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The assessment of balance and dizziness in the TBI patient

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Abstract.
OBJECTIVES: All degrees of traumatic brain injury (TBI) are associated with balance dysfunction and/or dizziness. The clinician assessing and managing patients with TBI should become familiar with vestibular and non-vestibular causes of dizziness and imbalance, and be able to perform screening tests to determine when referral to a vestibular specialist is warranted. This chapter outlines the clinical pathways to be followed in history-taking, physical examination, and assessment.
INTRODUCTION: Dizziness, vertigo, balance dysfunction and gait ataxia can have their origin in the vestibular system, elsewhere, or be multifactorial. The complex anatomy and physiology of the balance canals, otolithic organs, and vestibular nerves peripherally, and the vestibular nuclei centrally, as well as the neural connections between vestibular, oculomotor, and proprioceptive systems will be covered in clinically pertinent detail.
METHODS: A majority of diagnosis of dizziness/vertigo can be made after a proper history has been obtained. This can be challenging in all dizzy patients, and more so in the TBI patient in whom memory and recall may be impaired. The reader will learn how to use tools such as dizziness questionnaires as well as targeted history taking to elicit the information. Similarly, the addition of a programmatic, targeted physical examination of the dizzy patient will allow the clinician to fine-tune the diagnosis between peripheral and central causes.
RESULTS: Once history and examination have narrowed the diagnostic possibilities, appropriate testing – in the vestibular laboratory and radiologic testing – is indicated. The reader will learn when these tests should be considered, and what the findings will show. Treatment can then be targeted for maximal outcome.
CONCLUSION: Managing TBI is challenging; the addition of dizziness or balance complaints in these individuals makes it even more so. This paper seeks to provide a useful roadmap clinical pathway for assessment of these patients with appropriate and timely referral for treatment.

Keywords: Dizziness, balance disorders, vertigo, gait ataxia, TBI, vestibular dysfunction

1. Introduction

Humans maintain their sense of balance and orientation to gravity, both static and dynamic, by coordination of inputs from the vestibular, visual, and proprioceptive systems, which coordination takes place in the brainstem [1]. Of these, the vestibular system has the overriding input, and damage to either right or left vestibular input (or both) can have long-lasting sequelae, particularly in patients with additional problems with vision or proprioception, and/or with brain processing issues. Each vestibular system consists of five distinct end organs: three semicircular canals that are sensitive to angular acceleration as seen with head rotation; and two otolithic organs that are sensitive to linear accelerations and gravity [2]. Each semicircular canal is perpendicular to the others on the ipsilateral side, and coplanar with the contralateral canals. Each otolithic

organ has a curvilinear shape essentially at right angles to each other and coplanar with the other side. The neurosensory element of each semicircular canal, the crista and cupula, is perpendicular to the lie of the canal; the neurosensory element of the otolithic organs, the macule, is oriented around a central dividing line, the striola, with the hair cells oriented either toward the line in the utricle, or away in the saccule. Hair cell depolarization and hyperpolarization cause excitatory and inhibitory fibers to fire, with electricity carried along the superior and inferior vestibular nerves to the brainstem vestibular nuclei. Chapter 1 has reviewed the central neuro-anatomy in detail.

With a single, unilateral vestibular insult, such as viral labyrinthitis, the patient will experience severe vertigo, generally accompanied by nausea and/or vomiting, but this will be self-limited, will respond to supportive measures, and will resolve fully in a relatively short time period. This is because the other labyrinth, the visual and the proprioceptive inputs 'take over' and the brain response is recalibrated. This process can be delayed with the excessive use of vestibular suppressants beyond the acute vertiginous phase in these individuals, thereby suppressing the CNS response and recovery. The utility of the visual and proprioceptive systems to overcome inaccurate vestibular information is seen commonly. Visual preference is used when a ballet dancer performs several pirouettes in a row and maintains themselves on a toe shoe by fixating their vision on a single point at the beginning of each 360 degree turn. Patients with dizziness and vertigo will experience significant symptomatic relief if they sit up in an arm chair with full back, leg, and arm support with their eyes open and focused on a single non-moving point.

Traumatic brain injury causes loss of horizontal semicircular canal function and postural instability that is related directly to the head injury [3]. Blast injury can have resultant effects on the peripheral and/or central vestibular system. Difficulty recovering from vestibular injury is seen when there is more than one site of injury. Grossly, there are six inputs to the brainstem: 2 inner ears, 2 eyes, and 2 sides of the muscles down the back, legs and soles of the feet. Injury to one is usually recovered in a straightforward manner. However, injury to two or more, as is often seen in TBI, poses a much more complex problem. In the non-TBI patient with cataracts who sprains their ankle and then develops a serous (viral) labyrinthitis, recovery is very difficult as the other systems cannot be used to their full extent to overcome the acute vestibular injury. Give that patient a TBI that impairs communication, memory, and central processing, and the recovery is much more prolonged and frustrating.

