Traumatic Brain Injury and Vestibular Pathology as a Comorbidity After Blast Exposure

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Blasts or explosions are the most common mechanisms of injury in modern warfare. Traumatic brain injury (TBI) is a frequent consequence of exposure to such attacks. Although the management of orthopedic, integumentary, neurocognitive, and neurobehavioral sequelae in survivors of blasts has been described in the literature, less attention has been paid to the physical therapist examination and care of people with dizziness and blast-induced TBI (BITBI). Dizziness is a common clinical finding in people with BITBI; however, many US military service members who have been exposed to blasts and who are returning from Iraq and Afghanistan also complain of vertigo, gaze instability, motion intolerance, and other symptoms consistent with peripheral vestibular pathology. To date, few studies have addressed such "vestibular" complaints in service members injured by blasts. Given the demonstrated efficacy of treating the signs and symptoms associated with vestibular pathology, vestibular rehabilitation may have important implications for the successful care of service members who have been injured by blasts and who are complaining of vertigo or other symptoms consistent with vestibular pathology. In addition, there is a great need to build consensus on the clinical best practices for the assessment and management of BITBI and blast-related dizziness. The purpose of this review is to summarize the findings of clinicians and scientists conducting research on the effects of blasts with the aims of defining the scope of the problem, describing and characterizing the effects of blasts, reviewing relevant patients' characteristics and sensorimotor deficits associated with BITBI, and suggesting clinical best practices for the rehabilitation of BITBI and blast-related dizziness.

The risk of blast exposure to deployed US military service members is significant. Injury tterns in survivors of blasts are typlly complex and characterized by ltisystem involvement and varis degrees of severity.[1] Such patns may include orthopedic uma, limb loss, visual impairents, burns, and posttraumatic ess disorder.[2-8] Improvements in dy armor, vehicle-hardening meares, and advances in battlefield dicine have led to dramatic reducns in mortality rates for service mbers.[5,9,10] As a result, service mbers are surviving injuries that uld have been fatal a decade ago. nversely, many service members urn home with multisystem paology (ie, polytrauma) and signifint rehabilitation needs.

plosive attacks account for a ger percentage of casualties in curt conflicts than in other recent US nflicts.[9] Traumatic brain injury BI), which often results from blast posure, has been described as the gnature injury" of the wars in Iraq d Afghanistan.[8-11] The Departent of Defense (DOD) and the Dese Veterans Brain Injury Center imate that as many as two thirds medical evacuations from Iraq and ghanistan can be attributed to st exposure.[10] Neurologic pathol- as a result of blast exposure is mmon; 32% of service members wounded in war and evacuated to the Walter Reed Army Medical Center since January 2003 have been diagnosed with TBI.[12] The Defense Veterans Brain Injury Center has provided care for nearly 8,000 service members with TBI in support of Operation Iraqi Freedom (Iraq) and Operation Enduring Freedom (Afghanistan); however, this figure represents only service members who sustained trauma significant enough to require medical evacuation and does not reflect service members who were exposed to blasts but were able to return to duty. Despite recent advances in the battlefield diagnosis of TBI, mild TBI (mTBI) is difficult to diagnose in the war zone, and the condition likely is underreported.[10,13]

Although the multiple effects associated with blast injuries may inflict a wide range of pathologies, impairments, functional limitations, and disabilities in injured survivors, dizziness and vertigo are common symptoms in patients with blast-induced TBI (BITBI). The medical literature includes several perspective pieces, case studies, and epidemiological studies documenting "vestibular" symptoms in patients who have been exposed to blasts. In addition to nonspecific complaints of dizziness or unsteadiness, complaints of vertigo and oscillopsia, typically recognized as being more specific to vestibular pathology, have been reported.[5,14-24] Given the complexities of treating patients with polytrauma, it is essential that any vestibular complaints be assessed and managed in an efficient manner with the goal of providing an optimal return to activities of daily living, quality of life, and return to duty.[25,26]

From this point forward, we consider "dizziness" to be an imprecise term indicating light-headedness or a feeling that one is going to fall; it is not necessarily specific to vestibular involvement. Vertigo is considered to be an illusion of movement, typically perceived as spinning, and commonly indicates vestibular pathology. Oscillopsia is the perception that objects known to be stationary are moving in the visual environment. Oscillopsia occurs during head movement in people with vestibular hypofunction, indicating inadequate gaze stabilization by the vestibulo-ocular reflex.[27] The term "blast-induced traumatic brain injury" describes head injury sustained during an explosion as a result of 1 or more of the following effects of the blast: primary, secondary, or tertiary. This term encompasses the theoretical effects of the primary overpressure wave (primary blast effects) as well as the documented effects associated with blunt head trauma from flying debris (secondary blast effects) or from displacement of the individual (tertiary blast effects). Furthermore, it conforms to clinical management patterns in DOD and Veterans Health Administration facilities, where the majority of survivors of blasts with moderate to severe TBI receive acute care and subsequent rehabilitation.

The topic of blast injury assessment and management is timely given the conflicts in Iraq and Afghanistan; however, it is not without controversy. According to the US Army Surgeon General's Task Force on TBI, there is no objective evidence in humans to support the hypothesis that a primary overpressure wave from a blast causes neuronal damage and subsequent brain injury.[28] It is expected that current research in animal models and clinical work with survivors of blasts will yield important evidence in support of neurotrauma after blast injuries.

Clinicians serving in war zones, the DOD, and the Veterans Health Administration have contributed to the body of knowledge about blast inju-

ries and polytrauma in the current conflicts.[3-5,23,29-33] However, there remains a paucity of research related to the assessment and management of dizziness and vestibular sequelae in BITBI. With renewed attention to and education about BITBI, the rehabilitation community has an opportunity to contribute to the growing body of knowledge and to help investigate mechanisms of injury and effective recovery strategies.

The purpose of this review is to summarize the findings of clinicians and scientists conducting research on the effects of blasts with the aims of defining the scope of the problem, describing and characterizing the effects of blasts, reviewing relevant patients' characteristics and sensorimotor deficits associated with BITBI, and suggesting clinical best practices for the rehabilitation of BITBI and blast-related dizziness.

Epidemiology of BITBI

According to the Defense Veterans Brain Injury Center, blast injuries have been responsible for over 65% of the casualties in the ongoing conflicts in Iraq and Afghanistan.[12,32] In a recent study of injury patterns associated with Operation Iraqi Freedom, improvised explosive devices and mortars accounted for 78% of battle wounds treated at clinical facilities in Iraq.[33] It is estimated that as many as 16,000 service members have sustained blast-induced trauma.[12] Unfortunately, the actual number of service members who have been wounded likely is larger given the difficulties associated with monitoring blast exposure in a war zone and the failure of service members to report mild concussive injuries, particularly when there are no concomitant orthopedic or soft-tissue injuries. Recent findings for a brigade combat team (N=3,973) returning from Iraq after a year-long deployment indicated that 22.8% of service members had at least one TBI confirmed by a clinician and that 88% of these were caused by blasts. In addition, the rates of comorbid dizziness (59.3%) and balance problems (25.9%) after the blasts were among the top 3 complaints of service members (headache was first), suggesting that the prevalence of blast-related dizziness (and potential vestibular involvement) may be greater than previously reported.[34]

Much of what has been learned about the effects of blasts in the current conflicts has been obtained from the characterization of service members whose injuries were treated in polytrauma centers, such as the Walter Reed Army Medical Center and the Veterans Health Administration. Although much has been reported regarding the neuropsychiatric and orthopedic sequelae associated with blasts, considerably less attention has been paid to vestibular deficits. The incidences of dizziness, vestibular pathology, and TBI secondary to blast-induced barotrauma are unknown. In the literature on blunt (nonblast) TBI, the incidence of dizziness in patients with TBI has been reported to be as high as 80%, with the incidence of specific vestibular pathology ranging between 30% and 65%.[35-42] The characterization of blast-related dizziness is further complicated by the incredible physical and psychological stresses associated with injuries in a war zone.[10,43] A survivor of a blast may have sustained not only a wide array of physical injuries but also often intense psychological trauma that can further complicate the recovery process.

