Etiologies, Efficacy and Treatment of Unilateral Peripheral Vestibular Hypofunction (UPVH)

1. **Etiologies of UPVH**

   Patients who are diagnosed with UPVH, are a heterogeneous group [1, 2] including:

   a. Benign paroxysmal positional vertigo (BPPV): displaced otoconia cause abnormal vestibular output resulting in vertigo and imbalance. [1]
   b. Vestibular neuritis and labyrinthitis secondary to viral infection causing inflammation or vascular occlusion. [1-3] When hearing loss is present typically diagnosed as labrinthitis. [3]
   c. Perilymphatic fistula (PLF) when perilymph spreads into middle ear. [1-3]
   d. One-sided Méniére's disease secondary to elevated endolymph pressure (endolymphatic hydrodrops). [3]
   e. Post-operative vestibular problems: labyrinthectomy, neurorectomy, intra-tympanic injection of gentamycin [1, 2]
   f. Acoustic neuroma (masses at cerebellopontine angle) [1-3]
   g. Superior canal dehiscence: absence or thin temporal bone overlying superior SSC. [3]
   h. Trauma can lead to PLF, labyrinthine concussion, temporal bone fractures, or endolyphatic hydrodrops. [3]

   i. Differential diagnosis between pathologies is often difficult, and many patients receive label of "unilateral vestibulopathy of unknown cause" [2], but this generally is not an issue for physical therapists because treatment and prognosis is more related to presence of symptoms rather than a specific medical diagnosis. [1]A physical therapists needs to identify groups of symptoms in which physical therapy goals can be related and progress can be evaluated. [1]

2. **Efficacy of vestibular rehabilitation (VR)**

   a. A 2007 Cochrane review including 27 randomized control trials and found strong support that VR was more effective than the control group (sham exercises or standard medical care) in reducing dizziness, improving gait, ADLs, visual impairments and balance. [2]

      i. Outcome measures commonly used with UPVH patients: [2]
         1. Subject reported subjective improvement: improved or same/worse symptoms.
         2. Vertigo symptom scale (VSS): patient report of frequency of dizziness, imbalance and symptoms during previous month. [4, 5]OR Dizziness VAS [6]
         3. Dizziness Handicap inventory (DHI): self-report of perception of handicap caused by dizziness. MCID is 18 points. [4] Instructions and tool available at http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=1041
         4. Visual disturbance measured by Dynamic visual acuity (DVA): rating of visual acuity with passive head rotation. [3, 7]
         5. Gait ataxia and sway path as measure of disequilibrium.
         6. Sharpened Romberg test: static balance in tandem.
         7. Dynamic gait index (DGI): performance measure of balance and mobility.
         8. ADL performance:
            a. Veritgo Handicap Questionnaire (VHQ): self-report survey measuring activity restriction and social effects of dizziness. [8]
            b. Vestibular dysfunction- ADL scale (VD-ADL): self report survey measure effects on ADLs. [9]Copy of measure from Cohen HS et al 2000 is Supplementary Document 5.

b. Kulcu el al found that VR is better than medication in improving subjective reports of dizziness and health related quality of life in patients with UPVH. [10]

c. VR is effective during acute stage
    i. Vereeck et al found older individuals (>50 years old) may regain postural control more quickly and maintained it at 12 months than patients who receive general exercise recommendations. [11]
    ii. May reduce symptom duration and medication use at this stage. [12]

d. Refer to my supplementary evidence table (Document 6) for more resources evaluating efficacy of VR compared to standard medical care in improvement of balance and functional mobility.

3. <u>Impairments Commonly seen with UPVH</u>

a. Unilateral loss in input from labyrinths is interpreted as head movement, when no movement exists. [1]
b. During the acute stage impairments include: [1]
    i. Nystagmus
    ii. Postural instability
    iii. Poor compensatory responses to head movement
    iv. Altered perception of body orientation and movement

c. Upon loss of functioning of one labyrinth, vestibular nuclei on both sides of brainstem rely on input from visual, spinal, reticular and cerebellar systems to compensate for loss of vestibular neural activity and to better modulate vestibular response. [1] Typically within a few days or weeks, some of symptoms diminish secondary natural history of vestibular compensation. [1]

d. Sudden acute loss of all peripheral vestibular function in one ear leads to more severe symptoms than a gradual loss of input (e.g. trauma versus a slowly growing acoustic neuroma). [1]

e. **Static symptoms** typically resolve spontaneously in 3-14 days. [1]

