium. These stereocilia are arranged in ascending height in a step-like pattern leading up to the singular kinocilium. It is this arrangement that allows the hair cells to interpret the amount of movement occurring.

As previously mentioned, the cupula and the otolithic membrane are deflected by the flow of endolymph. If the deflection of the stereocilia is towards the kinocilium, the resting membrane potential is decreased (depolarization or excitation) thus causing an increase in hair cell stimulation. Conversely, if the hairs are deflected away from the kinocilium, the impulses are inhibited and the membrane potential is increased or hyperpolarized. One needs to keep in mind that the linear acceleration will be sensed by the otolithic membrane while the cupula is sensitive to angular acceleration.

The impulses that are received by the hair cells are sent to the CNS via the superior division of the vestibular nerve (cranial nerve VIII). The first order neurons terminate in one of the four vestibular nuclei. The neurons from the utricle and saccule end in the lateral and inferior nuclei while the afferents from the crista of the semicircular canals proceed to the superior and medial nuclei. From this point, they travel to one of four areas, each for different functions.

The fibers from the lateral nucleus descend into the vestibulospinal tract and end in the ventral column of the spinal cord. From this point, they proceed
to innervate the necessary skeletal muscles of the trunk and extremities that are needed to maintain balance. The fibers from the inferior nucleus descend into the medial longitudinal fasciculus (MLF). They then enter the nucleus of the accessory nerve and the ventral columns of the spinal cord. It is from this point that the neck musculature is innervated to maintain the position of the head.

The fibers from the medial nucleus are distributed to the autonomic centers of the brainstem and are responsible for such responses as vomiting and nausea which may occur if the vestibular system is overstimulated. Finally, the fibers form the superior nucleus enter the MLF and then terminate in the cells of cranial nerves III, IV, and VI. These three cranial nerves innervate musculature surrounding the eyes and are responsible for moving the eye. Control of coordinated eye movements is one of the important functions of the vestibular system. This is also an important contributing factor in the development of nystagmus.

When a person rotates his/her head from one side to the other, the eyes follow along and maintain a clear field of vision. The vestibulo-ocular reflex (VOR)\textsuperscript{13,14,17} is the reflex acct that makes this possible. The VOR works as follows: As the head is rotated to the right, the eyes will slowly move to the left as if it was fixed on a certain point in space. There comes a point where the eyes can no longer focus on that spot because the head has turned too far to the right and the eyes have moved as far as possible to the left. Once this limit of motion is reached, the eyes quickly turn to the right to catch up with the head
and once again focuses on a spot. This procedure is repeated over and over until the head comes to rest. The opposite motions occur if the head is turned to the left. The terms fast and slow phase, which will be introduced later, are derived from this reflex.

The horizontal semicircular canal is responsible for this reflex in the case of head rotation in the transverse plane. (In cases of BPPV, it is the posterior semicircular canal that is responsible due to the angle of head movements that precede the attacks.) As the head rotates to the right, the endolymph in the canal is shifted due to inertia. This change in flow causes the left sided crista to hyperpolarize and the right sided crista to depolarize. This in turn causes impulses to travel to the MLF and innervate the eye musculature. In this case, the cranial nerves III and VI are involved. The lateral rectus muscle of the right eye and the medial rectus muscle of the left eye cause the rotation to occur.

When all of these components are working in unison, our ability to view objects clearly while moving our head is excellent. However, if something goes awry, our body reacts in adverse ways. In the case of BPPV, the brain is sent incorrect messages saying that the head is moving when it is actually stationary. This leads to attacks of vertigo and nystagmus. The pathological processes that are involved in BPPV will be discussed in the following chapter.
CHAPTER III

PATHOPHYSIOLOGY OF THE VESTIBULAR SYSTEM

The mechanism of balance is dependent upon the integration of the visual, proprioceptive, and vestibular systems. The vestibular end organs, the semicircular canals, the utricle, and the saccule, communicate with the cerebellum and the vestibular nuclei by transforming changes in linear and angular acceleration along with changes in the force of gravity into biological impulses. These impulses allow the brain to monitor the position and movement of the head. The brain then uses this information to produce motor reflexes in the musculature of the eyes, limbs, and trunk. These reflexes are essential in maintaining our equilibrium and producing movement.

The vestibular system should be looked at as being comprised of two halves. In a normal resting state, the cerebellum and vestibular nuclei will receive symmetrical impulses from both the right and left vestibular end organs. When the head moves, the impulses will be altered from its state of equilibrium. The rate of firing will increase in one side of the vestibular system and decrease in the other. This creates a state of imbalance and a subsequent sensation of head movement which will initiate the stimulation of the vestibulo-ocular reflex.
(VOR) which is responsible for the corrective eye movements that allow a person to maintain clear vision while the head is rotating.

In the case of benign paroxysmal positional vertigo (BPPV), one ear is affected and an imbalance is created when the head is moved into certain positions. While the head remains at rest in the provoking position, the brain is being sent impulses that are incorrectly interpreted as head movement. This feeling of constant motion will bring about the sensation of vertigo. In addition, the VOR is activated and the eyes will be moved in the typical rotatory pattern of nystagmus.\textsuperscript{19,21} This explanation is not sufficient to fully understand the causes of BPPV. It is important to look at the underlying pathophysiological explanation of what is responsible for the unilateral lesions that cause the imbalance of signals sent to the brain.

When BPPV was originally described in 1921 by Barany,\textsuperscript{9} he felt that the problem originated in the otolithic organs. This idea was supported by Dix and Hallpike\textsuperscript{10} in 1952. Due to the increasing amount of physiological experience gained by the clinicians, the consensus was that BPPV was a canal-driven disorder but were still unclear on the specific cause.