There is an ongoing debate about whether the symptoms associated with mTBI, collectively referred to as postconcussion syndrome (PCS), originate from a pathophysiological process or are psychosomatic in nature. Proponents of a pathophysiologic etiology suggest that PCS is a manifestation of the delayed effects of diffuse axonal injury.[44] Others have maintained that PCS may be more strongly associated with pre- or postinjury psychological factors.[45] Rutherford[46] hypothesized that the delayed onset of PCS (days to weeks) often coincides with patients' return to daily activities. Although plausible, such an explanation does not account for the immediate onset of symptoms, such as headaches, dizziness, and nausea, in patients who have been exposed to blasts and have mTBI.[34,47] Although the occurrence of dizziness in conjunction with other PCS-like symptoms has been documented in patients who have psychiatric disorders and chronic pain but do not have brain injuries, several studies have demonstrated that somatic and cognitive symptoms are more likely to be associated with head trauma and loss of consciousness, whereas behavioral symptoms are more likely to be related to psychological distress.[46-50]

In summary, the extent of blast-related dizziness and BITBI may be difficult to assess. Even by the most conservative estimates of vestibulopathy in BITBI, it is likely that thousands of people have vestibular pathology but that only a small percentage have been formally assessed or treated.

Blast Effects

A blast results when solids or liquids are rapidly converted into a gas. In this state, the gas molecules become heated and highly pressurized. The heated gas expands into the surrounding air at speeds higher than that at which light travels, compressing the air and creating a peak overpressure wave or shock wave radiating from the point of detonation.[14,17,18,51] Closely following the shock wave is a blast wind that also radiates from the point of detonation. As the gas expands, the pressure drops and creates a vacuum or

Table 1.
Effects of Blast Injuries

Category	Types of Injuries	Patterns of Injuries[a]
Primary	Injuries from impact or shearing from overpressure wave	Brain (TBI),[17] viscera, lungs (pulmonary emboli),[54] tympanic membrane rupture or inner ear pathology (vestibular, cochlear, or both)[9,17,18,21,53]
Secondary	Injuries from projectiles (eg, shrapnel or debris)	Fractures, limb loss, TBI, soft-tissue injuries[52,53]
Tertiary	Injuries from displacement of the individual by blast wind	TBI, limb loss, fractures[17,53]
Quaternary	Other injuries	Burns, crush injuries, asphyxia, exposure to toxic substances, exacerbation of chronic illness[17,53]

[a] TBI=traumatic brain injury.

negative-pressure wave. Extreme pressure changes occur as the stress and shear waves of the blast hit the body.[52] The effects of the primary overpressure wave are nonlinear and complex. Although the damage produced by the overpressure wave typically decreases exponentially from the blast epicenter, if the explosion is detonated within an enclosed space or if the blast waves travel inside a vehicle, then the effects of the blast waves become additive as the waves reflect off walls, floor, and ceiling.[17]

Mechanisms of Injury

The effects of blasts are typically concomitant and not mutually exclusive. They are categorized as primary, secondary, tertiary, and quaternary (Tab. 1).[53] Primary blast injuries are caused by barotrauma attributable to either overpressurization or underpressurization relative to atmospheric pressure.[17] Primary blast injuries commonly affect the hollow organs in the chest, abdomen, and middle ear as well as the great vessels in the neck, the inner ear, and possibly the brain.[17,18,53,54] Given the relative exposure of the head and neck during a blast, middle and inner ear trauma is common. The ear traditionally has been considered a sensitive indicator of blast exposure, with 35% to 50% of survivors experiencing conductive, sensorineural, or mixed hearing loss.[14] Vestibular complaints are also common among survivors of blast injuries in the current conflicts; 15% to 40% complain of dizziness or vertigo.[14,21] Blast injuries to the eye can rupture the globe and cause blindness.[17] Primary blast injuries also may cause coup-contrecoup brain damage, pulmonary emboli, gastrointestinal tract rupture, or internal bleeding.[17]

The most common forms of closed head injury (CHI) related to blast exposure are diffuse axonal injury, contusion, and subdural hemorrhage. Of these, diffuse axonal injury is most frequently associated with mTBI and characterizes the vast majority of blast injuries sustained by service members.[52] Diffuse axonal injury occurs when shearing, stretching, or traction on small nerves leads to impaired axonal transport, focal axonal swelling, and possible axonal disconnection.[55,56] This pathophysiologic process is not unique to BITBI. Blunt TBI and mixed TBI (blunt trauma and barotrauma) may further contribute to comorbid dizziness and vestibular pathology in cases of secondary and tertiary effects of blasts. Temporal bone fractures, labyrinthine concussion, benign paroxysmal positional vertigo (BPPV), perilymphatic fistulae, and vascular or central lesions are commonly implicated as causes of vestibular pathology after head trauma.[18,36,41,42,57–59]

In 2006, Taber et al[52] reviewed the limited clinical findings in humans and animals exposed to a primary blast. Neuropathological changes in humans included small hemorrhages within white matter, chromatolytic changes in neurons (degeneration of Nissl bodies, an indication of neuronal damage), diffuse brain injury, and subdural hemorrhage.[52] Mott[60] described a few cases in which a primary blast was the proposed cause of death as a result of perivascular space enlargement, subpial hemorrhages, venous engorgement, white matter hemorrhages into the myelin sheath and perivascular spaces, and chromatolysis. Recent mTBI research in animal models suggested that the degree of injury may be more extensive than previously believed. The absence of focal axonal swelling (a neuropathological marker that is used to gauge the severity of head injury) in severe TBI may be one reason that the magnitude of mTBI is underestimated.[61] Other findings obtained in animal models of blast injury included widespread microglial activation (indicative of neural degeneration) in the cerebellar and cerebral cortices, pineal gland involvement, and functional deficits in coordination, balance, and strength (force-generating capacity) testing.[62–64] These data provide evidence of the insidious and potentially devastating cellular and functional effects of blasts.[52]

Table 2.
Clinical and Laboratory Tests for Vestibular Pathology in Subjects Exposed to Blasts