    i. Can include: nystagmus, skew deviation, and postural asymmetries in stance. [1]

    ii. Unilateral loss of peripheral vestibular function creates an asymmetry in the push-pull dynamic and disrupts the vestibulo-ocular reflex (VOR) and vestibular spinal reflex (VSR) during static posture leading to nystagmus and postural disturbances. [1]

    iii. Nystagmus in UPVH is typically horizontal in nature (secondary to asymmetries in horizontal canal mates); anterior and posterior directed nystagmus is uncommon because these eye movements if they occur, typically cancel each other out. [1]
        1. Slow phase is towards involved ear (visible with frenzel lenses or infrared goggles) [1]
        2. Fast/rapid phase is away from involved ear (used to describe nystagmus) [1]
        3. Nystagmus increases when head/gaze turned towards direction of fast phase [1]
        4. After 24 hours, nystagmus in UPVH may only be observed in darkness with infrared light because it can be prevented by visual fixation. [1]

    iv. <u>Ocular Tilt reaction</u>**:** due to unilateral loss of utricular input to vestibular nuclei. [1]
        1. Patient presents with head tilt to involved side, skew deviation, and/or conjugate ocular torsion.

2. Skew deviation is when eye on involved side positioned lower in orbit relative to contralateral eye. [1]
3. Skew deviation often causes diplopia and resolves quickly. [1]
4. Ocular torsion is when the upper pole of both eyes is moves towards the involved side.
5. Conjugate ocular torsion (up to 15 degrees) may resolve more slowly or remain. [1]

v. Postural asymmetries

1. Interruption in VSR produces asymmetries in muscle activities in lower extremities and can lead to imbalance. [1]
2. Lateropulsion posture: an offset of posture towards involved side that occurs during early stages and typically disappears in about a month. [1]

f. **Dynamic symptoms:** typically last longer than static symptoms and require longer vestibular rehabilitation. [1, 13]

i. Common symptoms are vertigo and gaze instability during head movements.
1. Visual or gaze instability with head movements occurs secondary to decreased VOR gain. [13]
2. Specific head movements generally provoke vertigo, and patient may avoid activities due to increased sensitivity to these movements. [13]

ii. Nonvestibular sensory and motor systems can naturally adjust to less functional slower head movements, but requires balanced VOR for stabilization of more functional dynamic movements (e.g. walking, running). [1, 13]

g. **Disequilibrium**: occurs postural instability during static and dynamic movements; can cause falls [1, 13]

i. Generally increases with head movements and when activities are performed at faster speed. For instance, crossing busy street while turning head sideways to look for oncoming traffic. [13]
ii. Disturbances of dynamic VSR are observed as gait ataxia. [1] Patients may ambulate with wider base of support, side-step, drift form one side to another while walking, and decrease in head and trunk rotations. [1]

h. Increased motion sensitivity
i. Difficulty differentiating between exocentric and egocentric motions in crowed or visually busy environments (e.g. large supermarkets) creates imbalance and/or dizziness. [13]
ii. Difficulty with perception of visual vertical orientation under static and dynamic conditions (subjective vertical is strongly tilted towards the involved side). [14]

i. **Physical deconditioning** and general fatigue can occur from prolonged avoidance of activities secondary to fear of reproducing symptoms and falling. [1, 13]

j. **Anxiety and or/depression**: from fear of reoccurrence, not understanding what is occurring, and isolation due to avoidance of activities that provoke symptoms. [1]

4. Treatment goals for patients with UVL

a. It is essential to identify patient's goals and take problem-based approach. [1] Typically a home exercise plan (HEP) is integral part of VR and directing VR towards patient-centered goals may help improve patient compliance. [1]

Common physical therapy goals include:

b. Reduce symptoms of dizziness [1, 13, 15]
c. Increase gaze stability and decrease symptoms during head movements to increase independence with static and dynamic ADLs and during ambulation [1, 13, 15]
d. Decrease disequilibrium: improve postural stability, functional balance and gait stability. [1, 13]
e. Improve cardiovascular condition and allowing increased activity and community participation. [1, 13, 15]