In 1969, Schuknecht\textsuperscript{22} provided what is considered the classic explanation of the cause of BPPV. While examining the temporal bones of two patients known to be diagnosed with BPPV, he discovered basophilic deposits attached to the cupula of the posterior semicircular canal. The basophilic "particles" were believed to be detached from the otoconial layer and gravitated
into the semicircular canal via the endolymph. The condition was termed "cupulolithiasis" by Schuknecht due to the combination of stones ("lithos") and the cupula. The positioning of the canal is situated inferiorly to the utricle with the head in an upright position and becomes a receptacle for the detached particles which in turn attach to the cupula which is embedded in the endolymph.

The endolymph and the cupula normally have the same specific gravity. The floating basophilic particles create a specific gravity differential between the endolymph and the cupula. This differential allows the particles to be free-floating in the endolymph. The particles are believed to act in one of two ways. First, due to the differential created, the posterior semicircular canal becomes oversensitive to changes in gravity. Thus, when the head is extended into the provoking position, the posterior canal is placed in the specific plane of stimulation and vertigo and nystagmus occur. This theory supports the idea that the particles remain free-floating within the canal.

Secondly, some authors believe that the particles themselves come in contact with the cupula and, due to the flow of the endolymph, cause the cupula to be deflectected. This so-called ampullofugal deflection causes a "burst" of nystagmus to occur along with the vertigo. It is not known if these two processes occur separately or in conjunction.

The particles that are responsible for this specific gravity differential and deflection are believed to be comprised of either calcium carbonate or a basophilic material. These particles originate from a degenerating
otocotonal layer of the utricular macula. The degeneration is caused by a number of factors. The most prevalent cause in people under the age of 50 is head injury or trauma. Other causes include infection, surgery, and degeneration with aging.

Further support for the theory of cupulolithiasis comes from the research performed on the posterior semicircular canal. There are two main factors that back up the suggestion that the posterior canal is critical in the pathology involved in BPPV. First, the crista of the posterior canal, when stimulated, cause a contraction of the ipsilateral superior oblique and the contralateral inferior rectus muscles. These are responsible for the rotatory and vertical movements of the eyes during nystagmus. Secondly, the posterior ampullary nerve when severed has been shown to eliminate unilateral BPPV.

Although there has been some agreement in the recent past on the underlying cause of BPPV, there continues to be discussion on some issues. One in particular is the idea raised by Brandt in 1990. He believes that BPPV is not a positional disorder as the name states, but rather a positioning disorder. Due to the fact that the head needs to be moved into the provoking position rapidly, as Dix and Hallpike suggest, the vertigo and nystagmus are caused in part by the quick change in position. This rapid movement causes the ampullofugal deflection of the cupula to occur just as is explained in the cupulolithiasis theory. The intensity of the symptoms is dependent upon the velocity of head movement into the precipitating position. Finally, he adds that
the symptoms of BPPV can be avoided altogether if the provoking position is assumed too slowly. It has been shown that attacks rarely occur if the time elapsed is more than six seconds.

The rapid positioning that Brandt discusses can also be related to the diagnostic criteria that separates BPPV from a central nervous system lesion. First, the delayed onset can be explained by the time needed for the endolymph to get moving. A few seconds are required for the endolymph to start flowing after the head moves. It is believed that the severity of symptoms are dictated by the amount that the cupula is deflected by the endolymph. The limited duration of the attack can be attributed to the cupula returning to its original position after it is deflected. The idea of fatigability is believed to be associated to the particles being dispersed from the cupula through repeated head movements. Finally, the symptoms may recur at a later time once the particles have had a chance to resettle into the posterior semicircular canal.

It is from these clinical findings that the diagnosis of BPPV is based. Such procedures as caloric testing, electronystagmography, and the Dix-Hallpike maneuver are presently being used by clinicians in order to help diagnose this problem. One must keep in mind that the symptoms encountered by the patient with BPPV are very uncomfortable and diagnosing the problem will take a great deal of cooperation by the patient who will often be reluctant to bring on the symptoms. Therefore, it is important for the patient and clinician to establish a good and trusting relationship.
Now that the pathological processes are fully understood, the following chapter will focus on how physicians and clinicians use their information to diagnose BPPV. By focusing on the patient’s history, objective findings, and special tests, they are able to correctly differentiate BPPV from other disorders of the vestibular system.
CHAPTER IV
DIAGNOSIS

The evaluation of a patient with benign paroxysmal positional vertigo (BPPV) is an important step in arriving at the proper method of rehabilitation. The symptoms that the patient exhibits will help determine the treatment protocol. The vestibular system evaluation can be divided into two parts. First, the patient is examined by a physician who, by using certain diagnostic techniques and criteria, will determine if the peripheral vestibular system is compromised and more specifically if BPPV is suspected. The physician may then decide to refer the patient to physical therapy for rehabilitation.

At this point, the physical therapist implements the second portion of the assessment which is much more functional in nature. The physical therapist's main objective is to construct a plan of rehabilitation based on the patient's history and symptoms. The two aspects of the vestibular evaluation will be examined in this chapter. The assessment process used by the physician will be looked at first followed by the techniques that allow physical therapists to determine the appropriate method of rehabilitation.