Tests	Structures, Pathways, or Process Assessed	Applications	Abnormal Findings	Interpretation
Head impulse test[65] (clinical)	Horizontal semicircular canals, superior branch of vestibular nerve	High-acceleration, moderate-velocity, low-amplitude head rotation with subject maintaining gaze on fixed target	Corrective saccade to target after head rotation	Abnormal angular vestibulo-ocular reflex (aVOR) attributable to peripheral vestibular hypofunction
Electronystagmography[66] (laboratory)	Extraocular muscles, horizontal semicircular canals, superior branch of vestibular nerve, vestibular and oculomotor pathways within central nervous system	Exposure to aural and visual stimulation (eg, calorics, moving targets)	Abnormal nystagmus, abnormal eye movements	Abnormal 8th cranial nerve; abnormal smooth pursuit or saccades attributable to pathology within peripheral or central vestibular pathways, oculomotor pathways, or both
Rotary chair test[66] (laboratory)	Horizontal semicircular canals, superior branch of vestibular nerve	Sinusoidal rotation at frequencies of 0.01–0.64 Hz; clockwise and counterclockwise rotation at 60°/s and 240°/s	Abnormal nystagmus, abnormal eye movements	Abnormal aVOR gain or phase attributable to pathology within peripheral or central vestibular or oculomotor pathways
Positional test[66,67] (clinical or laboratory)	Semicircular canals	Movement of involved canal into gravity-dependent position	Patient-reported complaints of vertigo and pathologic nystagmus	Abnormal presence of otoconia in semicircular canal (ie, benign paroxysmal positional vertigo)
Dynamic visual acuity test[66,68] (clinical)	Horizontal semicircular canals, vestibular nerve	Active or passive head movement while visualizing optotype direction	Inability to identify target during head movement	Abnormal aVOR attributable to peripheral vestibular hypofunction; uncompensated aVOR
Computerized dynamic posturography,[69] sensory organization test (SOT), motor control test (MCT) (clinical or laboratory)	Integration of multisensory input for balance	Challenge of balance with equipment and software under different conditions	SOT: inappropriate responses to inaccurate sensory inputs; MCT: delayed motor responses to unpredictable perturbations	SOT: age- and height-referenced responses to sway in sagittal plane; MCT: balance dysfunction and impaired reactive latencies
Balance Manager Dynamic inVision System,[70] gaze stability, perception time, target acquisition, target tracking (clinical)[a]	Horizontal semicircular canals, vestibular nerve, vestibular and oculomotor pathways	Head movement while visualizing letters; tracking of moving targets	Abnormal oculometric features compared with those of subjects who were healthy and matched for age	Behavioral measure suggesting cerebellar dysfunction; damage to central oculomotor pathways, vestibular pathways, or both

[a] The Balance Manager Dynamic inVision System (NeuroCom International Inc, 9570 SE Lawnfield Rd, Clackamas, OR 97015) provides oculomotor and vestibular testing not available in other NeuroCom systems. Novel assessments include perception time, target acquisition, and target tracking. Gaze stability testing is provided in commercially available models such as the SMART Equi-Test System (NeuroCom International Inc). Visual testing typically is performed in a darkened room with a viewing distance of 390 cm (13 ft). Perception time is measured by calculating the time (in milliseconds) that a randomly presented target must be on the screen before accurate recognition by a subject. Target acquisition is the time (in milliseconds) required to make a saccade from the center of the screen to the new optotype position. Target tracking is the speed (in degrees per second) at which a subject can accurately track a symbol. Gaze stabilization is the speed (in degrees per second) at which a subject can move his or her head and accurately hold a target in view.[70]

Patients' Characteristics and Sensorimotor Deficits in BITBI

To date, few reports have characterized vestibular findings in detail, and a definitive incidence of blast-related vestibular pathology has not been established.[54] Table 2 summarizes common clinical and laboratory measures, the structures that they assess, and how the tests should be applied. Additionally, abnormal test findings and guidelines on how such findings should be interpreted are included. For a more-detailed review of vestibular testing techniques, see Schubert and Minor[27] as well as the references cited in Table 2.[65–70]

In a retrospective review and case study, Scherer et al[21] reported dizziness (39%), vertigo (24%), and oscillopsia (27%) in the days or weeks after blast exposure and the persis-

ence of some symptoms months after the injury. Shupak et al[22] described symptoms and objective hearing and vestibular findings in 5 Israeli soldiers exposed to a blast. Patient-reported complaints included vertigo (60%), hearing loss (80%), tinnitus (80%), and otalgia (20%). Tympanic membrane perforation occurred at a rate of 80% (8 of the 10 assessed ears). None of the soldiers had head trauma, loss of consciousness, or amnesia. Clinical and laboratory assessments were performed for all soldiers within 1 week of the incident and included tympanometry; pure-tone and speech audiometry (the auditory brain stem response was tested in 1 patient); a sinusoidal rotary chair test; electronystagmography (ENG), including measures of spontaneous, gaze-evoked, positional, and positioning nystagmus; and bithermal caloric testing. Three of the 5 patients (60%) were diagnosed with unilateral vestibular hypofunction, and 1 patient was diagnosed with BPPV. Although all of the subjects reported the resolution of symptoms by their 1-month follow-up, 2 of the 3 diagnosed with unilateral vestibular hypofunction were shown to have permanent damage to the peripheral vestibular system. That study[22] was the first to document vestibular pathology independent of a TBI diagnosis in soldiers exposed to a blast.

In a single-subject study, Sylvia et al[20] documented vestibular and balance deficits in an active-duty Marine exposed to the backblast from a shoulder-launched missile system. Although the Marine experienced a brief loss of consciousness and confusion at the scene, the full effects of the blast were reportedly mitigated by a Kevlar* helmet and hearing protection and by the fact that the Marine was not directly facing the explosion. On subsequent evaluation, he reported an intense frontal headache, photophobia, nausea, tinnitus, dizziness, oscillopsia, and abnormality of gait. The results of a clinical neurological examination and a brain computed tomography examination and the integrity of the tympanic membranes were all normal. The service member was discharged 11 days after the blast exposure with a medical diagnosis of mTBI. At a 1-month follow-up, he reported dizziness, headaches, and fatigue as well as difficulty sleeping and increased irritability. Cognitive testing revealed deficits in attention, concentration, processing speed, and memory. At 6 weeks after the insult, the subject underwent a comprehensive vestibular examination (he continued to report oscillopsia and imbalance). The audiometry results were normal; however, the subject had abnormally low vestibulo-ocular reflex gains at frequencies of 0.02, 0.08, 0.32, and 0.64 Hz in a sinusoidal rotary chair assessment, refixating saccades with leftward head impulse testing, and left lateropulsion during gait testing. In aggregate, the authors reported that the findings were consistent with a chronic left peripheral vestibular lesion that was uncompensated for both dynamic vestibulospinal reflexes and the vestibulo-ocular reflex. When the Marine returned to duty, he continued to report balance-related difficulties at night, suggesting possible performance impairments that could jeopardize his safety or the safety of his team members. Interestingly, the authors reported that the results of a vestibular examination at a 4-month follow-up were normal, suggesting transient peripheral vestibular hypofunction of several months' duration. The vestibulo-ocular reflex phase (a measure of eye position relative to head position during rotary chair testing and a more persistent indicator of chronic pathology) was not reported.[20]

In a study examining the effects of a blast in a confined space, Cohen et al[15] documented a 1994 bus bombing that resulted in 22 people being killed, 48 people being injured, and 23 people being hospitalized. Seventeen patients were monitored for 6 months in an outpatient otolaryngology clinic. Vestibular testing included ENG and computerized dynamic posturography (CDP). A total of 41% of survivors of the blast reported dizziness, 35% had deficits in postural stability, and 12% complained of positional vertigo. The ENG testing did not reveal abnormalities in the 13 people who were tested. A total of 71% of the survivors with initial complaints of dizziness continued to be symptomatic at the 6-month follow-up.