5. Interventions

a. Treatment should begin as early as possible, even if initially tolerable duration of treatment is brief. [1]

b. Interventions should be individualized to the patient's impairments and functional limitations because response to VR is highly specific to the type of exercises performed during training (position of head on body, speed/frequency, target distance, etc). [1, 2, 15]

c. From assessment identify most relevant symptomatic movements/positions and have patient practice these through targeted exercises that are aimed to increase CNS compensation for vestibular pathologies. [2, 15]

d. Patient education

i. Education is critical component of VR for every patient.
ii. It should include explanation of impairment and diagnosis (visuals may help), prognosis and rationale for treatment to manage their functional deficits and symptoms.
iii. Patients must be told that is normal to sometimes experience worse symptoms before getting better, as this is critical for vestibular adaption. [1]
iv. The HEP is a critical component to effective VR, and patients who better understand the rationale behind treatments will be more likely to complete HEP, thus facilitating their recovery. [1]
v. Directing patients to resources such as Vestibular Disorders Association (VEDA) can be helpful. http://vestibular.org/

e. VR exercises

i. Generally includes habituation, adaptation and substitution exercises, postural control and/or repositioning maneuvers.
ii. Patients with UPVH are a heterogeneous patient population, therefore not all above therapies will be appropriate for all patients. [1, 2]
iii. The physiological rationale for each is explained below, followed their application in treatment of impairments and functional limitations. [2, 16]

iv. Adaptation
   1. Repetitive generation of a retinal slip during head movement that creates and error signal in CNS. [1, 4, 13, 17]
   2. With visual fixation on a target during head movements the CNS compensates and decreases the error by stabilizing the visual image on retina by increasing VOR gain. [13]

v. Habituation
   1. Symptom provoking-movement is used to reduce the responsiveness of the CNS to repetitive provocative stimuli and to help re-balance the tonic activity within the vestibular nuclei that contributes to vestibular and visual/somatosensory mismatch. [1, 2, 4, 17]
   2. At first there are only temporary reductions in symptoms, but persistent exposure (weeks to months) allows structural changes to occur and more permanent reduction of dizziness/symptoms experienced with these specific movements. [13]

vi. Sensory Substitution/Compensation
   1. Visual and somatosensory inputs to vestibular nuclei are used instead of dysfunctional vestibular input to assist in postural control. [1, 13]
   2. Substitution may not be available in certain environments where these systems cannot provide reliable inputs (e.g. dark, on foam). [1, 17]

vii. Postural control exercises, falls prevention, relaxation training, re-conditioning exercise, functional/occupation training which all use motor learning principals to change movement behavior and promote safer more balanced movement. [1]

viii. Repositioning maneuvers: BPPV diagnosis and treatment will not be discussed in detail in this module.
   1. Repositioning maneuvers can be incorporated into overall VR plan of care for UPVH if patient has BPPV and goal is to shift vestibular debris. [1, 15]

   2. Generally, canalith repositioning maneuver (CRM) is most effective treatment for BPPV [18], though vestibular rehabilitation (VR) in addition to CRM has been shown to improve gait compared to use of CRM alone. [19] Others include:

   3. **Posterior Canal BPPV**: ~20% of all vertigo, ~90% of BPPV cases. Presents as upbeating or torsional nystagmus; teatment includes canalith repositioning maneuver (CRM) [18], Semont and liberatory maneuver. [15]

   4. **Lateral/Horizontal Canal BPPV:** 3-12% of BPPV. Presents as horizontal nystagmus; an example treatment is Log roll exercise (sidelying with affected ear down, supine, sidelying affected ear up, nose down, and sitting) [15]

   5. **Anterior Canal BPPV**: 2% of BPPV. Presents as downbeating nystagmus with/without torsion; example treatment is the Kim maneuver (deep dix hallpike, supine with head lower 60 degrees below supine) [15]

   6. For more information and helpful illustrations of maneuvers: http://www.dizziness-and-balance.com/disorders/bppv/bppv.html

f. **Gaze stabilization Interventions**: To reduce visual blurring and dizziness when performing tasks that require head movement and visual tracking. [13]

i. VOR 1X and X2 viewing exercises improve gaze stability by increasing VOR gain via adaption. [13, 17]