During the initial evaluation, the patient's history is the first area covered. The patient is first asked to describe the dizziness that
he/she is feeling. It is important to have patients describe the symptoms they are experiencing in their own words. The dizziness that the patients describe can then be placed into one of four categories: 1) vertigo, 2) syncope or lightheadedness, 3) disequilibrium or unsteadiness, or 4) dizziness. The category of vertigo would be appropriate for someone describing an illusion of movement of their surroundings around them or of themselves rotating. This category is usually related to a problem with the peripheral vestibular system.\textsuperscript{28}

Once the overall sensation of dizziness is described, the patient is then encouraged to describe his/her initial episode or attack in great detail.\textsuperscript{28,30} This is a very important area of the evaluation. Listening to the symptoms that the patient describes will assist the clinician in diagnosing the problem. Such areas as time course, precipitating factors, related symptoms, and predisposing factors should be covered.\textsuperscript{28,30} The vertigo associated with BPPV and other acute forms due to labyrinthine disorders are very short in duration. Peripheral lesions occur in brief episodes that are usually abrupt in onset followed by decreasing intensity as the symptoms disappear. Vertigo of central origin tends to be of longer duration and there are no signs of fatigability.\textsuperscript{1} Some forms of vertigo such as those caused by viral infection last for several days.\textsuperscript{29}

The conditions preceding the attack are very important. Vertigo that is the result of peripheral vestibular lesions is often elicited by rapid movement of the head. Therefore, a patient who describes rolling over in bed or looking over his/her shoulder to back up the car immediately prior to the attack would most
likely be suffering from a peripheral vestibular lesion. Meniere's disease, on the other hand, is often associated with hearing loss, earache, and an increase in tinnitus.\textsuperscript{11,30} Other precipitating factors can include coughing or sneezing which lead to changes in middle ear pressure in a condition called perilymph fistula. Also, loud noises can cause dizziness in individuals with endolymphatic hydrops.

Finally, there are other factors that can help the physician in his/her evaluation.\textsuperscript{28-30} Symptoms such as nausea and vomiting often accompany dizziness that is of vestibular origin. These symptoms are not usually associated with other causes of dizziness. Along with these symptoms, the physician needs to examine the patient's overall medical history to see if there are any predisposing factors. Many severe systemic disorders can have dizziness as a resulting symptom. These symptoms can mimic those found in BPPV and this must be recognized by the physician. In addition to these systematic disorders, such events as head injury, surgery, and ear infection can lead to vertigo.\textsuperscript{22} The patient must be asked about any medications that he/she has been taking. Many drugs can produce dizziness in a patient with an intact and healthy vestibular system. Once the history is complete, the physician then begins the general examination.

Inspection of the ears, nose, and throat must be included to rule out any possible pathology.\textsuperscript{28-30} Of these three areas, the ears are the most significant and will yield the most information. In the ear, the external canal and tympanic
membrane are examined. The canals are checked for excess cerumen or earwax. A build up of cerumen may lead to canal obstruction which can cause hearing loss and may lead to a sense of imbalance or dizziness. The integrity of the tympanic membrane is viewed through an otoscope. Most patients with vestibular disorders often have an intact membrane. Assessment of the nose and throat are not a necessity for the diagnosis of BPPV but are usually included in a physician’s general examination.

The final area to be covered is the eyes. Examination of the eyes is an important step in discovering the underlying cause of the vertigo. The eyes should be examined in a well-lit area with the patient sitting in an upright position. Dix suggests the following movements be performed to obtain important information. First, the patient is asked to perform movements on command. This is done by having the patient follow the examiner’s finger. Second, convergence is tested by having the patient follow the examiner’s finger towards the patient’s face. Next, doll’s head movements are performed. The patient fixates on a certain object and then rotates the head trying to maintain view of the object. This can also be performed with head extension and flexion.

Finally, the eyes are observed for spontaneous nystagmus. If nystagmus is noticed within the range of 30° to the left or right of primary gaze in the vertical plane, it should be considered pathological. Nystagmus that is present beyond the 60° range should not be considered pathological. Normal
individuals may experience nystagmus at the extreme ranges along the vertical plane.

In the case of a patient with BPPV, these four movements would not reveal any significant information. This is due to the positioning of the patient and the lack of rapid head movements used in the tests. In order to diagnose the patient with suspected BPPV, one must use the Dix-Hallpike maneuver.\textsuperscript{10}

With the patient sitting on a table or bed, close to one end, the therapist rapidly moves the patient to a supine position allowing the head to extend approximately 45° below the level of the table (Figure 3). As the head is extending, the therapist rotates the head 45° to one side. The patient is then observed for vertigo and nystagmus. The patient is asked to keep his/her eyes open for as long as possible to allow for optimal observation. The nystagmus is best seen with the patient wearing Frenzel's glasses which magnify the view of the eyes and help eliminate fixation of the eyes.

The position is held for one minute and the patient is asked to describe any symptoms that come about. After the minute has passed, the patient is returned to the upright position and the symptoms are allowed to dissipate. Once the patient is symptom free, he/she is returned to the supine position with the head extended in a straight back position without rotation. This is again held for one minute and the patient is observed for symptoms. The patient again returns to sitting to allow the symptoms, if any, to clear. Finally, the procedure is
followed one last time with the patient supine and head extended and rotated to the opposite side as the first maneuver.

If the test does bring on the symptoms, the examiner will notice the following in a patient with BPPV. First, the onset of the vertigo or nystagmus is delayed on the average of 7.8 seconds with the longest latency being 40 seconds.6 Second, the symptoms are of short duration, usually between five and ten seconds and almost always less than one minute.6,22 Third, upon repeated testing, the attacks become less and less severe. Often the symptoms cannot be provoked after the third or fourth consecutive positionings.22 These findings can be used to help differentiate BPPV from vertigo caused by other vestibular disorders.