Van Campen et al performed the largest systematic evaluation of survivors of a blast to date in a 2-paper series after the Oklahoma City bombing; the first report focused on audiologic sequelae,[71] and the second report documented vestibular sequelae.[25] In the latter study, investigators evaluated 30 subjects with complaints of dizziness, vertigo, or imbalance over the course of 1 year using a questionnaire, ENG, and CDP. The onset of symptoms in this group was variable; 48% of subjects reported the immediate onset of dizziness on the day of the blast, and 63% reported that this symptom occurred within 48 hours afterward. Sixteen of the 24 subjects evaluated 1 year later (67%) reported troubling symptoms. The ENG findings indicated that 30% of subjects had positional nystagmus, 11% had BPPV, 7% had bithermal caloric weakness, 4% had abnormal smooth pursuit, and 4% had gaze-evoked nystagmus. The CDP results were mixed; 68% of subjects had normal function, 15% showed a vestibular pattern (difficulty standing with eyes closed/moving platform and difficulty standing with moving surround/moving plat-

* E. I. du Pont de Nemours & Co, 1007 Market St, Wilmington, DE 19898.

form), 13% had surface-dependent deficits (difficulty standing on a moving platform), and 4% had physiologically inconsistent results.[24]

In a recent abstract, Hoffer et al[72] compared dizziness in service members exposed to a blast with dizziness in service members not exposed to a blast. The investigators reported on 34 patients who had experienced a CHI without a blast component (CHI group) and 21 patients who had experienced a blast injury only (blast group). Each group provided a detailed history and underwent audiologic and vestibular testing. In the CHI group, 59% (20/34) were classified as having posttraumatic migraine-associated dizziness, 6% (2/34) were classified as having posttraumatic exercise-induced dizziness, and 35% (12/34) were classified as having posttraumatic spatial disorientation. The investigators described 2 subgroups within the blast group—a group with vertigo and a group without vertigo. Headaches and chronic unsteadiness were common in both subgroups. Another difference between the CHI and blast groups was the report of headaches and dizziness beginning *during* exercise in the blast group but the report of the onset of dizziness *after* exercise in the CHI group. Finally, the blast group had significantly more people with hearing loss and neurocognitive disorders. These data suggest that there are pathophysiologic differences between BITBI and blunt trauma–induced TBI.

In summary, the current body of literature describing the vestibular-like symptoms attributable to a blast exposure consists primarily of case studies and single-subject reports. Nonetheless, these studies and reports consistently described patients with persistent symptoms (eg, postural instability and oscillopsia) and physiologic findings characteristic of vestibular pathology (eg, positional nystagmus and asymmetry on ENG testing). Although the current body of literature on the effects of blast exposure seems to support correlations among blast exposure, vestibular pathology, and TBI, the small amount of evidence in these studies precludes inferences of causality. To date, the absence of a gold standard or at least agreement about diagnostic measures for blast-related dizziness has made definitive links among blast exposure, vestibular pathology, and TBI elusive. The rehabilitation community has an opportunity to contribute data toward a consensus on optimal diagnostic and treatment practices.

Toward a Consensus on Clinical Best Practices

It is important to use evidence-based diagnostic and rehabilitation strategies in the management of service members with BITBI.[17] In recent years, clinicians across disciplines in the DOD and the Veterans Health Administration have significantly advanced knowledge about blast sequelae with the publication of policy reports, systems-of-care reports, editorials, and clinical case reports.[4,5,20,21,30,73–76] Additionally, selected studies of the characterization and treatment of dizziness after concussive injuries in personnel on active duty and in the literature on sports-related trauma and brain injury may be relevant to the management of head trauma from secondary and tertiary blast effects.[26,27,77–82] Despite this growing body of literature describing the presentation of BITBI and blast-related dizziness, there is no official consensus on how to assess blast injuries or on how to conduct the clinical examination.

The American Physical Therapy Association's *Guide to Physical Therapist Practice*[83] emphasizes clinical management based on a framework for assessing the level of pathology (eg, asymmetric peripheral vestibular function), impairment (eg, gaze or gait instability), functional limitations (eg, ability to perform job-related tasks), and disability (eg, inability to function in one's chosen vocation). For the care of patients with polytrauma attributable to blast effects, this model provides a rehabilitation-focused complement to traditional medical (diagnosis-driven) or mechanism-of-injury–driven models of clinical management.[84] The application of the World Health Organization's *International Classification of Functioning, Disability and Health* (ICF)[85] expands traditional concepts of the physical therapy plan of care through an evaluation of the impact of identified deficits relative to a service member's unique social and vocational contexts. Clinical integration of the ICF model into the assessment of dizziness in service members who have been exposed to a blast can aid rehabilitation providers in their efforts to articulate how even subtle disruptions in body structure and function can dramatically affect participation and disability.

Physical Therapy Assessment of BITBI and Vestibular Pathology

History and Physical Examination

For clinicians caring for service members who have been injured, tools such as the Military Acute Concussion Evaluation[13] and the Walter Reed Army Medical Center Blast Injury Questionnaire[21] may be useful in directing the initial screening and characterization of patients with blast exposure and suspected TBI or vestibular pathology. Other self-report measures, such as the Dizziness Handicap Inventory[86] and the Activities-specific Balance Confidence Scale,[87] provide insight into a patient's self-perceived limitations, which may have deleterious effects on rehabilitation or social function-

g. These measures are ideally administered initially and during llow-up visits. Pierce and Hanks[88] ported that participation in activies of daily living is one of the best redictors of quality of life in people ith TBI.

he physical examination should be irected toward an assessment of nction implicated in the history nd systems review.[20,21] The clinical ssessment of symptomatic blast-xposed personnel should include easures of vestibulo-ocular reflex nction, positional testing, and meaures of posture and gait stability. roviders at the DOD have advoated the screening of service members who have been exposed to a last with a series of questionnaires, udiology measures, and clinical vesibular tests (eg, noncomputerized assive dynamic visual acuity test nd head impulse test) (Figure).[21] he Figure is adapted from a DOD lgorithm detailing the process for he care of people with TBI.[89] It ighlights critical management pracices for personnel with blast injuries om the time of the injury (combat one) to the eventual discharge from ehabilitation. The Figure informs ecisions about patient care by guidng appropriate provider referrals, ighlighting the specific capabilities f interdisciplinary team members, ighlighting commonly used assessments for testing people who have been exposed to blasts, and presentng management options for blast-elated dizziness. In aggregate, the lgorithm illustrates the importance f interdisciplinary cooperation for ptimal diagnostic, prognostic, and herapeutic practices and outcomes.