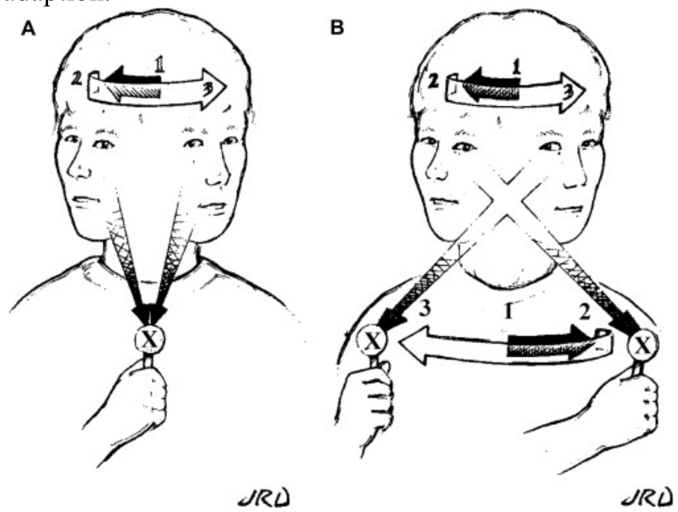

Figure 1.

In: Hall CD, Cox LC. The role of vestibular rehabilitation in the balance disorder patient. *Otolaryngol Clin North Am*. 2009;42:161-9, xi.

1. **VOR 1X** (left picture, A): Patient views a small stationary object while horizontally turning head back and forth to the point where gaze instability or symptoms are induced. [20] Then repeat using vertical head movements.

2. **VOR 2X** (right picture, A): The target moves in the opposite direction to the head rotation. [20] Both horizontal and vertical head rotation with pared target movements are performed.

3. Concentration and focus on task is important, patient should not be distracted by conversation or other activities. [1]

ii. Patient should stop/modify and exercises if symptoms increase significantly on VAS or Borg or significant nausea or vomiting occurs. [1, 6] Symptoms should resolve between 2-15 minutes.

iii. Frequent but short (~1 minute) rest periods during 20-30 minutes of training or changes in context state improve adaptation. It is suggested that consolidation occurs during this period to stimulate adaption to saccade amplitude. [13, 21]

iv. In ACUTE stage of UPVL use 1X and 2X exercises with caution because they can cause severe vertigo, nausea and vomiting. [1] After 1-3 days if acute-stage patients are unable to perform X1 viewing exercises, they perform slow easy head movements and increase visual inputs (e.g. a bright room and curtains open) to start to encourage central adjustments to vestibular system. [1]

v. How to begin and progress VOR 1X and 2X adaption exercises:

1. Duration (primary variable): begin with 1 minute, progress to 2 minutes of each exercise. [13, 17]
2. Frequency: begin 2x a day, progress to 5x a day. [13, 17]

3. Background/distraction: begin with 1 minute with plain background, progress to conflicting full field background (checkerboard), progress outdoors [13, 17]
4. Position: begin in standing if tolerated, if not sitting. Progress to walking forward, backward, step up and down. [13, 17]
5. Speed: begin slowly at at least 2 Hz (because VOR function required for head movements at 2 Hz and faster), progress to faster speed as long as target is in focus and stable. [13]
6. Distance: start with target at 3 feet, progress to target at 8-10 feet. [13]
7. Incorporate ADLs, community or work-related activities or games that require visual tracking or gaze fixation, in the context and environment where they need to perform them (e.g. bouncing and catching a ball off floor, wall or ceiling at various speed and neck positions, walking through grocery store shopping mall). [1, 13]

vi. Recent (2013) study concluded that by targeting only the involved side of UPVH, the VOR gain adaption was possible in only 15 minutes treatment using incremental velocity error signals (10% increase each set) during self-generated head rotations. [22] A laser was used to create a target on wall during active turns towards involved side, subjects performed 10 sets of 30 head impulses (15 to each side) with 30-60 second rest periods between sets. [22]

vii. The use of optokinetic stimulation using checkerboard curtain, disco ball, or computer optokinetic stimulation can improve gaze stability by increasing gain of VOR via habituation. [1] This may not be as effective as head movement combined with a visual stimulus, but may help reduce visual-vestibular mismatch. [1] For a "low tech" computer-based example visit: http://www.youtube.com/watch?v=kAPtu1WTHYc

g. **Intervention for motion-provoked Vertigo, visual Vertigo, motion sensitivity** or dizziness provoked by full field of repetitive moving visual environments or visual patterns. [1, 13]

i. Physical therapist first identifies specific positions or movements that provoke patient's symptoms.