The crux of making an accurate assessment of dizziness and balance lies in obtaining a clear history from the patient [4]. The patient will tell you the diagnosis, if you let them, and if you guide them through the tale. The word 'dizzy' means many things to many people. It is important that the clinician be able to help the patient distinguish between spinning vertigo, lightheadedness, sensation of impending loss of consciousness, gait imbalance, and falls. Some of the frustration for patients with imbalance issues is the number of times they have to engage with various medical professionals to recount their stories and obtain a diagnosis and treatment plan. Although this is obviously frustrating for all patients, individuals suffering from even mild TBI have particular communication and memory issues which may cause them to fail to convey their histories thoroughly, accurately, or in a 'logical' (to the examiner) manner.

There are some straightforward solutions to this problem. One, it is strongly recommended by most balance professionals that the patient presenting for evaluation of any type of 'dizziness' is asked to fill out a questionnaire, ideally well before their appointment time, so that they can focus on the questions and answer them thoroughly. The questionnaire helps the patient go through the various types of 'dizziness' as described above, and also asks about attendant symptoms such as headache, nausea, vomiting, anxiety response, hearing loss, tinnitus, and other symptoms more attributable to the central nervous system. Additionally, patients are asked to think about aggravating and alleviating factors. Having the patient fill this form out and bring it in to the visit, and incorporating it both into the medical record and into the assessment portion of the visit is beneficial in streamlining the process and targeting diagnosis. A sample 'dizziness questionnaire' is appended to this chapter.

A second, more elegant but more labor-intensive, solution to the repetitive story-telling by the patient is as follows. Establishment of a multidisciplinary team wherein the patient has to tell their story only once, to the entire group, rather than to each individual healthcare provider separately, greatly enhances both patient and provider experience in these complex dizzy cases. The Vestibular Disorders Evaluation Center established in 2010 at the James J Peters Veterans Administration Medical Center in the Bronx, NY is staffed by a neurotologist, an audiologist with particular interest and expertise in the vestibular system, and a vestibular

physical rehabilitation therapist. The VDEC is based on a similar program at Walter Reed Army Medical Center, [5] but serves veterans of all ages as opposed to active-duty or recently-discharged military personnel. With the dramatic rise in civilian TBI and complex dizziness disorders in non-military persons, a similar program is being organized by the author at the New York Head and Neck Institute at Lenox Hill Hospital in Manhattan, NY.

2. History

The patient will tell you the diagnosis, if you let them, and if you listen. For peripheral vertiginous disorders, look for 'million dollar clues' that will lead you directly to the diagnosis. These include the following. "I get dizzy for a few seconds just rolling in bed." This is a hallmark complaint for benign paroxysmal positional vertigo (BPPV), the most common single entity diagnosis in a dizzy clinic, and seen very commonly in TBI. BPPV is surprising seen in 9% of all patients over 65 seeing a physician for other problems, [6] so it should always be considered in the differential diagnosis. It may accompany other types of vestibular disorders, and should be 'cleared' with office-based treatments as quickly as possible to allow for proper identification and treatment of the other problems. "I am very light sensitive during my dizzy spells." This is frequently seen in Migraine Associated Dizziness (MAD), which is also called Atypical Migraine or Migraine-Associated Vertigo (MAV). Another complaint often heard in MAD/MAV is that patients have great difficulty in visually-stimulating surroundings, such as in a supermarket or superstore with fluorescent lights, high ceilings and long, multiply-stocked shelves, or watching a 3-D or action movie, or scrolling on the computer. Migraine is frequent following TBI. "One ear feels like it's going to explode when I am dizzy." Aural (ear) fullness and roaring tinnitus before episodic spinning vertigo are the sine qua non of Meniere's Disease (MD), which is a syndrome of endolymphatic hydrops – increased pressure in the innermost fluid layer in the inner ear. Triggers for MD include salt, caffeine, dehydration, and emotional stress. A newer member of the 'million dollar clue club' is "sounds make my vision jump or blur." This is seen in the disorder of superior semicircular canal dehiscence (SSCD), which is also eponymously named 'Minor's syndrome.' Although this is an unusual inner ear diagnosis, we now have both clinical and radiographic testing that can confirm the presence of SSCD, and treatments can be offered to ameliorate this problem.

Central vertigo may present with less discrete symptoms and is often not episodic or fatigable. It can be associated with other CNS complaints including paresthesias and limb weakness. Metabolically induced vertigo presents episodically, but frequently related to missed meals, missed medications, etc.