Computerized behavioral measures such as the dynamic visual acuity test nd CDP are widely used in rehabiltation settings to assess gaze and postural stability, respectively, in patients with blast exposure, dizziness, or TBI.[15,24,25,70] Vestibular function testing (eg, rotary chair test and ENG) is also advisable in this patient population.[27,66] The sensitivity of clinical gait analysis to vestibular deficits may be enhanced by the administration of a standardized measure, such as the Dynamic Gait Index[90] or the Functional Gait Assessment.[91] For people with suspected mTBI and cognitive deficits (in addition to dizziness), therapists should consider dual tasking in balance and gait activities to identify processing and reaction time impairments.[79–82,92]

Because some clinicians caring for service members who have been exposed to a blast are reporting associations between physical exertion (eg, running) and the onset of symptoms (eg, headache and vertigo), current DOD guidelines recommend exertional testing in patients with TBI before a return to full duty.[91] A service member's successful (ie, asymptomatic) completion of such testing may offer reasonable assurance that symptoms will not recur with physical stress, gauge readiness to return to work, and serve as a long-term rehabilitation goal guiding discharge from physical therapy.

Table 3 provides a concise review of vestibular impairments associated with TBI. Because the underlying pathology associated with blast-related dizziness has not been causally established in prospective studies, Table 3 shows an impairment-based approach to assessment; references for both blast-related TBI and non–blast-related TBI are cited as the foundation for current best-practice guidelines.

Impairments to Participation

Patients who have been exposed to a blast and are symptomatic may have significant barriers to participation in their unique social context. For service members with blast injuries, persistent dizziness and TBI may contribute to disability by limiting their ability to serve in a prior capacity on active duty. In the context of the ICF model, it is conceivable that a patient with severe neurologic pathology (eg, spinal cord injury or cerebrovascular accident) may have significant impairments (eg, paralysis or hemiparesis) but not necessarily have barriers at the level of participation given sufficient time and success with rehabilitation. The converse is equally conceivable for a service member with subtle impairments in the demanding context of military service. For instance, an individual who requires a high degree of gaze stability to function as an aviator or sniper may be effectively disabled by even a minor vestibular deficit. Similarly, a service member who demonstrates little objective impairment during vestibular testing but who continues to complain of motion intolerance may be ineffective or, worse, detrimental to mission performance in an operational environment.

Physical Therapy Management of BITBI and Vestibular Pathology

Individualized vestibular physical therapy has been shown to be beneficial for survivors of blasts with vestibular symptoms. Recently, Gottshall[70] reported that service members with blast-related balance problems demonstrated significant improvement in gaze and gait stability after 16 weeks of vestibular physical therapy. Dynamic visual acuity, target acquisition, and target tracking improved after 4 weeks. In addition, horizontal gaze stabilization test scores improved significantly after 12 weeks, and vertical gaze stabilization test scores improved significantly between 12 and 16 weeks—coinciding with patients' self-reports of resolving dizziness while running. The Dynamic Gait Index showed significant improvement at week 8 and continued improvement at week 12.

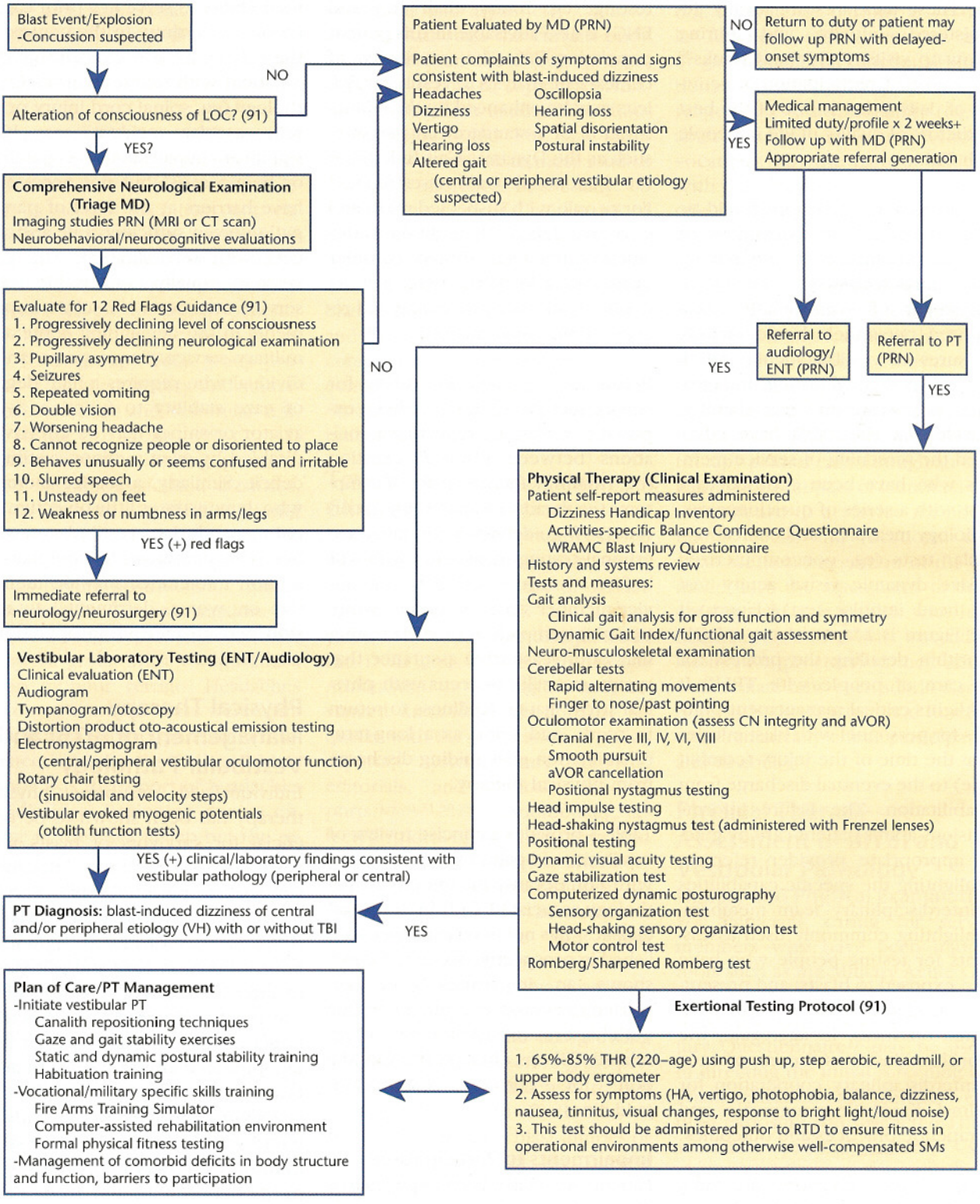

Figure.
Integrated rehabilitation management of blast-related dizziness. LOC=loss of consciousness; PRN=as needed/indicated by provider; RTD=return to duty; MRI=magnetic resonance imaging; CT=computed tomography; ENT=ear, nose, and throat (otolaryngology MD); PT=physical therapy; PCM=primary care provider; 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 vestibulo-ocular reflex.