The symptoms should present themselves when the affected ear is pointed downward in the provoking position. The nystagmus that is observed has the fast phase beating towards the undermost ear in a rotatory fashion.22 When the head is held in the right head hanging position, the rotation is counterclockwise in direction. The rotation will occur in the opposite direction if the head is extended and rotated to the left. If the Hallpike positioning does bring on an attack, the patient will often show great distress by closing his/her eyes, yelling out, and clutching at the clinician as he/she attempts to sit up.6,10,22 If this occurs, the patient must be reassured that the symptoms will soon subside and in order to get better, he/she must go through some distress.
Along with the Dix-Hallpike maneuver, there are two additional ways to diagnose a patient's nystagmus. Caloric stimulation and electronystagmography are currently being used by clinicians in order to gain more precise information about the nystagmus presented by their patients. First, the caloric test is a procedure that allows the clinician to test each ear separately.\textsuperscript{1,11,21,28} Normal functioning of the vestibular system occurs when both the left and right vestibular organs receive identical stimuli.\textsuperscript{28} The caloric test attempts to offset this balance and create nystagmus. In normal individuals, the elicited nystagmus should be equal as both sides are tested. If a lesion is present in one ear, the clinician should see unequal showings of nystagmus with the compromised side showing decreased results.\textsuperscript{1,11,21,28}

The test is simple and can be performed without sophisticated and expensive equipment. The patient is first checked for excess earwax and the integrity of the tympanic membrane is examined.\textsuperscript{28} Once the patient is cleared to proceed, he/she is positioned on a bed or table in a supine position. The head is inclined forward 30° in order to bring the horizontal semicircular canals into the vertical plane.\textsuperscript{1,11,21,28} Once this position is achieved, the clinician irrigates each ear separately with water that is either seven degrees Celsius above or below body temperature. The water flowing into the external auditory meatus creates a temperature gradient between the outer ear and the endolymph in the semicircular canal. Through conduction, the temperature of the endolymph changes and begins to circulate due to an alteration in its specific gravity.
The direction of flow depends on the type of water used to irrigate the ear. Warm water causes the endolymph to rise and a resulting cupular deflection towards the utricle (ampullopetal flow) occurs. This deflection is much like that occurring during head movement in the vertical axis. The movement of the cupula produces horizontal nystagmus with the fast component beating towards the same ear. Cold water, on the other hand, produces the opposite effect. The endolymph becomes heavier and sinks causing the cupula to be deflected away from the utricle (utriculfugal flow). The nystagmus created by the cold water beats towards the contralateral side. The mnemonic "COWS" (Cold Opposite, Warm Same) is often used to remember which direction the nystagmus should beat when using the different temperatures. Therefore, one could predict that if the right ear was stimulated with cold water that the nystagmus would beat towards the left ear.

According to Weiss, the affected ear should show a decreased response. One cannot specifically diagnose BPPV by the caloric test itself, but it can lead to identifying which ear is affected. Glasscock et al state that if the sensation of vertigo and nystagmus that is a result of the caloric test is identical to the symptoms that they feel during an attack, then one can predict that the labyrinth is the cause of the vertigo. If the sensation is different, then the vertigo is most likely of central origin. In recent years, the caloric test has been combined with another diagnostic technique in order to make it more of a quantitative
measurement. This diagnostic technique is referred to as electronystagmography or ENG.

ENG is a simple and effective method of measuring eye movements.\textsuperscript{31,32} For many reasons, ENG is becoming more popular. The test can be done with the eyes open or closed and in both the light and dark.\textsuperscript{11,28,31,32} This is important because Frenzel's glasses are not needed. Even though Frenzel's glasses help suppress fixation of the open eye, the nystagmus can still be inhibited by patients focusing on the light that is mounted on the inside of the glasses.\textsuperscript{11} Therefore, it is preferred to measure nystagmus with either the eyes closed or with the eyes open in darkness. Along with this important benefit, ENG allows clinicians to obtain a permanent record of the patient's eye movements, determine if the lesion is peripherally or centrally originated, and to locate which ear is compromised.\textsuperscript{11,28,31}

The principle of ENG lies in the fact that there exists a potential difference between the retina and the cornea. Electrodes are placed around the eye which monitor each eye movement. For example, when the eye moves to the left, the electrode that is placed near that direction becomes more positive and the electrode on the other side of the eye becomes less positive.\textsuperscript{11,31} The electrodes are each connected to a pen recorder that moves according to each eye movement. Along with the electrodes placed around the eyes, a ground electrode is placed on the forehead.
A typical ENG test battery is comprised of seven procedures. The saccade test is performed first for calibration purposes. The patient is instructed to look back and forth between two points that are positioned 20° apart. The recorder is adjusted so that for every 20° of eye movement the pen moves 20 mm. Once the equipment is calibrated, the other six maneuvers are performed.

The gaze nystagmus test has the patient fixating on points that are 20° to 30° to the right and left of center. Next, the patient follows a point that is moving back and forth in a pendular motion called the sinusoidal eye tracking test.

Third, the optikinetic test (OPK) is performed. This test consists of patterns of vertical stripes that move across the visual plane at various speeds. The OPK is followed by positional tests that consist of the patient positioned in both supine and sitting with the head positioned in different orientations. The final two tests have already been discussed. The Hallpike positional test and the caloric test are both performed using ENG to help better interpret the nystagmus.

Once the tests are performed, the results need to be interpreted. In the case of BPPV, the nystagmus takes on a certain character. There is a three to ten second latency period before the nystagmus is recorded. The nystagmus is very intense at the onset but fades within 30 seconds to a minute. The nystagmus of BPPV contains both linear and rotational components. Finally, it has been shown that the direction reverses when the patient is taken out of the provoking position and returned to sitting.
It should be noted that the tests just mentioned can be performed by a variety of professionals. Depending upon the clinic that the patient attends, the physician may perform all of these tests. The clinic may have audiologists or other clinicians who perform the ENG recordings. Some physicians may have connections with a clinic specializing in vestibular rehabilitation who perform such tests. Therefore, one must understand that these are general guidelines to follow when conducting a vestibular examination.