In particular, when evaluating the TBI patient with dizziness, the interview should be structured so that neither the patient nor the examiner will miss important historical elements. The presence of a knowledgeable third party in the room is very helpful, particularly when there are memory or recall issues as part of the TBI. It is helpful for the examiner to have the pre-completed Dizziness Questionnaire so that the questions can flow smoothly. Questions that are important in TBI include:

1. Details of the event causing the TBI
    a. Military evidence shows that there are significantly different sequelae from blunt head injuries versus blast trauma, with blast injuries causing more and more persistent damage overall [7].
2. Description of dizziness
    a. Type (dizzy/vertigo/lightheaded/etc.) – detailed on Dizziness Questionnaire
    b. Initial onset date and details of first episode
    c. Constant vs. episodic
        - If episodic, frequency and duration of episodes. This information is helpful both at the initial evaluation as well as after ongoing care, in order to assess degree of improvement in an objective manner.
    d. Is dizziness getting better, worse, or staying the same?
    e. Provoking factors
    f. Alleviating factors
    g. Associated symptoms
        - Otologic
        - Non-otologic, including visual, neurologic, metabolic
    h. Medications given for dizziness and their effects/side effects
3. Association of headache? If yes, obtain detailed headache history. A history of dizziness and/or migraines in the family is of interest in both MD and MAD.
4. Past medical and surgical history, family history, medications (legal, illegal, over-the-counter, and herbal) – current and past use.

5. Dietary history – with particular attention to salt/caffeine/nicotine/alcohol/hydration. There is less clear evidence regarding sugar or carbohydrate intake; however, celiac disease can present with metabolic type dizziness and must be kept in that differential.
6. History of consumption of ototoxic agents, whether medicinal or recreational. There are 'hidden' ototoxins such as quinine, which is found in tonic water, and aspirin, which is found in many OTC remedies for other problems.

The Dizziness Handicap Inventory (DHI) is a validated tool of self-reported dizziness-related disability [8]. It is a helpful tool in that the patient fills it out themselves, and then it is graded by the examiner. The 25 question items are subgrouped into three content domains representing functional, emotional, and physical aspects of dizziness and unsteadiness. It has high inter-test reliability and validity and can be used as an initial assessment measure as well as an outcomes tool.

3. Physical examination

After the history has been obtained, a targeted dizziness examination is indicated. This examination is separate from any other type of physical exam that the healthcare provider is conducting – general medical exam, ENT exam, PT exam, etc. J. Goebel has detailed 'The 10 Minute Examination of the Dizzy Patient,' which organizes the steps and explains what they mean [9]. In addition to those tests, evaluation of eye vergence is very helpful. The points of the examination are detailed in Table 1, which is adapted from Goebel, J., 10 Minute Examination of the Dizzy patient.

4. Targeted office-based testing

Nontargeted vestibular testing is not as effective as testing that is personalized to the patient's presentation and examination findings. Vestibular function tests as a whole are NOT good "rule out" tests. They carry good specificity but only modest sensitivity. In evaluating the dizzy patient, it is prudent to use the otovestibular testing laboratory to quantify deficits you already suspect, and/or as a baseline measure before ablative intervention or rehabilitation is undertaken. Patient preparation before testing is important, as consumption of central nervous system sedating agents including many psychotropic medications, sleeping pills, meclizine and alcohol within 24 to 72 hours of the test (based on the lab and the patient) may result in non-useful information, particularly from the VNG, Rotary Chair, and Posture Platform. The same holds for CNS stimulants such as caffeine and Ritalin, etc. Patients who are nervous are advised to bring an escort as well as their medication, which they are allowed to take immediately after the test is completed.

As the cochlea Is the anterior portion of the inner ear, an audiogram is usually indicated, looking for hearing loss in general, its type and frequency information, as well as for asymmetry between ears or with word recognition. Either of those may indicate a retrocochlear lesion along the eighth cranial nerve or auditory brainstem. Otoacoustic emission testing reveals functionality of type II auditory hair cells and its utility in diagnosis of inner ear disorders is being discovered. Auditory brainstem response testing (ABR, also known as BEAR or BERA testing) is indicated in cases of asymmetric sensorineural hearing loss or otherwise unexplained hearing loss and is considered part of the retrocochlear testing protocol. If the hearing level is poorer than 80 decibels, ABR cannot be performed on that ear. Serial audiometry is also a possible option for asymmetric SNHL [10]. Electrocochleography (ECoG) is often helpful if the suspected diagnosis is MD, but can be normal in up to 25% of MD patients. Vestibular evoked myogenic potential (VEMP) testing can be performed using air conduction (AC) and bone conduction (BC) click stimuli and can be measured at the neck (cervical) or extra-ocular muscles (ocular). AC cervical VEMP- threshold versus amplitude measurements look for sacculocollic function, which is a function of the inferior vestibular nerve (IVN), while BC ocular VEMP- threshold vs. amplitude measurements look for utriculo-ocular function, reflecting the superior vestibular nerve (SVN).