ii. The Motion Sensitivity Quotient can be used to identify symptom-producing positions as well and be used to evaluate intervention effectiveness. [23] The MSQ is valid and reliable assessment where patient rates intensity and duration of dizziness during 16 position changes. [17, 23] (Refer to Supplemental Document 4)

iii. Guidelines for habituation to symptom-producing positions:
1. Avoid movements/positions that produce severe symptoms, if too aggressive with treatment patient may not return or do home exercise plan (HEP). [1]
2. Perform 2-3 repetitions of movements specific to patient (from MSQ, DGI, or assessment) that provoke gaze instability and mild-moderate symptoms. [13, 17]
3. Allow symptoms to subside and wait an additional 30 seconds before next repetition. [13]
4. Perform 2-5 sets daily, can incorporate them into patient's daily routine. (e.g. perform neck diagonals while unloading dishwasher) [1, 13]
5. Progress them by increasing speed and varying position and activity. [13]
6. Typically takes 4 weeks for symptoms to decrease. At 8 weeks exercises can be gradually decreased to 1x/day. [1]

7. Using habituation exercises, 93% of patients with unilateral peripheral vestibular deficits reported successful reduction of symptoms by discharge. [17]
8. If treatment fails, counsel patients about activity or environment modification. [1]

iv. Cawthorne-Cooksey exercises (Refer to Supplemental Document 7)

1. Developed in 1940s; most PTs use select components of these exercises. [1, 15]
2. Include head and total body movements and tasks requiring coordination of eyes and head. [15]
3. Position progression: in bed, to sitting, to standing, to mobility with slow eye movements then progressing to fast. [15]

v. Optokinetic stimulation exercises challenge visuo-vestibular conflict and have been shown to be effective with patients with poor visual preference. [24, 25]

h. Intervention for postural instability

i. Most common complaints are dynamic instability when turning quickly and walking in the dark, on uneven surface, and/or when there is change in light intensities (e.g. opening door to outside, walking through shadows or dark movie theater). [1]

ii. Balance and gait interventions will vary largely based on patient's assessment/patient history/medical diagnosis.[1] The Computerized Dynamic Posturography (CDP), Clinical Test of Sensory Interaction and Balance (CTSIB) or Sensory Organization Test (SOT) can provide useful assessment and evaluation tools.

iii. Substitution exercises are aimed to alter visual, somatosensory and vestibular inputs to recruit substitution of somatosensory and visual inputs and "force use" of remaining vestibular inputs to maintain postural control. [1, 13, 17]

1. <u>Visual variation</u>
    a. Keep eyes closed during activity, introduce full visual field background (conflicting checkerboard), incorporate head movement, decrease lighting, and/or use busy conflicting visual environment or moving stimuli (train moving pass train platform) [1, 13]

2. <u>Somatosensory variation</u>
    a. Varying support surface: conforming (foam, wobble board, trampoline, balance beam), uneven (grass, gravel, ramps) or moving surface (treadmill, escalator) [1, 13]
    b. Changing base of support (feet apart, together, tandem, SLS). [1, 13]

3. Tasks that <u>force use vestibular system</u> (patient who experiences disequilibrium when visual and somatosensory input for balance is poor).
    a. Start by reducing single sensory system and then progress to involving several sensory alterations.
    b. e.g. walking backward, sidestepping braiding performed with eyes closed and marching on foam with eyes open and eyes closed, walking across exercise mat or mattress in dark. [1, 15]
    c. Incorporate head movements in various orientations and dual tasking (ball toss, cognitive tasks) during exercises.

iv. Other balance interventions should be task-oriented and address patient's specific deficits. Some examples provided below.

1. Patient with instability with movement that incorporates head turns during gait (e.g. when shopping for groceries the patient must scan right and left and avoid hitting other shoppers). [1]
    a. Start by walking down hallway while moving head left/right and up/down.
    b. Perform same task while avoid objects placed in walking path.
    c. Perform exercise in area with complex visual environment.

2. Patient with instability when gait is unexpectedly disrupted. [1]
    a. Improve anticipation.
    b. Obstacle course and physical therapist give commands for patient to stop or avoid an obstacle.
    c. Walking or pivoting to right/left or forward/backward upon command.

v. These exercises are to be performed every day for at least 60 minutes (they can be split over several sessions) and for them to be effective, patients should expect to experience mild-moderate symptoms. [15]

vi. Tim Hain, MD provides other excellent examples of interventions for postural instability and balance deficits. Access then at: http://www.dizziness-and-balance.com/treatment/rehab/balance%20exercises.html

i. **Intervention for physical deconditioning** [1, 13]

i. Start regular walking program (start with 15-20 minutes, work up to 30 minutes, 3-5 times a week). [1, 13] Benefits include[1]:
    1. Decreases or prevents deconditioning
    2. Provides realistic balance challenges to patient's CNS

ii. Walking on uneven terrain, walking through shopping mall, crossing the street, going up flights of stairs, riding escalator. [1, 15]

iii. If muscle weakness or lack of flexibility identified in assessment incorporate progressive resistive exercises or flexibility into interventions and try to incorporate into functional activities.