The physical therapist's role in the evaluation thus far has been minimal due to the medical nature of the tests. However, it is the information gained from these tests that allow the therapist to conduct his/her own evaluation.\textsuperscript{27} The therapist needs to have access to the patient's current and past medical history, medication history, and the testing results in order to perform an adequate evaluation. Once the medical history is complete, the therapist can proceed to the rest of the evaluation.

The subjective history is taken first in order for the therapist to gain an understanding of the patient's symptoms and complaints first hand.\textsuperscript{27} Also, there may be new information not included in the physician's report that may help with the evaluation. The patient's symptoms should be documented fully so that they can be referred to later in the rehabilitation process. The therapist must discover what conditions precede the attacks of BPPV and note if any functional activities have been limited due to BPPV. Finally, the patient should set forth
some goals of therapy. Allow them to discuss what they want out of the therapy sessions and then decide if these goals are attainable.

The objective testing of a patient with BPPV by a therapist is much more functional in nature than that of a physician. This evaluation is much like any other evaluation conducted by a therapist. Active and passive range of motion are tested first. This is followed by manual muscle testing of the upper and lower extremities. A functional gait assessment is performed along with simple ambulation. The patient is also examined for any postural deviations. Sitting and standing balance are then tested using such tests as the Romberg test, single leg stance, and weight shifting. In a patient with BPPV, these should all be within normal limits because the only time the patient is compromised is in the provoking position.

The area of testing that therapists need to pay most attention to is that of positional and movement testing. This is a way of clinically assessing the positions and movements that bring on the patient's symptoms. The testing positions and movements used in this portion of the evaluation attempt to simulate those that occur in the everyday life of the patient. Once again, the therapist must keep in mind that the patient may be reluctant to assume the positions that provoke the attacks of BPPV. They must be reassured that this testing will in fact lead to the answer to their problems.

As stated earlier, one of the most revealing positional tests is the Hallpike maneuver. Although this may have been tested previously by the physician or
other clinician, it may be important to perform this test again. This will allow the therapist to view the attack first hand and set a baseline to which progress can be compared. The patient can be asked to rate the discomfort level on a scale that can be used again later in the rehabilitation. There are other positional tests that include changing to and from positions such as sitting, standing, supine, and head hanging. These will be discussed in further detail in the next chapter during the discussion of a treatment protocol.

In reviewing the evaluation techniques just presented, one must keep in mind that the patient’s history is a critical aspect of the examination and can lead to a proper diagnosis much of the time especially in the case of BPPV. Weiss¹ reminds us that many vestibular function tests such as ENG are costly procedures and are not always needed to reach a correct diagnosis. He also stresses that they “do not replace the careful clinical examination of patients with vertigo.”¹(61) The therapist must remember that the evaluation is really the start of the treatment. As the therapist becomes familiar with the precipitating factors and symptoms associated with the patient’s attacks, he/she can begin constructing a plan of treatment. The following chapter will focus on a variety of rehabilitation techniques that are currently being used by clinicians.
CHAPTER V
TREATMENT

Although benign paroxysmal positional vertigo (BPPV) was first described in 1921 by Barany,9 it was not until 1944 that a method of “vestibular habituation therapy” was described by Cawthorne33 as a means to treat this dysfunction. Since this time, many other approaches have been developed to treat BPPV. Other treatment protocols have ranged from exercise therapy in various forms3,6,25,26,34 to surgical intervention5,36-38 and the use of antivertigo drugs.4,29 The focus of this chapter will be on the treatment approaches of vestibular habituation and of exercise therapy. The reason for this approach is threefold. First, these two approaches have reported high success rates and have increasingly been used by physical therapists.12 Secondly, there are questions on the efficacy of pharmacological intervention.6,8,25 Finally, surgery is usually implemented when all other treatment approaches have failed.3,5,25,26,36

The theory of using habituation training with patients suffering from unilateral vestibular dysfunction was originated by Cawthorne in 1944.33 In combination with Dr. Cooksey, he originated a protocol of exercise that would tend to bring on a patient’s symptoms. The patient is instructed to perform the exercises repeatedly until the symptoms eventually subside. The theory behind
habituation lies in the belief that the patient will eventually be able to assume the provoking position and be symptom free. The therapeutic mechanisms at work are that of adaptation and compensation. As the patient repeats the provoking position, the posterior semicircular canal continues ending signals to the CNS. According to the adaptation theory, the resulting action taken by the CNS is reduced and eventually extinguished.

The Cawthorne-Cooksey exercises (Table 1) are arranged in a progressive manner. The exercises start with simple eye and head movements with the patient lying in bed. From here, they are performed in the sitting and standing positions with more functional activities included. Finally, the patient performs exercises while moving about. Another important aspect of the Cawthorne-Cooksey exercises involves the patient performing them with eyes both opened and closed. Having the eyes closed puts more of a strain on the vestibular system due to the loss of the visual input. Finally, the patients are encouraged to join in group therapeutic sessions. Group therapeutic sessions allow for moral support from others with similar medical conditions and provides a more economical format.