The video-electronystagmography (VNG) evaluation is a very helpful tool in the evaluation of the dizzy patient. It assess both central and peripheral (horizontal semicircular canal (SCC)) inputs. The caloric portion of the VNG is performed using warm, cool, and, when needed, ice air or water infusions into the ear canals. The VNG also consists of a number of separate tests including positional testing, saccade testing, smooth pursuit, and optokinetic testing with eye movement recordings and/or videooculography. Central oculomotor tests are usually normal in peripheral vestibular disease but are an

Table 1
Targeted examination of the dizzy patient

Examination	Action, requirements	Normal	Abnormal
Spontaneous nystagmus	Target fixation, neutral gaze, Frenzel goggles	No nystagmus or excessive saccades	PERIPHERAL: Jerk nystagmus: Direction-fixed, increases with Frenzels CENTRAL: Direction-changing, increases with fixation, Pendular (may also be congenital)
Gaze nystagmus	Hold eccentric gaze at 20–30 degrees for 10 seconds in horizontal and vertical plane	Physiologic end gaze or No nystagmus	CENTRAL: Jerk nystagmus in direction of gaze (flocculus, drugs, alcohol) Downbeat Nystagmus in lateral gaze (Arnold Chiari, midline cerebellum)
Smooth pursuit	Track finger moving 60 degrees through 60 degree horizontal and vertical arc	Smooth pursuit eye movements	CENTRAL: Saccadic pursuit (cerebellum, brainstem, parietal lobe) OTHER: Irregular tracking – related to age, acuity, medications, attention
Saccades	Alternate gaze between fingers	Rapid, accurate, conjugate eye movements	CENTRAL: Over/under-shoots (Dorsal vermis, fastigial nuclei) Slow saccades (brainstem) Late saccades (frontal lobe, brainstem, Parkinson's syndrome) Disconjugate (MLF syndrome)
Vergence test	Hold finger or pen at 1 foot from nose; bring in towards face.	Normal near-point of convergence break is 5 cm and recovery is-7 cm from nose [23]	OCULOMOTOR abnormality: no convergence break or recovery in normative range
Head thrust test	Thrust head 20–30 degrees while fixating on a target	No loss of fixation	PERIPHERAL: refixation saccade (loss of visual-ocular reflex – VOR)
Headshake test	Shake head for 20 seconds at 2 Hz (horizontal and vertical) with eyes closed, then open eyes (with Frenzel goggles on) and observe	No nystagmus	PERIPHERAL: Unidirectional nystagmus in plane of headshake (toward intact side, except in Endolymphatic Hydrops (MD)) CENTRAL: Vertical nystagmus after horizontal headshake
Dynamic visual acuity test	Have patient read eye chart with head still, then with 0.2 Hz headshake	Less than 3 line drop in acuity with headshake	PERIPHERAL: More than 3 line drop in acuity with headshake (bilateral VOR loss)
Fixation suppression test	Have subject fixate on own thumb while rotating body in exam chair	No nystagmus	CENTRAL: nystagmus in direction of rotation (flocculus)
Position tests – aka dix-hallpike tests	Place head in left and right Hallpike position and left and right lateral position, and supine	No nystagmus	BPPV: Torsional, geotropic nystagmus with upbeat and ageotropic horizontal components (downmost posterior SCC). BPPV: Horizontal paroxysmal nystagmus (downmost lateral SCC) PERIPHERAL/CENTRAL: Horizontal sustained nystagmus
Aural pressure/sound Tests	Stimulate ear with either positive pressure, loud sound, or mastoid vibration	No nystagmus	PERIPHERAL: Upward deviation with ipsilateral eye torsion toward nose or downbeating nystagmus = SSCD. Horizontal nystagmus = Perilymph Fistula.
Cerebellar limb tests	Finger-to-nose Heel-shin Rapid alternating motion	Accurate movements	CENTRAL: dysmetria, dysdiadochokinesia (lateral lobe cerebellum)

Table 1
(*continued*)

Examination	Action, requirements	Normal	Abnormal
Somatosensation tests	128 Hz tuning fork to lateral malleolus (ankle)	Vibration sense present	Loss of vibration sense
	Move great toe up/down	Correctly identifies action	Incorrectly identifies action Important in diagnosis of SOMATOSENSORY component of dysequilibrium
Posture tests	Romberg, Sharpened Romberg, Eyes closed standing on foam	Minimal sway; no falls	VARIABLE/APHYSIOLOGIC: Excessive sway, falls on Romberg PERIPHERAL/MIXED/APHYSIOLOGIC: Falls on foam, eyes closed
Gait tests	Observation of gait Fukuda step test	Normal gait <45 degree rotation on Fukuda step test	Wide based, ataxic: Parkinsonian, musculoskeletal gait >45 rotation on Fukuda test

important indication of brainstem/cerebellar function. Unilateral vestibular caloric loss is the most significant indicator of peripheral loss. In cases of bilateral caloric weakness, ice water and a prone position may be used to bring out the information from the stronger side. The spontaneous recording capability of VNG may be used for miscellaneous tests, such as post-headshake/vibration/hyperventilation induced nystagmus, and pressure induced eye movements.