iv. Return to other normal activities as long as they are being safe and are within their tolerance (e.g. golf, tennis, or swimming which should be resumed only with someone supervising). [1, 15]

j. Anxiety/ Depression: screen for psychological disorders and refer or encourage patient to seek support and assistance from health care professionals, family or community members. [1, 26]

6. <u>Most effective form of vestibular rehabilitation?</u>

a. A 2011 Cochrane review that included 27 trials and 1668 patients did not find consistent evidence that form of vestibular rehabilitation superior to another. [2]

b. **Simulator based activities have added benefit**

    i. Optokinetic stimulation
        1. Pavlou el found that customized home-based VR exercise program with optokinetic simulation was more effective in improving dizziness, postural instability and visual vertigo symptoms than home VR program without optokinetic stimuli. [27]
        2. Visual motion DVD, and online video are more economical, clinic friendly and easier to incorporate into HEP compared to "high tech" optokinetic stimulation options. [28]

    ii. Computerized dynamic posturography (CDP)
        1. Rossi-Izquierdo et al found that CDP improved visual and vestibular input and limits of stability compared to optokinetic stimulation in patients with unilateral vestibular hypofunction. [25]
        2. Corna et al found that VR with moving platform may improve balance and functional mobility to greater degree than Cawthrone-Cooksey exercises. [29]

c. **Debate over level of supervision required for VR effectiveness**

    i. All VR typically has HEP component that compliments and builds on VR provided in clinic. [30-32]
    ii. Yarley et al found that VR including education, demonstration and follow up with exercise booklet is better than just HEP booklet alone. [8]
    iii. Minimalistic version of VR can still be effective. [33, 34] Kammerlind et al found no difference between treatment groups receiving 1 VR visit and HEP versus those receiving in clinic VR sessions. [34]
    iv. Many patients need feedback to ensure they are performing exercises correctly and to progress difficulty of exercises. [4]
    v. Pavlou et al found direct positive relationship between supervision, motivation and compliance. [28]
    vi. Supervision may improve patient psychological status and lead to greater development of external locus of control (confidence). [26, 28]

7. **Dosage of VR**
    a. In general, there is no strong evidence for dosage (frequency, intensity, duration) and specificity of vestibular rehab (the combination of compensatory, adaptation, substitution or task-specific) that is more efficious clear secondary to heterogeneity of studies and this patient population. [2]
    b. Generally, suggested that exercises should be performed 3-5 times a day, and duration or number of reps of each should be determined by assessment. [1] Patients should perform all their exercises in the clinic prior to adding to a HEP to assess their response. [1]

8. **Follow up or being accessible** to patients during rehabilitation will improve compliance to HEP, especially for patients who may be concerned about the HEP triggering symptoms. [1]

9. **Re-evaluation** should be completed at subsequent patient visits and HEP progressed so that VR continues to challenge patient. [1]

10. <u>Recovery expectations</u>

a. Recovery and return to prior activities generally can be expected with VR, though 10-30% of reported subjects with UPVH do not improve. [1]
b. In study of 209 patients 75% to 88% of patients demonstrated significant improvement in the outcome measures after VR. [35]
c. Hall et al found a VR model reduced fall risk in patients with UPVH with 77% sensitivity and 90% specificity. [20]

d. Possible factors affecting outcome
   i. Degree of deficit [35]
   ii. Physical co-morbidities [35]
   iii. Psychiatric and psychological factors[34]
   iv. Coping strategies[1]
   v. Etiology of vestibular deficit[1]
   vi. Time from onset[34]
   vii. Medication: can delay or slow recovery; a randomized control trial showed that patients taking medications did not show improvement in symptoms whereas patients performing vestibular exercises did.[1]
   viii. Limit or delay of recovery[1]

e. **Decompensation or relapse** can occur with extreme fatigue, stress, illness, change in medication or prolonged period of inactivity.[1] Frequency of performance of HEP is correlated with sustaining improvement of gait speed at one year. [32] Patients are recommended to return to HEP if relapse occurs and if symptoms persist to return to clinic for re-evaluation.[1]

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