The theory of habituation training was taken to a new level when Norre expanded on the ideas set forth by Cawthorne and Cooksey. Norre developed 34 exercises that resembled the Cawthorne-Cooksey exercises and were used as both a test battery and as a means of rehabilitation. The area in which Norre differed the greatest was his belief in having a specific set of exercises for each
Table 1. — Diagnostic protocol developed by Norre

<table>
<thead>
<tr>
<th>POSITION CHANGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting to supine</td>
</tr>
<tr>
<td>Supine to right side</td>
</tr>
<tr>
<td>Supine to left side</td>
</tr>
<tr>
<td>Supine to sitting</td>
</tr>
<tr>
<td>Standing and turning to the right</td>
</tr>
<tr>
<td>Standing and turning to the left</td>
</tr>
<tr>
<td>Sitting and place nose on right knee</td>
</tr>
<tr>
<td>Sitting and place nose on left knee</td>
</tr>
<tr>
<td>Sitting and turning head clockwise</td>
</tr>
<tr>
<td>Sitting and turning head counterclockwise</td>
</tr>
<tr>
<td>Sitting and bending forward</td>
</tr>
<tr>
<td>Sitting to standing</td>
</tr>
<tr>
<td>Sitting with head moving into flexion and extension</td>
</tr>
<tr>
<td>Sitting and moving into right Hallpike position</td>
</tr>
<tr>
<td>Right Hallpike position to sitting</td>
</tr>
<tr>
<td>Sitting and moving into left Hallpike position</td>
</tr>
<tr>
<td>Left Hallpike position to sitting</td>
</tr>
<tr>
<td>Head hanging</td>
</tr>
<tr>
<td>Supine to sitting</td>
</tr>
</tbody>
</table>

individual patient. Eventually, the 34 exercises were reduced to 19 as the maneuvers that did not induce vertigo and nystagmus in enough cases were eliminated from the protocol.

All 19 maneuvers of the test battery (Table 2) are used for each patient who may be a candidate for vestibular rehabilitation. As each exercise is executed, it is reported whether or not vertigo and nystagmus are elicited. The intensity and duration of the vertigo is also recorded. The maneuvers that are positive for vertigo are marked with the symbol M+ and the symbol Ny+ is used to indicate those that are positive for nystagmus. Therefore, a maneuver that is positive for both vertigo and nystagmus is categorized as M+Ny+. Another category involves those maneuvers that bring on vertigo without accompanying nystagmus. These are marked with the symbol M+Ny-. Once all 19 maneuvers are completed, an exercise protocol is constructed using the maneuvers that were marked M+Ny+. When this process is followed, each individual will have his/her own rehabilitation protocol.

Norre\textsuperscript{39(p884)} states that the exercises should be "executed in a rather vivid way." The maneuvers are to be performed five times successively. This would be repeated two to three times daily in the patient's home. As did Cawthorne and Cooksey, Norre believed that the mechanism of adaptation was responsible for the patient's ability to work through the symptoms and in time assume the provoking position in a symptom-free manner.
Table 2. — Cawthorne-Cooksey Exercises

<table>
<thead>
<tr>
<th>PROGRESSION OF EXERCISES</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Performed in supine:</td>
</tr>
<tr>
<td>A. Eye movements -- *progress from slow to quick</td>
</tr>
<tr>
<td>- up/down, side to side, focus on object moving towards and away from eyes</td>
</tr>
<tr>
<td>B. Head movements -- *progress from slow to quick</td>
</tr>
<tr>
<td>*start with eyes open and progress to eyes closed</td>
</tr>
<tr>
<td>- flexion/extension, cervical rotation</td>
</tr>
<tr>
<td>II. Performed in sitting:</td>
</tr>
<tr>
<td>- Repeat A and B from supine position</td>
</tr>
<tr>
<td>- Circling and shrugging of the shoulders</td>
</tr>
<tr>
<td>III. Performed in standing:</td>
</tr>
<tr>
<td>- Repeat I and II as stated above in standing</td>
</tr>
<tr>
<td>- Transfer from sitting to standing with eyes open and then shut</td>
</tr>
<tr>
<td>- Toss ball from one hand to the other in front of face</td>
</tr>
<tr>
<td>- Pass ball from one hand to the other between the knees</td>
</tr>
<tr>
<td>- Transfer from sitting to standing while turning around inbetween</td>
</tr>
<tr>
<td>IV. Performed while moving about:</td>
</tr>
<tr>
<td>- Walk around a circle while a person in the middle throws a ball to be caught and thrown back to person.</td>
</tr>
<tr>
<td>- Ambulate across treatment area with eyes open and shut</td>
</tr>
<tr>
<td>- Walk up and down a slope with eyes open and closed</td>
</tr>
<tr>
<td>- Ascend and descend stairs with eyes open and closed</td>
</tr>
<tr>
<td>- Perform game that requires bending down, stretching, and stooping</td>
</tr>
</tbody>
</table>

While that idea of habituation training focuses on reducing the effects of the CNS, the exercises introduced by Brandt and Daroff focus on the otolithic matter that is believed to be the cause of BPPV. Brandt and Daroff feel that through repetitive movements of the head, the disruptive particles can be dislodged from the cupula and recollected back into an area of the labyrinth that will not cause vertigo. The final collection area of the otolithic particles is not completely known but is proposed to be an area called the labyrinthine recessus.

The process is performed as follows (Figure 4). The patient is taken from the seated position into the provoking position much similar to that of the Hallpike maneuver. In this case, however, the patient remains on his/her side as opposed to the supine position and the head is allowed to stay on the table with the lateral aspect of the occiput in contact with the table. This placement of the head allows for proper alignment of the posterior semicircular canal. This position is maintained until an attack of vertigo is achieved. The patient is then instructed to remain in the position until the symptoms have fully dissipated. The patient then immediately returns to the seated position for 30 seconds. This change in position often causes nystagmus in the opposite direction. The patient then assumes a position that mirrors the original sidelying position for 30 seconds.