Rotary Chair (RC) testing is an elegant means in which to assess vestibular function in patients who cannot tolerate even air caloric irrigations, or who have significant bilateral caloric weaknesses, or complex findings on other tests. RC platforms are not common, and require specially skilled audiologists and otolaryngologists to interpret the findings correctly. RC evaluation is indicated in suspected bilateral vestibular loss, poor unilateral compensation, head trauma, and central vestibular and/or oculomotor disease. RC is able to test both the lateral SCC using broad frequency sinusoidal testing looking for gain, phase and asymmetry, and step velocity for time constant, as well as the utricle by using eccentric rotation for dynamic subjective visual vertical testing.

Computerized Dynamic Posturography (CDP) evaluation is also a helpful tool to assess integration of visual, vestibular and somatosensory cues for stance using the sensory organization test battery (SOT), and for identification of impaired motor latency responses (MLR) in suspected neuropathy or CNS disease. It is also very helpful in cases of suspected secondary gain or malingering, to check for physiology and consistency of postural responses. Additionally, it is a good tool to assess outcomes of treatment by measuring SOT and MLR at different points in time.

5. Targeted radiology testing

As vestibular studies should be targeted to the patient and the presentation, so should radiologic testing of the dizzy patient, with or without TBI. Generally, the patient has had at least one brain study, usually a CT at the time of the injury and an MRI in a more delayed fashion. If there is asymmetry seen on testing, including on audiogram, the patient with TBI should undergo a targeted MRI of the brainstem and internal auditory canals with and without gadolinium-DTPA enhancement to look for tumors along the eighth nerve (vestibular schwannomas, formerly referred to as acoustic neuromas), as well as evidence of cochlear or vestibular concussions or other lesions along that pathway. If MRI is not possible, but asymmetry is present, a contrast-enhanced CT scan of the temporal bones and brainstem is a reasonable alternative. If that, too, is not possible, ABR or, at the least, serial audiometry to look for progression, can be offered, as discussed above.

Computerized tomography scanning (CT) of the temporal bones is indicated in cases of suspected Superior Semicircular Canal Dehiscence (SSCD), skull base fracture, and in cases of asymmetrical findings where MRI cannot be performed. Techniques for obtaining the correct images to rule in or out these pathologies are detailed and well-defined, and the neuroradiologist should be apprised of the clinical question so that the study can be tailored appropriately.

Magnetic resonance angiography or CT angiography is indicated in certain cases of pulsatile tinnitus associated with dizziness. In patients who have had TBI, the index of suspicion for secondary or delayed vascular anomalies should exist and this type of imaging may be considered.

What must be kept in mind is that patients with TBI who are dizzy have already had a number of tests and that it is exhausting for them to keep going back for more tests. Reviewing the past tests and ordering only the ones that may help at this point is especially important in this population of vulnerable individuals. MR scanners are very noisy and can be horrid for patients in whom hyperacusis (sensitivity to loud sounds) is an issue, as well as for those patients with claustrophobia. Adding an anxiolytic for the MR study may result in negative repercussions from the medication. These are discussions to be had in detail with the patient and their companion before deciding on further testing.

6. Treatment

Once the most likely diagnosis or diagnoses has been established, and confounding factors have been taken into consideration, targeted treatment can be instituted. A summary of some available treatments is provided in Fig. 1 [11]. Medical treatment may include antimigraine medications, diuretics if the problem is endolymphatic hydrops, vasodilators, antihistamines, and other agents as indicated [12, 13]. Severe, acute vertigo may be treated with systemic corticosteroids, despite lack of convincing data regarding efficacy [14]. The use of vestibular suppressants such as meclizine or diazepam is generally limited to acute-phase treatment of disabling vertigo. Chronic use of vestibular suppressants in an ongoing manner is to be avoided, as this will suppress the central nervous system response and impair recovery [15]. The same holds true for anti-emetic agents; they are very useful in acute vertigo, but not indicated for long-term use in general. An important aspect of medical therapy for dizziness and TBI includes evaluation of medications that have been prescribed for the brain injury, and eliminating those that may result in balance disturbance [16, 17]. Invasive medical treatments such as intratympanic injection of steroids or aminoglycosides can result in long-lasting relief in a significant number of patients. Surgical treatment is indicated in cases of disabling dizziness and vertigo, when medical treatments are inadequate, such as in perilymphatic fistula, endolymphatic hydrops, unilateral vestibular hypofunction, and superior semicircular canal dehiscence.

