Tighe_M Module 4 Assignment

Introduction and Epidemiology

Benign Paroxysmal Positional Vertigo (BPPV) is the most common adult vestibular disorder, with a lifetime prevalence of 2.4%. [(1)] It is the most common cause of dizziness encountered in the clinical setting, and may account for 20-30% of patients with dizziness. Some estimates show the incidence in the general population is 64/100,000, and as many as 50% of individuals over the age of 65 with dizziness symptoms are resulting from BPPV. [(3), (5)]

Some 5.6 million clinical visits in the United States each year are due to symptoms or issues related to BPPV. The average cost to arrive at a diagnosis of BPPV is $2000 per patient, including multiple clinical visits and diagnostic testing, and the total health care cost of BPPV issues in the U.S. is $2 billion annually. In addition to these direct costs, 86% of patients with BPPV report either interrupted activities of daily living (ADLs) or lost days at work from the disorder, itself, or the clinical visits needed to diagnose and treat BPPV. Included in these statistics are issues of unnecessary diagnostic testing to finally arrive at the diagnosis of BPPV, and inappropriately prescribed vestibular suppressant medications. [(2),(4), (5)]

Two other issues to consider that will make it even more important for physical therapy clinicians to appreciate BPPV are two particular patient populations we will commonly encounter in the clinical setting. BPPV is not a disease of aging, but it is significant to note the number of patients over the age of 65 who develop BPPV, [(14)] as this is such a fast-growing segment of the U.S. Population. Besides the obvious issues of hospital admissions for fall-related injuries related to BPPV, consider too, those for dehydration if excessive vomiting occurs from vertigo signs and symptoms (S&S), especially in an elderly population. [(2)]

The second patient group is related, unfortunately, to the major geopolitical situation that has affected the U.S. for most of the last decade. Since the terrorist attacks of September 11, 2001, the U.S. military has found itself engaged in 12 years of continuous deployment against an enemy that has utilized improvised explosive devices (IEDs) to inflict casualties on American soldiers. There is a difference between the mechanism of injury inflicted by High-Order Explosive (HE) via a supersonic over-pressurization wave, and Low-Order Explosive (LE) blasts via ballistics, fragmentation, the “blast wind”, and blunt trauma. But a post-blast vertigo, with in some cases trauma to the semi-circular canals, has been identified, as well as a vertigo related to mild traumatic brain injury (mTBI). [(6)] The long-term ramifications of these injuries is yet to be assessed, but may be clinically relevant to physical therapy in the identification and treatment of BPPV.

Relevant Anatomy and Physiology [5], [8], [9]

The vestibular system has three primary functions: (1) subjective sensation of motion and spatial orientation of the head, (2) adjust muscular activity and body position to maintain posture, and, (3) to stabilize a fixation point for the eyes when the head moves, keeping a stable image on the retinas.

The primary sensory organ used to accomplish this task is the vestibular labyrinth, located in the inner ear and anatomically closely associated with the cochlea, so close that it shares a common special nerve, Cranial Nerve VIII, the Vestibular-Cochlear Nerve (nee, the Acoustic Nerve). The vestibular labyrinth contains 5 structures: 3 semi-circular canals (anterior, posterior, and horizontal) and 2 otolith organs (the utricle and the saccule).

The semi-circular canals (SCCs) are filled with a substance called endolymph; slightly more dense than water, it contains a high concentration of potassium, smaller concentrations of sodium, and importantly (in the case of BPPV) no otoliths (small bone chips or cystals made of calcium carbonate). The SCCs are aligned in three co-planar pairs between the right and left vestibular labyrinths: both horizontal canals are one pair, while the anterior and contralateral posterior canals each form the other two pairs. They detect rotational movements of the head. At one end of each canal is a closed dilation called the ampulla; within each ampulla is a thick, gelatinous cap that runs perpendicularly across the canal called the cupula. The cupula contains many hair cells called kinocilia and that point to a larger hair cell called the stereocilium. Bending of the hair cells opens potassium channels, initiating an increase in action potentials to that particular canal's input to the Vestibulo-cochlear Nerve.