Table 3.
Clinical Presentation of Common Vestibular Symptoms and Findings Associated With Traumatic Brain Injury (TBI)[a]

Impairment	Recommended Tests[b]	Evidence[c]
Increased motion sensitivity (motion sickness)	Clinical examination, Motion Sensitivity Quotient, clinical test of sensory integration of balance	Abnormal posturography[77]
Oscillopsia or gaze instability	Clinical examination, computerized dynamic visual acuity	Abnormal computerized dynamic visual acuity in patients with acute-stage mTBI,[78] significant dizziness-related disability relative to that in subjects without mTBI[78]
Vertigo	Clinical examination, positional test (Dix-Hallpike), electronystagmography	Isolated BPPV in absence of documented head injury (PBI),[d,18,51] blast-induced BPPV secondary to head trauma,[d,15] BPPV in 15% of patients with TBI,[92] BPPV secondary to primary overpressure wave (PBI),[d,22] nonpositional vertigo in 5/5 service members located within 300 cm (10 ft) of blast epicenter,[d,23] abnormal Dix-Hallpike test results (positional testing)[25]
Posttraumatic migraine-associated dizziness	Clinical examination, computerized dynamic posturography, rotary chair test	Abnormal aVOR gain, phase, symmetry in sinusoidal rotary chair test,[25,26] abnormal high-frequency aVOR gain,[25,26] normal CDP findings[25,26]
Spatial disorientation	Clinical examination, computerized dynamic posturography, rotary chair test, videonystagmography	Abnormal CDP findings,[25,26] abnormally low vestibulo-ocular reflex gains and abnormal phase shifts during midfrequency (0.32 and 0.64 Hz) sinusoidal rotary chair test,[25,26] visual fixation abnormalities during videonystagmography[25,26]
Gait deficits	Clinical examination, motion capture test (gait laboratory)	Lower gait speed (acute), subnormal gait speed (chronic), conservative gait strategy with dual tasking,[79] abnormality of gait,[d,20] significantly reduced gait speed and stride length in subjects with TBI[92]
Postural instability	Clinical examination, computerized dynamic posturography	Significantly lower composite CDP scores in subjects with mTBI than in control subjects who were healthy,[92] 15% of subjects who were exposed to blasts demonstrated a "vestibular pattern" of postural instability, that is, abnormal on conditions 5 and 6 (ie, moving force plate with eyes closed [sensory organization test condition 5] and deficits with moving force plate and moving surround (sensory organization test condition 6)[d,24]
Vestibular hypofunction	Clinical examination, caloric examination (electronystagmography), rotary chair test, computerized dynamic visual acuity	80% incidence of vestibular pathology in patients with TBI per caloric assessment: 7/10 had unilateral vestibular hypofunction and 1/10 had bilateral vestibular hypofunction[92]

[a] Data include vestibular pathology secondary to blast exposure and TBI.
[b] Clinical examination included cervical range of motion, vertebral artery test, oculomotor examination, head impulse test, passive dynamic visual acuity test (noncomputerized), and Dix-Hallpike test.
[c] mTBI=mild traumatic brain injury, BPPV=benign paroxysmal positional vertigo, PBI=primary blast injury, aVOR=angular vestibulo-ocular reflex, CDP=computerized dynamic posturography.
[d] Testing was performed for patients exposed to blasts.

Preliminary data suggested that this battery of tests may be a sensitive measure of behavioral vestibular function in patients with blast-induced head injuries.[70]

Vestibular physical therapy interventions commonly include gaze stability exercises to facilitate central angular vestibulo-ocular reflex gain adaptation[93]; substitution exercises, which are believed to increase the recruitment of compensatory saccades to help with gaze stability[93,94]; habituation techniques (eg, the Motion Sensitivity Quotient) to mitigate the hypersensitivity to head movements that is characteristic of motion intolerance[95,96]; and static and dynamic balance and gait exercises to address postural instability.[97] Benign paroxysmal positional vertigo is managed with the canalith repositioning maneuver.[98,99] Current reha-

bilitation practices at medical centers (such as the Walter Reed Army Medical Center) include training with therapeutic technologies that challenge survivors of blasts by exposing them to virtual reality stimuli. Although training devices such as the Computer-Assisted Rehabilitation Environment and the Fire Arms Training Simulator have been used to mitigate complaints of increased motion sensitivity, they are particularly effective at addressing barriers to participation because of their utility in simulating military tasks.

Conclusion

Little is known about the specific pathophysiology of blast injuries and the resultant effects on the peripheral or central vestibular system. It is not known how these processes may affect the cortical and subcortical structures responsible for motion perception, spatial orientation, equilibrium, and gaze stability. Clinicians and researchers working with patients exposed to blasts must develop sensitive screening and assessment measures to identify vestibular pathology in this patient population, quantify the degree of impairment attributable to a blast, and formulate appropriate treatment strategies to ensure optimal participation and minimal disability.

Both authors provided concept/idea/project design and writing. Dr Schubert provided project management, facilities/equipment, institutional liaisons, and consultation (including review of manuscript before submission).

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References

1 Rustemeyer J, Volker K, Bremerich A. Injuries in combat from 1982–2005 with particular reference to the head and neck: a review. *Br J Oral Maxillofac Surg.* 2007; 45:556–560.

2 Gajewski D, Granville R. The United States Armed Forces Amputee Patient Care Program. *J Am Acad Orthop Surg.* 2006;14(10 spec no):S183–S187.

3 Javernick M, Doukas W. Process of care for battle casualties at Walter Reed Army Medical Center, part I: Orthopedic Surgery Service. *Mil Med.* 2006;171:200–202.

4 Pasquina P, Gambel J, Foster L, et al. Process of care for battle casualties at Walter Reed Army Medical Center, part III: Physical Medicine and Rehabilitation Service. *Mil Med.* 2006;171:206–210.

5 Lew HL, Poole JH, Vanderploeg RD, et al. Program development and defining characteristics of returning military in a VA Polytrauma Network Site. *J Rehabil Res Dev.* 2007;44:1027–1034.

6 Cancio LC, Horvath E, Barillo DJ, et al. Burn support for Operation Iraqi Freedom and related operations 2003, 2004. *J Burn Care Rehabil.* 2005;26:151–161.

7 Gaylord KM, Cooper DB, Mercado JM, et al. Incidence of posttraumatic stress disorder and mild traumatic brain injury in burned service members: preliminary report. *J Trauma.* 2008;64(2 suppl): S200–S206.

8 Martin EM, Lu WC, Helmick K, et al. Traumatic brain injuries sustained in the Afghanistan and Iraq wars. *Am J Nurs.* 2008; 108:40–48.

9 Okie S. Reconstructing lives: a tale of two soldiers. *N Engl J Med.* 2006;355: 2609–2615.

10 Warden D. Military TBI during the Iraq and Afghanistan wars. *J Head Trauma Rehabil.* 2006;21:398–402.

11 Xydakis MS, Fravell MD, Nasser KE, Casler JD. Analysis of battlefield head and neck injuries in Iraq and Afghanistan. *Otolaryngol Head Neck Surg.* 2005;133:497–504.

12 Defense Veterans Brain Injury Center. *OIF/OEF Fact Sheet: June 2008.* Washington, DC: Walter Reed Army Medical Center; 2008.