These three position changes comprise the maneuver. During each session, the maneuver is repeated until the nystagmus has fully subsided.
Figure 4. Exercise therapy for positional vertigo. Adapted from: Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol.* 1980;106:485.
Brandt and Daroff suggest that this entire procedure be performed every three hours while awake until two consecutive vertigo-free days are achieved. Brandt and Daroff argue against central compensation as the mechanism at work in their treatment approach. They state, "The fatigability of the vertigo during individual sessions was too rapid for a habituative central mechanism that requires hundreds of repetitions."25(p485)

In 1988, Semont34 described a single treatment approach using positioning similar to those found in Brandt and Daroff’s therapy. Named the Liberatory maneuver, this procedure works by floating the otolithic material from the posterior semicircular canal into the common crus to the utricle. Once the involved ear is identified, the patient is quickly taken from the seated position into a side-lying position with the involved ear directed towards the floor. The head should be slightly declined. The patient is observed for vertigo and nystagmus. The observed nystagmus must have the fast phase directed towards the undermost ear with a torsional component. The patient remains in this position until the symptoms subside. After two to three minutes in this position, the patient’s head and neck are grasped by the examiner with both hands and the patient is rapidly taken to the opposite side-lying position. In this position, the nystagmus will be directed towards the upper ear. The patient remains in this position for five minutes and is then very slowly returned to sitting.
After the procedure has been completed, the patient is to keep his/her head completely vertical for the next 48 hours. The following "Instructions to Patients" is given:

Whatever the position of your body, you must keep your head vertical for the next 48 hours. Imagine your head being hung by an invisible string to the ceiling. You must not bend your head forward or backwards. You must not go to the barber, hairdresser, or dentist. No exercise. When men shave under their chins, they should bend their bodies forward in order to tense the skin and keep their head vertical. No eye drops. Shampoo only under the shower. At night, lie on your back with plenty of pillows to keep your head vertical while your trunk is about 30° to 45° elevated off the bed. Put something at the bottom of the bed in order not to slip down during the night.\(^6\)

When the 48 hour period has been completed, the patients are instructed not to sleep on their compromised side for the next week. If the treatment is not successful, it is performed again one week later.

The final type of exercise therapy to discuss is that of the particle repositioning maneuvers. Two very similar techniques are described that attempt, through a series of head maneuvers, to float the particles into the
common crus. These two procedures differ from Brandt and Daroff and the Liberatory maneuvers in that only the head is moved once the provoking position is established. They do share the same goal of relocating the particles to an area of the labyrinth that does not cause vertigo with head movements.

Welling and Barnes\textsuperscript{26} refer to their procedure as the Particle Repositioning Maneuver while Epley\textsuperscript{3} titled the technique the Canalith Repositioning Procedure. The positioning of the head and patient's body are very similar and will be described here as one (Figure 5). The patient is once again taken from the seated position to the Hallpike testing position with the involved ear pointed down and the head extended off the end of the table at 45°. This position will bring on the patient's symptoms. The symptoms should then be allowed to dissipate. At this point, the patient slowly rolls into the opposite head hanging position and the symptoms are allowed to dissipate if any are present.

Once the vertigo has subsided, the patient is asked to slowly roll onto his/her side while the examiner supports the head maintaining the 45° of extension. This side-lying position allows the head to rotate further to the point where the patient is facing the floor. The patient's head should now be 180° from the original Hallpike position. At this point, the particles should be floating away from the cupula towards the common crus. The patient remains in this position for three to four minutes and is then slowly returned to sitting.
SEATED ➔ SUPINE WITH RIGHT HEAD HANGING ➔ SUPINE WITH LEFT HEAD HANGING ➔ LEFT SIDELYING

Figure 5. Particle repositioning maneuver. Adapted from: Welling DB, Barnes DE. Particle repositioning maneuver for benign paroxysmal positional vertigo. Laryngoscope. 1994;104:947.
As was seen in Semont's Liberatory maneuver, the patient is instructed to keep the head aligned vertically for the next 48 hours. Epley\textsuperscript{3} feels that the patient should stay off the involved side for the next five days while Welling and Barnes state that normal head movements can be resumed as soon as two days. Although the head maneuvers of the two procedures are similar, Epley differs in one major way. Epley believes that the addition of a bone vibrator over the mastoid process will help in the movement of the particles. Welling and Barnes\textsuperscript{26} do not use the added vibration but state that it may aid in dislodging any particles that may be attached to the cupula.

One purpose of creating the variety of exercise treatments just described was to find an alternative to invasive surgery.\textsuperscript{3} Although the surgical technique described by Gacek\textsuperscript{37} has been successful in a number of patients, Epley\textsuperscript{3(p399)} feels that the invasive procedure involves "significant risk and postoperative morbidity." This has led many clinicians to reserve surgery to those cases that cannot be cured through exercise therapy.\textsuperscript{3,5,25,26,36}

In such a case where all noninvasive attempts have failed, there are two surgical procedures available. The first procedure developed by the aforementioned Gacek\textsuperscript{37} involves transection of the posterior ampullary nerve. This nerve innervates the ampulla of the posterior semicircular canal. Once the nerve is severed, the patient is relieved of symptoms of BPPV due to the fact that the brain no longer receives faulty messages regarding head movements.
The surgery holds the risk of sensorineural deafness and labyrinthine destruction.  

Epley\(^3\) feels that the second surgical procedure "promises less morbidity and risk." Developed by Parnes and McClure,\(^3\) the posterior semicircular canal occlusion procedure attempts to block the flow of endolymph within the canal. The lack of endolymphatic flow limits the cupular deflection that leads to the problems of vertigo.

While these two surgical procedures have shown some success, the use of antivertigo drugs has raised some questions.\(^5,8,25\) Troost and Patton\(^6\) state, "In general, medication has not successfully abated these symptoms, and many patients have sought assistance from a variety of medical specialists and tried a large number of medications unsuccessfully before the exercise therapy." This belief is also reinforced by Brandt and Daroff\(^25\) who feel that medications "have not proven to be particularly efficacious."