Below the SCCs lie the otolith organs, the utricle (the more superior of the structures, it connects to the SCCs) and the saccule. The otolith organs are also filled with endolymph. Their sense structure is called the macula, and it differs from the cupula in that it is overlaid by a membrane that contains gel, hair cells, and embedded otoliths. This "otolithic mass" provides inertia and responds to gravity (you could consider it, therefore, a "gravity detector"). The utricular macula is parallel to the ground when the head is upright, while the saccular macula is oriented vertically in the upright position. An apt analogy may be to compare the utricle and saccule to an old carpenter's level with a horizontally and a vertically oriented tube. Within each is a bubble that responds to gravity, and that moves if the level either moves, or is placed on an un-level surface.

How The Vestibular System Works [5], [8], [9]

The otolith organs are designed to detect changes in acceleration, not to respond to steady-state conditions. An effective description is to consider what you feel when you travel by air, say in a large airliner. With take-off, you are subjected to a large acceleration force; even with your eyes closed, you are acutely aware of the sensation of movement. As the plane attains cruising altitude and begins to "level off", you are subject to a gradual deceleration force that,

once again, you can feel. Once the "seatbelt sign" is turned off, and the captain makes the familiar "You are now free to move about the cabin" announcement overhead, you longer have the sensation of movement. You can walk about the interior of the plane with as little difficulty as you would if you were still on the ground. You are still in motion, of course, travelling at over 600 miles per hour! However, your otolith organs are no longer giving you the sensation of change of movement, because they (and you) have accommodated to the straight-line (linear) motion of the plane.

The SCC's are designed to detect three-dimensional changes in the position of the head or of continuous movement of the head. Consider the mathematical structure of a 3-D graph (Cartesian coordiates) with an x-axis, a y-axis, and a z-axis. In each ear, the Horizontal canal (HSC) would be oriented to the x-axis, the Superior (SSC) canal to the y-axis, and the Posterior canal (PSC) to the z-axis.

The easiest way to describe how this canal system works is to utilize the co-planar pairing of the horizontal semi-circular canals (the HSCs). With the head tilted 30 degrees forward (the downward portion of nodding yes) these canals are now parallel to the ground. When you turn your head to the left the bony labyrinth (the semi-circular canals) turn to the left along with the head, and at the same speed as the head. Recall, however, that the semi-circular canals are filled with endolymphatic fluid. This fluid has an inertia that initially, briefly, resists movement. This initial resistance deforms the cupula in the left HSC, triggering an increase in action potentials from the HSC neural connections to Cranial Nerve VIII. This same movement and inertial response triggers a reduction in action potential release in the right HSC. The result is detection in the brain of head rotation to the left.

If the head continues to rotate to the left (for example, consider a child sitting on a Sit-and-Spin toy), the endolymph quickly overcomes inertia, and now moves at the same speed as the labyrinth system; the cupula continues to be deformed, generating further action potential releases in the left Cranial Nerve VIII. This gives the brain the sense of continuous spinning to the left. When left head movement ceases (the child stops spinning to the left on the Sit-and-Spin) the same inertia that initially resisted endolymphatic fluid movement now keeps it transiently in motion. This deforms the cupula, triggering action potential release, continuing to give the brain the sensation of left head rotation, until the endolymph ceases movement. At this point, action potential firing from both the left and right Cranial Nerve VIII horizontal canal inputs becomes equal, and the sensation of movement ceases.

The mechanism is similar for the paired "vertical canals" (the ipsilateral superior and contralateral posterior canals). If the head rotates forward and to the right, for example, action potential firing rate is increased in the right SSC and reduced in the left PSC, resulting in the detection of, in this case, nodding the head down and to the right (consider what you are doing when you look down to change the channel on the radio while driving the car).

Relevant Neural Pathways [5], [8], [9]

The relevance of all this, and what we will need to consider when attempting to diagnose and treat BPPV, is the neurological connections of the vestibular system. Cranial Nerve VIII synapses to the ipsilateral Vestibular Reflex Center in the Medulla Oblongata and to the Flocconodular Lobe of the Cerebellum. Additionally, fibers cross (decusate) to the contralateral thalamus, and from there to the Primary Sensory Cortex of the Parietal Lobe. Here is ultimately where the sensation of movement is registered by the conscious brain.