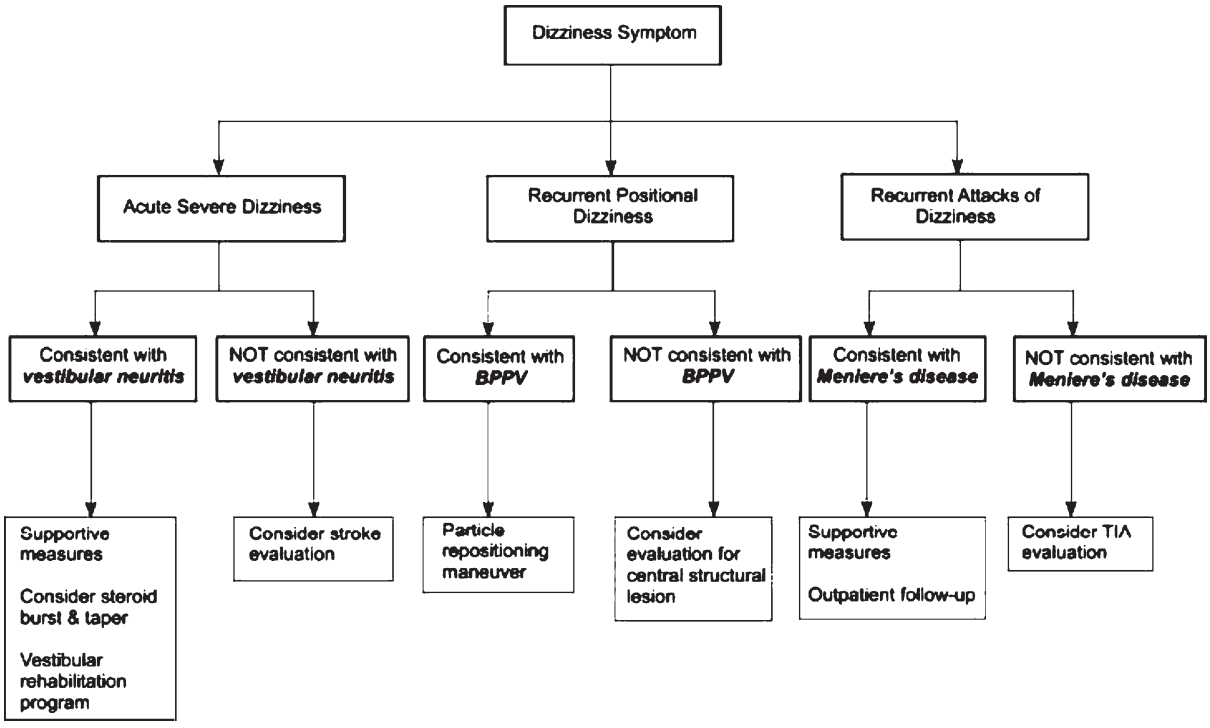

Fig. 1. Dizziness symptoms and Targeted treatments. From: Kerber KA. Vertigo and dizziness in the Emergency Department. Emerg Med Clin North Am 2009 Feb;27(11):39-viii.