13 French L, McCrae M, Baggett M. The Military Acute Concussion Evaluation (MACE). *Journal of Special Operations Medicine.* 2008:8:68–74.

14 Cave KM, Cornish EM, Chandler DW. Blast injury of the ear: clinical update from the global war on terror. *Mil Med.* 2007;172: 726–730.

15 Cohen JT, Ziv G, Bloom J, et al. The ear in a confined space explosion: auditory and vestibular evaluation. *Isr Med Assoc J.* 2002;4:559–562.

16 Cudennec Y, Buffe P, Poncet J. Otologic teachings and features of a bombing attempt. *Mil Med* 1995;160:467–470.

17 DePalma RG, Burris DG, Champion HR, Hodgson MJ. Blast injuries. *N Engl J Med.* 2005;352:1335–1342.

18 Kerr A. Blast injuries to the ear. *The Practitioner.* 1978;221:677–682.

19 Cernak I, Savic J, Zunic G, et al. Recognizing, scoring, and predicting blast injuries. *World J Surg.* 1999;23:44–53.

20 Sylvia FR, Drake AI, Wester DC. Transient vestibular balance dysfunction after primary blast injury. *Mil Med.* 2001;166: 918–920.

21 Scherer M, Burrows H, Pinto H, Somrack E. Characterizing self reported dizziness and otovestibular impairment among blast injured traumatic amputees: a pilot study. *Mil Med.* 2007;172:731–738.

22 Shupak A, Doweck I, Nachtigal D, et al. Vestibular and audiometric consequences of blast injury to the ear. *Arch Otolaryngol Head Neck Surg.* 1993;119:1362–1367.

23 Chandler D, Edmond C. Effects of blast overpressure on the ear: a case report. *J Am Acad Audiol.* 1997;8:81–88.

24 Van Campen LE, Dennis JM, King SB, et al. One-year vestibular and balance outcomes of Oklahoma City bombing survivors. *J Am Acad Audiol.* 1999;10:467–483.

25 Hoffer ME, Gottshall KR, Moore R, et al. Characterizing and treating dizziness after mild head trauma. *Otol Neurotol.* 2004;25: 135–138.

26 Hoffer ME, Balough BJ, Gottshall KR. Posttraumatic balance disorders. *Int Tinnitus J.* 2007;13:69–72.

27 Schubert MC, Minor LB. Vestibulo-ocular physiology underlying vestibular hypofunction. *Phys Ther.* 2004;84:373–385.

28 Office of the Surgeon General, US Army Medical Department. Traumatic Brain Injury (TBI) Task Force Report. Available at: http://www.armymedicine.army.mil/prr/tbitfr.html. Released January 17, 2008. Accessed June 3, 2009.

29 Murray CK. Epidemiology of infections associated with combat-related injuries. *J Trauma.* 2008;64:232–238.

30 Friedmann-Sanchez G, Sayer N, Pickett T. Provider perspectives on rehabilitation of patients with polytrauma. *Arch Phys Med Rehabil.* 2008;89:171–179.

31 Cernak I, Savic J, Lazarov A. Relations among plasma prolactin, testosterone, and injury severity in war casualties. *World J Surg.* 1997;21:240–246.

32 The Office of Patient Care Services, Department of Veterans Affairs, Veterans Health Administration. VHA handbook 1172.1. Polytrauma rehabilitation procedures. Available at: http://www1.va.gov/vhapublications/ViewPublication.asp?pub_ID=1317. Issued September 22, 2005. Accessed June 3, 2009.

33 Murray CK, Reynolds JC, Schroeder JM, et al. Spectrum of care provided at an echelon II medical unit during Operation Iraqi Freedom. *Mil Med.* 2005;170:5516–5520.

34 Terrio H, Brenner L, Ivins B, et al. Traumatic brain injury screening: preliminary findings in a US Army brigade combat team. *J Head Trauma Rehabil.* 2009;24: 14–23.

35 Maskell FW, Chiarelli P, Isles R. Dizziness after traumatic brain injury: results from an interview study. *Brain Inj.* 2007;21: 741–752.

36 Shumway-Cook A. Assessment and management of the patient with traumatic brain injury and vestibular dysfunction. In: Herdman S, ed. *Vestibular Rehabilitation*. 3rd ed. Philadelphia, PA: FA Davis Company; 2007:444–457.

37 Barber HO. Head injury: audiological and vestibular findings. *Ann Otol Rhinol Laryngol*. 1969;78:239.

38 Pearson BW, Barber HO. Head injury: some otoneurologic sequelae. *Arch Otolaryngol*. 1973;97:81–84.

39 Griffith MV. The incidence of auditory and vestibular concussion following minor head injury. *J Laryngol Otol*. 1979;93:253.

40 Gibson W. Vertigo associated with trauma. In: Dix R, Hod JD, eds. *Vertigo*. New York, NY: Wiley; 1984.

41 Healy GB. Hearing loss and vertigo secondary to head injury. *N Engl J Med*. 1982; 306:1029–1031.

42 Herdman SJ. Treatment of vestibular disorders in traumatically brain-injured patients. *J Head Trauma Rehabil*. 1990;5:63.

43 French LM, Parkinson GW. Assessing and treating veterans with traumatic brain injury. *J Clin Psychol*. 2008;64:1004–1013.

44 Dixon CE, Taft WC, Hayes RL. Mechanisms of mild traumatic brain injury. *J Head Trauma Rehabil*. 1993;8:1–12.

45 Szymanski HV, Linn R. Review of the postconcussion syndrome. *Int J Psychiatry Med*. 1992;22:357–375.

46 Rutherford WH. Post concussion symptoms: relationship to acute neurological indices, individual differences, and circumstances of injury. In: Eisenberg HM, Benton AL, eds. *Mild Head Injury*. New York, NY: Oxford University Press; 1989: 217–228.

47 Ryan LM, Warden DL. Post concussion syndrome. *Int Rev Psychiatry*. 2003;15: 310–316.

48 Fox DD, Lees-Haley PR, Earnest K, Dolezal-Wood S. Base rates of post concussive symptoms in health maintenance organization patients and controls. *Neuropsychology*. 1995;9:606–611.

49 Fox DD, Lees-Haley PR, Earnest K, Dolezal-Wood S. Post concussive symptoms: base rates and etiology in psychiatric patients. *Clin Neuropsychol*. 1995;9:89–92.

50 Smith-Seemiller L, Fow NR, Kant R, Franzen MD. Presence of post concussion syndrome in patients with chronic pain versus mild traumatic brain injury. *Brain Inj*. 2003;17:199–206.

51 Kerr A. Blast injury to the ear: a review. *Rev Environ Health*. 1987;7:65–79.

52 Taber KH, Warden DL, Hurley RA. Blast-related traumatic brain injury: what is known? *J Neuropsychiatry Clin Neurosci*. 2006;18:141–145.

53 Centers for Disease Control and Prevention. Blast injuries: essential facts. Available at: http://www.bt.cdc.gov/masscasualties/blastessentials.asp. Updated March 25, 2008. Accessed June 3, 2009.