As mentioned above, one of the vestibular system functions is gaze stabilization. This is accomplished by motor neuron connections between the semi-circular canals and Cranial Nerves III, IV, and VI (the Occulomotor, Trochlear, and Abducens Nerves, respectively) responsible for movement of the eyes. For the Horizontal canals, the connection is with the motor neurons of the Abducens (control of the contralateral Lateral Rectus muscle) and the Occulomotor (control of the ipsilateral Medial Rectus muscle). For the Vertical Canals, this involves motor neurons for the Occulomotor (control of the Superior and Inferior Recti and Oblique muscles) and the Abducens (control of the Superior Oblique muscles).

To illustrate this control mechanism, consider again the example of turning the head to the left. To maintain gaze stability, the contralateral Lateral Rectus muscle (in the right eye) and the Ipsilateral Medial Rectus muscle (in the left eye), will pull together, moving the eyes (and therefore, gaze) to the right. This is a normal response, described as the Vestibular Occular Reflex (VOR). The eyes move in the direction opposite to head movement in order to keep an object of focus or fixation centered on the fovea.

Benign Paroxysmal Positional Vertigo (BPPV) [2],[3],[5], [8], [9],[15]

BPPV is believed to be caused by what is termed vestibular lithiasis; that is, debris (most commonly believed to be displaced otoliths) gets into the SCCs, provoking abnormal sensory input via the Vestibulocochlear Nerve. The symptoms of BPPV can include: episodic, mild to intense complaints of dizziness, blurred vision, unsteadiness, vertigo, nausea, vomiting, and losses of balance. These symptoms are usually provoked by placing the head in a certain provoking position, but can sometimes occur with just simple movement. Three identified pathologies include canalithiasis (free-floating debris in a SCC), cupulalithiasis (debris that is attached, adhered, or impinging on the cupula), and otolithic debris jammed into a canal or cupula.

In canalithiasis, otoconia are freely mobile in either the posterior semi-circular canal (the most common location, cited in 85-95% of cases) or the horizontal canal (cited in 5–15% of cases). This free-floating debris falls to the lowest point of the affected canal, displacing

endolymph. This resulting endolymph flow deflects the cupula, provoking symptoms that can include vertigo and nystagmus in the plane of the involved SCC.

In cupulolithiasis, otoconia (or debris) become adherent to the cupula. This increases the density of the affected cupula and makes it sensitive to gravity. In the provoking position, the pull of gravity deflects the cupula, provoking the symptoms, again in the plane of the involved canal.

BPPV is further defined as Ideopathic (or Primary) in which the cause of the detachment could not be established, and Secondary (or Acquired) in which head trauma or inner ear disease is the initiating pathology. Examples include vestibular neuritis, a blow to the head, concussion, exposure to HE or LE blasts, and Meniere's disease. Some references state that any inner ear disease that detaches otoconia but does not destroy SCC function can induce BPPV.

Physical Therapy Diagnosis

Understanding the normal functioning of the vestibular system, and the pathophysiology of BPPV as described above, the diagnosis of BPPV becomes rather straightforward. The two "gold standards" for diagnosis are the history of the onset of symptoms, and eye-findings during positional testing, typically the Dix-Hallpike Maneuver.

In history, patients will describe a provoking mechanism (for example, rolling in one direction on the bed, but not the other, provokes vertigo). Vertigo is defined as an illusion of movement, usually a sense of spinning, and can be in a horizontal, vertical, or rotary direction. This is sometimes accompanied by a sense of disequilibrium (a feeling of being off-balance) or of floating. The direction of the sense of vertigo can indicate which semi-circular canal is involved, and whether it may be a canalithiasis or cupulolithiasis issue.