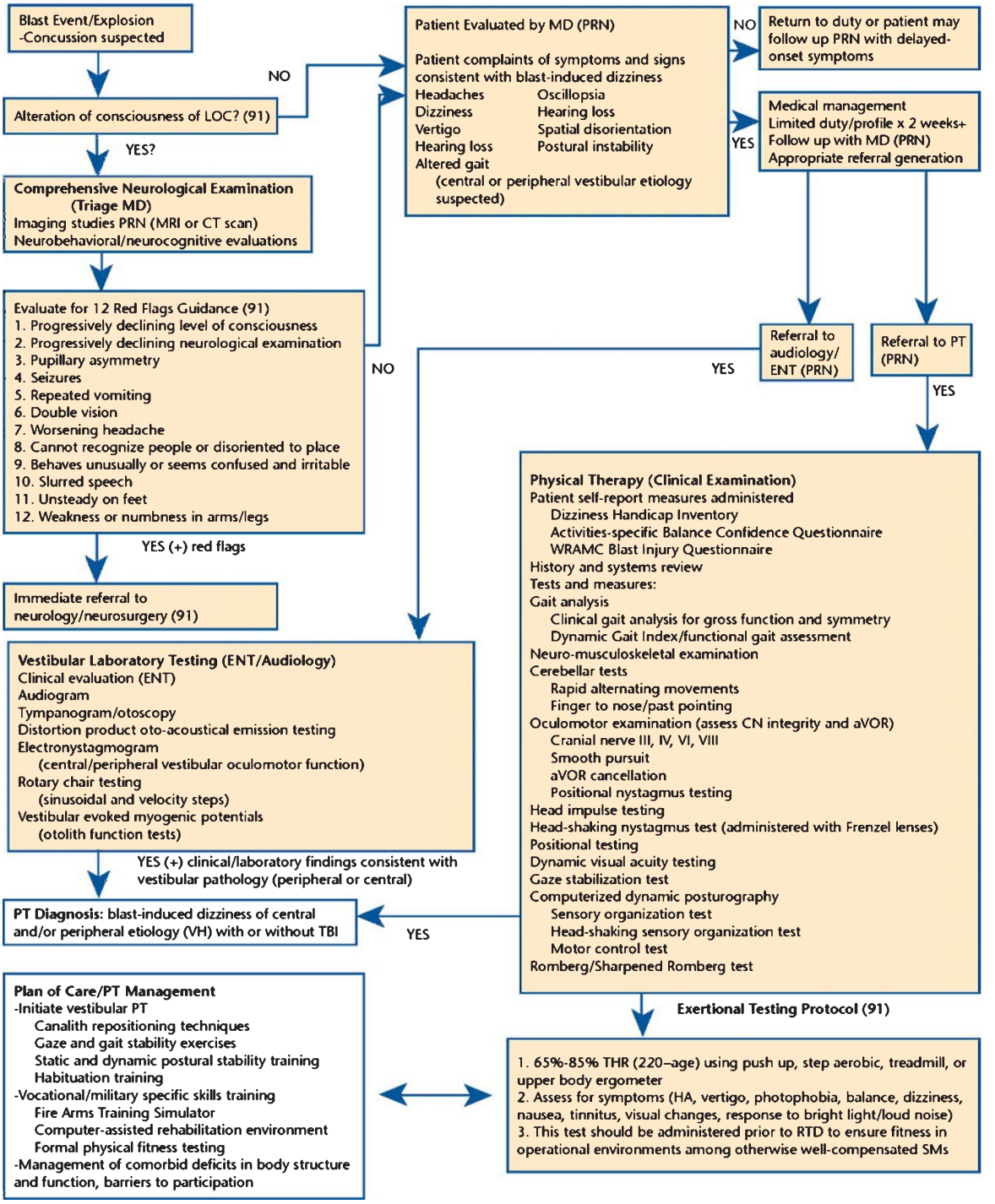

Fig. 2. Walter Reed Army Medical Center Evaluation and Treatment Algorithm for Patients with Blast Injury TBI and Vestibular Dysfunction. Reproduced from Scherer MR, Shubert MC. Traumatic brain injury and vestibular pathology as a comorbidity after blast exposure. Physical Therapy. Sep 2009;89(9):980-992. LOC = loss of consciousness; PRN = as needed; RTD = return to duty; MRI = magnetic resonance imaging; CT = computed tomography; ENT = ear, nose, throat (otolaryngology physician); PT = physical therapy; PCM = primary care provider; VH = vestibular hypofunction; ED = emergency department; SM = service member; TBI = traumatic brain injury; THR = target heart rate; WRAMC = Walter Reed Army Medical Center; CN = cranial nerve; aVOR = angular vestibule-ocular reflex.

The mainstay of treatment for balance dysfunction is vestibular physical therapy, which helps to restore core balance sense, often by relying on visual cues [18–20]. Systematic review reveals moderate to strong evidence of the effectiveness of vestibular PT for unilateral peripheral vestibular dysfunction [21]. Benign paroxysmal positional vertigo (BPPV) should be looked for, and treated with canalith repositioning procedures when it is found. Although active Meniere's disease does not respond to vestibular PT, patients in the chronic form of the disease who have balance or gait issues do well with that treatment modality. The same is true for a number of unilateral peripheral vestibular disorders. Central vestibular disorders are more difficult to treat, but patients in these conditions can respond well to vestibular PT, particularly as their comorbidities come under control. A different type of physical therapy that benefits TBI patients who have vergence dysfunctions, often seen with TBI and migraine, is vision therapy, which retrains the eye muscles to improve functionality in three-dimensional space [22]. Figure 2 summarizes the Walter Reed Army Medical Center algorithm.

7. Summary

Evaluation of any patient with complex dizziness is challenging. Evaluation of the patient with traumatic brain injury and dizziness is much more so. Keeping the history as thorough as possible without tiring the patient through unnecessary repetition is very important. Having them fill out the questionnaire in their own home at their own pace and bringing that in to review with you is an excellent way to minimize missed information, and to empower the patient to speak on their own behalf. The presence of a knowledgeable companion for the patient during office visits has been shown to be helpful in general; [23] it is particularly so in cases of TBI when recall, both of the patient's history as well as what was discussed or planned during the current office visit, may be difficult. If the physician has the availability of a team approach (such as the VDEC described above), we have shown that it cuts down the number of visits and number of repetition of history needed by more than half and streamlines the path to diagnosis and institution of treatment.

Patients with traumatic brain injury find themselves isolated by their injuries and by the change in brain processing that affects all parts of their daily lives. Addition of dizziness, 'the invisible affliction,' to this can break down any existing coping mechanisms. BPPV is frequently seen after TBI, both in the immediate period and in a delayed fashion. The clinician should test for and clear the BPPV and then proceed to further vestibular assessment. If oculomotor abnormalities are identified, particularly vergence issues that cause difficulty in processing visual movement in the Z plane (near-far), targeted vision treatment for that is indicated. Targeting further testing and minimizing doctors' visits also enhance recovery in these patients. If a vestibular team is not available on site, close communication with treating physicians and other healthcare providers, to prevent duplication or errors in testing, medication, services, etc. as well as to overcome barriers to reaching care, is very important in all patients but particularly in the subset of dizzy patients with TBI.