Finally, Fujino et al\(^4\) performed a controlled study on the efficacy of vestibular training versus the use of antivertigo drugs. They concluded that the first choice in treatment should be that of vestibular training. They came to this conclusion by comparing three groups: one with vestibular training and a placebo drug, one with antivertigo drugs, and the other with a combination of the drugs and training. The two groups with the vestibular training component showed a greater improvement rate than the medication group alone. They stated that the combination group did not differ significantly from the training
group, thus suggesting that the use of vestibular training itself is the preferred method of rehabilitation.

Whichever method of rehabilitation is chosen to treat the patient with BPPV, the goal of each is identical. The relief of symptoms is the first priority of the therapist. Once this is achieved, the patient must then regain the confidence to resume the positions that caused the vertigo. If the patient cannot be cured with the exercises described, the option of surgery is always available. Physical therapists must make every attempt to use the rehabilitation exercises before surgery is discussed. If used properly, these exercises should provide significant improvement for the patient with BPPV.
CHAPTER VI
CONCLUSION

Benign paroxysmal positional vertigo (BPPV) is a common disorder of the peripheral vestibular system in which the inner ear and its connections to the central nervous system are the areas that are affected. BPPV can lead to such symptoms as vertigo, nystagmus, nausea, and vomiting. With rapid changes in head position, the patient will complain of brief attacks of vertigo that are at times disabling. The head position which causes the problem of BPPV is very specific. The head is often rapidly extended and rotated as if one would look over one's shoulder or roll over in bed. The majority of the cases of BPPV will occur unilaterally with the involved ear turned downward during the head positioning.

The fact that the head needs to be positioned in such a specific manner has led to the discovery that the posterior semicircular canal is responsible for the pathological process. As discussed in Chapter III, Schuknecht\textsuperscript{22} proposed that the cupula of the posterior semicircular canal is affected by floating particles that originate in the otoconial layer. Cupulolithiasis is the term given to the pathophysiological process in BPPV. Schuknecht believes that the particles adhere to the cupula and deflect it causing the brain to interpret head movement.
when none is actually occurring. Others\textsuperscript{22} feel that the particles change the specific gravity of the endolymph. This in turn causes the cupula to become oversensitive to changes in head position.

The key diagnostic test for BPPV is the Dix-Hallpike maneuver.\textsuperscript{10} This maneuver is described in detail in Chapter IV. The clinical findings are latency of onset, short duration, fatigability, and rotational nystagmus with the fast phase beating towards the undermost ear. The importance of these findings are two-fold. First, it allows the clinician to differentiate BPPV from a vertigo of central vestibular system origin. Secondly, the therapist can use the benefits of fatigability, along with other therapeutic techniques, to rehabilitate the patient.

The treatment of BPPV can be divided into four general categories that were examined in Chapter V. These are habituation training, exercise therapy, surgery, and drugs. The habituation training and exercise therapy can be administered by trained physical therapists. These two conservative methods are often attempted before the more invasive and dangerous surgical procedures are employed. Pharmacological intervention has not been proven effective at this point as compared to the success of habituation training and exercise therapy.

It is the opinion of this author that the disorder of BPPV is being diagnosed and treated in a successful manner in certain regions of the country. The Dix-Hallpike maneuver has been used since 1952 and continues to diagnose BPPV when performed correctly. The high rates of treatment success
reported by Brandt, Semont, and Norre support this theory as well. As new techniques become available, one should see these rates increase somewhat.

One area that continues to be a problem is the fact that in most rehabilitation techniques, the patient must fight through numerous attacks before getting better. This author feels that the addition of certain drugs that reduce the effects of BPPV should be incorporated into these therapeutic sessions. By lessening the affect of the symptoms, more patients would be willing to try these forms of rehabilitation. Many patients begin the treatment but soon quit because of the continued attacks. This would also allow the therapist to try many different techniques in a given treatment session that may have been too excruciating without the aid of drugs. More research in this area would be beneficial.

Although the pioneering treatment procedures outlined in Chapter V were originated in 1944, it seems that the field of physical therapy is only starting to become involved. This author's conversation with a local physician supports this theory. Although there are two main outpatient clinics in the city, he finds that he cannot refer patients because of the lack of experience and familiarity in the area of BPPV. He ends up treating the patient himself, often trying many maneuvers until something is successful. This is a situation that shows there is room for growth of the physical therapy field in the Midwest region of the country.

There are, however, questions that still need to be answered. By explaining some of these mysteries, the treatment of BPPV can only be
enhanced. First, pathophysiologically, do the particles actually attach to the cupula and deflect it or is the specific gravity of the endolymph changed? This has not yet been determined. New drugs could possibly be developed to prevent this. Secondly, how exactly do the single-treatment procedures work? Is the head rotated enough during the Liberatory maneuver to float the particles out of the posterior canal? According to Semont,\textsuperscript{34} it does. Finally, what improvements can be made in the rehabilitation procedure to improve efficacy and reduce patient discomfort? Hopefully, this last question will be answered in the near future.

As for now, the diagnosis and treatment of BPPV will continue on its current path until a new means of therapy is created. Therapists need to become more proficient with the techniques and familiar with the benefits that physical therapy has to offer to the patient afflicted with BPPV. If this is made possible, more physicians will be able to refer patients to physical therapy for rehabilitation. This may be an important step in assuring an increasing efficacy and rate of rehabilitation for future patients diagnosed with BPPV.
REFERENCES


29. Dix MR. The clinical evaluation and pharmacological treatment of vertigo.