The vertigo associated with BPPV is usually transient, lasting only for 30 seconds or less, but can be very intense. Especially for patients with sensitivity to motion, the vertigo can provoke motion sickness-like nausea or even vomiting. Common provoking stimuli are rolling over in bed, extending the neck to look up, and bending forward. The provoking direction can often be used to identify the affected ear (for example, vertigo that onset with rolling to the right but not with rolling to the left may indicate a right-ear involvement). The vertigo can sometimes come in clusters or spells, sometimes several times a week, or even several times a day.

Nystagmus is a rapid, involuntary rhythmic movement of the eyes. It can present horizontally, vertically, or in a rotational direction. There is usually a fast twitch and a slow twitch component, sometimes referred to as a corrective saccade. There are multiple conditions that can produce nystagmus; in the diagnosis of BPPV, it is usually significant in the performance of positioning tests: for this discussion, the Dix-Hallpike and the Roll Test.

The Dix-Hallpike Test is used to diagnose posterior canal BPPV. Each posterior canal (that is, each ear) needs to be tested. To test the right ear, the patient is seated edge of bed, in such a position that being brought to supine will allow their head to extend past neutral off the edge of the bed. The examiner rotates the patient's head 45 degrees toward the ear being tested. The patient is quickly lowered to supine with the head maintained at 45 degrees rotation and is dropped back into around 30 degrees of cervical extension. You are looking for reproduction of the patient's complaints of vertigo, and the appearance of a rotational nystagmus with a fast-twitch counter clockwise. Parnes et al decribed this as the superior pole of the eye beating toward the affected (or downward or gravity-dependent) ear. The nystagmus should have a brief latency period (should onset within 1 to 5 seconds of attaining the position) and should last only briefly (around 30 seconds, similar in time to the duration of the vertigo complaint). The patient is then brought quickly up to the starting position, and observed again for both vertigo complaints, and a nystagmus in the opposite direction of that noted in the supine, neck extended position. With BPPV, the phenomena should fatigue out with repeated testing.

For suspected BPPV involving the horizontal canals, the Roll Test is commonly used. The patient is placed in supine position, in about 20 degrees of cervical flexion. The examiner then rotates the head 90 degrees toward the canal being tested. The nysgamus, if provoked in this case, will be either fast-twitch geotropically (downgoing, in the direction of gravity) or occasionally agogeotropically (opposite the direction of gravity). Typically, this latency period will be shorter, of a stronger intensity, and less prone to fatigue.

International Classification of Functioning (ICF) Considerations [10], [11], [12]

I freely admit that my experience with the ICF is very limited; in point of fact, I only became acquainted with it after returning to academia to pursue my transitional Doctorate of Physical Therapy degree last fall. Applying the "top half" of the ICF model, I would consider these issues (a partial list, to be sure) in the ICF domains of health conditions, impairments, activity limitations, and participation restrictions.

<u>Health</u> <u>Conditions</u>

- Ideopathic (Primary) BPPV
- Acquired (Secondary) BPPV
- Posterior (85-95% of the time) or Horizontal Canal involvement.
- Canalithiasis
- Cupulolithiasis

<u>Impairments</u>

- Nystagmus – consider the direction to identify involved SCC.

- Movement-induced vertigo, nausea, vomiting.
- Position-induced vertigo, nausea, vomiting.
- Migraine headache associated with vertigo, nausea, vomiting.

Activity Limitations

- Unable to move the head in a certain way.
- Unable to sit or lay in certain positions.
- Difficulty transferring.
- Difficulty ambulating.
- Associated deconditioning of aerobic and musculoskeletal systems.

Participation Restrictions

- Unable to get up and go to work due to the symptoms.
- Unable to engage in work related tasks due to symptoms.
    - Manual laborer: unable to climb ladder or safely get up on roof.
    - Police, ambulance, or taxi drivers: unable to drive their vehicles safely.
    - Diver: becomes disoriented underwater.
    - Surgeon: unable to place head in position needed to perform operations.
- Unable to drive the car, check blind spot, due to vertigo symptoms.
- Student : unable to return to school, college, graduate work, residency.
- Unable to engage in previously enjoyed recreation activities.
- Unable to appropriately care for children or dependent family members as before the onset of BPPV.