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References

[1] Uchino, Y., & Kushiro, K. (2011). Differences between otolith- and semicircular canal-activated neural circuitry in the vestibular system. *Neurosci Res*, *7*(14), 315-327.

[2] Rutka, J. A. (2004). Physiology of the vestibular system. In: P. S. Roland, & J. A. Rutka (eds). *Ototoxicity* (pp. 24-25). London: BC Decker.

[3] Akin, F. W., & Murnane, O. D. (2011). Head injury and blast exposure: Vestibular consequences. *Otolaryngol Clin North Am*, *44*(2), 323-34, viii.

[4] Rubin, D. I., & Cheshire, W. P. (2011). Evaluation of "dizziness" in the neurology office. *Semin Neurol*, *31*(1), 29-41.

[5] Scherer, M. R., & Shubert, M. C. (2009). Traumatic brain injury and vestibular pathology as a comorbidity after blast exposure. *Physical Therapy*, *89*(9), 980-992.

[6] Oghalai, J. S., Manolidis, S., Barth, J. L., Stewart, M. G., & Jenkins, H. A. (2000). Unrecognized benign paroxysmal positional vertigo in elderly patients. *Otolaryngol Head Neck Surg*, *122*(5), 630-634.

[7] Hoffer, M. E., Balough, B. J., & Gottshall, K. R. (2007). Post-traumatic balance disorders. *Int Tinnitus J*, *13*(1), 69-72.

[8] Jacobson, G. P., & Newman, C. W. (1990). The development of the Dizziness Handicap Inventory. *Arch Otolaryngol Head Neck Surg*, *116*(4), 424-427.

[9] Goebel, J. (2001). The 10-minute examination of the dizzy patient. *Semin Neurol*, *21*(4), 391-398.
[10] Stachler, R., Chandrasekhar, S. S., et al. (2012). Clinical practice guideline: Sudden hearing loss. *Otolaryngol Head Neck Surg*, *146*(3 Suppl), S1-S35.
[11] Kerber, K. A. (2009). Vertigo and dizziness in the emergency department. *Emerg Med Clin North Am*, *27*(11), 39-viii.
[12] Huppert, D., Strupp, M., Muckler, H., & Brandt, T. (2011). Which medication do I need to manage dizzy patients? *Acta Otolaryngol*, *131*(3), 228-241.
[13] Hain, T. C., & Uddin, M. (2003). Pharmacologic treatment of vertigo. *CNS Drugs*, *17*(2), 85-100.
[14] Fishman, J. M., Burgess, C., & Waddell, A. (2011). Corticosteroids for the treatment of idiopathic acute vestibular dysfunction (vestibular neuritis). *Cochrane Database Syst Rev*, *5*, CD008607.
[15] Bronstein, A.M., & Lempert, T. (2010). Management of the patient with chronic dizziness. *Restor Neurol Neurosci*, 28(1), 83-90.
[16] Han, M. H., Craig, S. B., Rutner, D., Kapoor, N., Ciuffreda, K. J., & Suchoff, I. B. (2008). Medications prescribed to brain injury patients: A retrospective analysis. *Optometry*, *79*(5), 252-258.
[17] Cianfrone, G., Pentangelo, D., Cianfrone, E., Mazzei, F., Turchetta, R., Orlando, M. P., & Altissimi, G. (2011). Pharmacological drugs inducing ototoxicity, vestibular symptoms and tinnitus: A reasoned and updated guide. *Eur Rev Med Pharmacol Sci*, *15*(6), 601-636.
[18] Harris, L. R., Jenkin, M., Dyde, R. T., & Jenkin, H. (2011). Enhancing visual cues to orientation: Suggestions for space travelers and the elderly. *Prog Brain Res*, *191*, 133-142.
[19] Hain, T. C. (2011). Neurophysiology of vestibular rehabilitation. *NeuroRehabilitation*, *29*(2), 127-141.
[20] King, W. M., & Shanidze, N. (2011). Anticipatory eye movements stabilize gaze during self-generated head movements. *Ann NY Acad Sci*, *1233*, 219-225.
[21] Hillier, S. L., & McDonnell, M. (2011). Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. *Cochrane Database Syst Rev*, *2*, CD005397.
[22] Ciuffreda, K. J., Rutner, D., Kapoor, N., Suchoff, I. B., Craig, S., & Han, M. E. (2008). Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. *Optometry*, *79*(1), 18-22.
[23] Wolff, J. L., Boyd, C. M., Gitlin, L. N., Bruce, M. L., & Roter, D. L. (2012). Going it together: Persistence of older adults' accompaniment to physician visits by a family companion. *J Am Geriatr Soc*, *60*(1), 106-112.