American Physical Therapy Association (APTA) Guide Patterns

The *APTA Guide to Physical Therapy Practice* identifies the following practice pattern directly applicable to BPPV: [7]

5D: Impaired Motor Function and Sensory Integration Associated With Non-Progressive Disorders of the Central Nervous System – Acquired in Adolescence or Adulthood.

Further, consider the recommended **ICD-9** codes from the same edition: [7]

386 Vertiginous Syndromes and Other Disorders of the Vestibular System

386.5 Labyrinthine Dysfunction.

Physical Therapy Interventions

Treatment of BPPV is sometimes difficulty simply due to the transient nature of the disorder. As noted above, primary BPPV often has no immediately discernible cause, and because of this sometimes subsides before treatment interventions by licensed health care providers can be initiated.

Typical treatment focuses on repositioning techniques to try to get the free-floating debris to move from the semi-circular canals back into the utricle. Typical of these repositioning techniques is the Epley Maneuver.

- Patient is placed in a longsitting position on the bed. Patients head is rotated 45 degrees toward the side that provokes the worst vertigo.
- Patient is then quickly brought back into supine and cervical extension. This position is held by the clinician until the vertigo subsides, and sometimes for an additional 30 seconds.
- With head continued to be held in cervical extension, passively rotate the head to the same angle facing the opposite way. Hold this position x 30 seconds or until any vertigo ceases.
- Patient is then assisted to rolling onto the side they are facing (the provoking side should now be facing up. Position is again held x 30 seconds or until any vertigo subsides.
- Patient is then assisted back up to sitting, but should be in a dangled sit versus the initial longsitting position.

For the horizontal canal, an effective, simple approach is the "Gufoni Manoeuvre". It consists of the following portions:

- Patient sits upright on the edge of an examination table.
- Patient is then briskly tilted from seated to sidelying position:
    - Toward the healthy side for geotropic nystagmus.
    - Toward affected side for apogeotropic nystagmus.
- Head is then downturned 45 degrees.
- Patient is maintained in this position x 2-3 minutes.
- Patient is then passively returned to original sitting position.

One issue noted was occasional conversion of horizontal canal BPPV into posterior canal BPPV. A recently developed "Modified Gufoni Maneuver" appeared to address this:

- Patient sits upright on the edge of an examination table.
- Patient is then tilted to full sidelying, but in 2 stages:
    - Rapid excursion for 45 degrees, quick stop, maintain this x 15 seconds.
    - Slow completion of the last 45 degrees to full sidelying.

- Head is then turned downward 45 degrees, maintained in this position x 2 minutes.
- Patient is then passively returned to original position.

While much BPPV literature does not distinguish between BPPV from trauma versus other causes, it is worth considering, especially with the increasing awareness of concussion in athletes and blast injuries in soldiers, as noted by the Hofer study above. (6) A 2004 study by Gordon et al did identify a difference in idiopathic BPPV (iBPPV) versus traumatic BPPV (tBPPV), in the response to the typical method of treatment (physical interventions including Epley maneuvers, "barbecue rolls", etc;) Typically, these canalith repositioning maneuvers are one-time interventions that succeed in moving the displaced otoconia out of the semi-circular canals and back into the utricle. Gordon reported that 67% of his patients with tBPPV required repeat treatment with these maneuvers in subsequent therapy sessions to attain complete symptom resolution, versus 14% of iBVVP patients. Further, in a 22-month follow-up of this same cohort, tBPPV patients reported a 57% recurrence rate of symptoms versus 19% of his iBPPV patients. (13)

The Gordon study suggested a possible cause of this apparent resistance to treatment, and the frequehcy of recurrence, may be directly related to the initial trauma, itself. The force of the trauma (be it blow to the head, exposure to blast wave), causes violent otoconia detachment, which causes microscopic hemorrhages (or "tissue shearing") and biochemical changes that enhance otoconial clot formation. After a successful otolith repositioning maneuver, microscopic changes may then re-activate production of new clots, thereby producing more canalithiasis debris. (13)

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