54 Argyrous G. Management of primary blast injury. *Toxicology*. 1997;121:105–115.

55 Mendez CV, Hurley RA, Lassonde M, et al. Mild traumatic brain injury: neuroimaging of sports-related concussion. *J Neuropsychiatry Clin Neurosci*. 2005;17:297–303.

56 Hurley RA, McGowan JC, Arfanakis K, et al. Traumatic axonal injury: novel insights into evolution and identification. *J Neuropsychiatry Clin Neurosci*. 2004; 16:1–7.

57 Nelson JR. Neurootologic aspects of head injury. *Adv Neurol*. 1979;2:107–128.

58 Berman J, Fredrickson J. Vertigo after head injury: a five year follow up. *J Otolaryngol*. 1978:7:237–245.

59 Furman J, Whitney S. Central causes of dizziness. *Phys Ther*. 2000;80:179–187.

60 Mott FW. The microscopic examination of the brains of two men dead of commotio cerebri (shell shock) without visible external injury. *J R Army Med Corps*. 1917;29: 662–677.

61 Povlishock JT, Katz DI. Update of neuropathology and neurological recovery after traumatic brain injury. *J Head Trauma Rehabil*. 2005;20:76–94.

62 Kaur C, Singh J, Lim MK, et al. The response of neurons and microglia to blast injury in the rat brain. *Neuropathol Appl Neurobiol*. 1995;21:369–377.

63 Kaur C, Singh J, Lim MK, et al. Macrophages/microglia as "sensor" of injury in the pineal gland of rats following a non-penetrative blast. *Neurosci Res*. 1997;27: 317–322.

64 Moochhala SM, Lu J. Neuroprotective role of aminoguanidine in behavioral changes after blast injury. *J Trauma*. 2004;56:393–403.

65 Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol*. 1988;45: 737–739.

66 Fife T, Tusa R, Furman J, et al. Assessment: vestibular testing techniques in adults and children—report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2000;55:1431–1441.

67 Roberts R, Gans R. Background, technique, interpretation, and usefulness of positional/position testing. In: Jacobsen J, Shepherd N, eds. *Balance Function Assessment and Management*. San Diego, CA: Plural Publishing; 2008:171–196.

68 Herdman SJ, Tusa RJ, Blatt PJ, et al. Computerized dynamic visual acuity test in the assessment of vestibular deficits. *Am J Otol*. 1998;19:790–796.

69 Shepard N, Janky K. Interpretation and usefulness of computerized dynamic posturography. In: Jacobsen J, Shepherd N, eds. *Balance Function Assessment and Management*. San Diego, CA: Plural Publishing; 2008:359–378.

70 Gottshall, K. Vestibular-visual-cognitive interaction tests in patients with blast trauma. In: Association for Research in Otolaryngology Midwinter Meeting; February 14–19, 2009; Baltimore, MD. Abstract 180.

71 Van Campen LE, Dennis JM, Hanlin RC, et al. One-year audiologic monitoring of individuals exposed to the 1995 Oklahoma City bombing. *J Am Acad Audiol*. 1999;10:231–247.

72 Hoffer M, Gottshall K, Balough B, Balaban C. Vestibular difference between blast and blunt head trauma. In: Association for Research in Otolaryngology Midwinter Meeting; February 14–18, 2008; Phoenix, AZ. Abstract 964.

73 Springer BA, Doukas WC. Process of care for battle casualties at Walter Reed Army Medical Center, part II: Physical Therapy Service. *Mil Med*. 2006;171:203–205.

74 Sayer NA, Chiros CE, Sigford B, et al. Characteristics and rehabilitation outcomes among patients with blast and other injuries sustained during the global war on terror. *Arch Phys Med Rehabil*. 2008;89: 163–170.

75 Lew HL. Rehabilitation needs of an increasing population of patients: traumatic brain injury, polytrauma, and blast-related injuries. *J Rehabil Res Dev*. 2005;42:xiii–xvi.

76 Scherer M. Gait rehabilitation with body weight-supported treadmill training for a blast injury survivor with traumatic brain injury. *Brain Inj*. 2007;21:93–100.

77 Hoffer M, Gottshall M, Kopke R, et al. Vestibular testing abnormalities in individuals with motion sickness. *Otol Neurotol*. 2003;24:633–636.

78 Gottshall K, Drake A, Gray N, et al. Objective vestibular tests as outcome measures in head injury patients. *Laryngoscope*. 2003;113:1746–1750.

79 Parker T, Osternig L, Van Donkelaar P, Chou L. Recovery of cognitive and motor function following concussion. *Br J Sports Med*. 2007;41:868–873.

80 Parker T, Osternig L, Van Donkelaar P, Chou L. Gait stability following concussion. *Med Sci Sports Exerc*. 2006;38: 1032–1040.

81 Drew A, Langan J, Halterman C, et al. Attentional disengagement dysfunction following mTBI assessed with the gap-saccade test. *Neurosci Lett*. 2007;417: 61–65.

82 Broglio SP, Tomporowski PD, Ferrara MS. Balance performance with a cognitive task: a dual-task testing paradigm. *Med Sci Sports Exerc*. 2005;37:689–695.

83 Guide to Physical Therapist Practice. 2nd ed. *Phys Ther*. 2001;81:307–318.

84 Scott S, Belanger H, Vanderploeg R, et al. Mechanism-of-injury approach to evaluating patients with blast-related polytrauma. *J Am Osteopath Assoc*. 2006;106:265–270.

85 *International Classification of Functioning, Disability and Health (ICF)*. Geneva, Switzerland: World Health Organization; 2001.

86 Jacobson GP, Newman CW. The development of the Dizziness Handicap Inventory. *Arch Otolaryngol Head Neck Surg*. 1990; 116:424–427.

87 Powell LE, Myers AM. The Activities-specific Balance Confidence (ABC) Scale. *J Gerontol A Biol Sci Med Sci*. 1995;50: 28–34.

88 Pierce C, Hanks R. Life satisfaction after traumatic brain injury and the World Health Organization model of disability. *Am J Phys Med Rehabil.* 2006;85:889–898.

89 DVBIC Working Group on mTBI Management. *Symptom Management in Mild Traumatic Brain Injury*. Washington, DC: Department of Defense; 2008.

90 Shumway-Cook A, Woolacott M. *Motor Control Theory and Practical Applications*. Baltimore, MD: Williams & Wilkins; 1995.

91 Wrisley D, Marchetti G, Kuharsky D, Whitney S. Reliability, internal consistency, and validity of data obtained with the Functional Gait Assessment. *Phys Ther.* 2004; 84:906–918.

92 Basford J, Chou L-S, Kaufman K, et al. An assessment of gait and balance deficits after traumatic brain injury. *Arch Phys Med Rehabil* 2003;84:343–349.

93 Schubert M, Migliaccio A, Clendaniel R, et al. Mechanism of dynamic visual acuity recovery with vestibular rehabilitation. *Arch Phys Med Rehabil.* 2008;89:500–507.

94 Bockisch CJ, Straumann D, Hess K, Haslwanter T. Enhanced smooth pursuit eye movements in patients with bilateral vestibular deficits. *Neuroreport.* 2004;15: 2617–2620.

95 Godbout A. Structured habituation training for movement provoked vertigo after severe traumatic brain injury: a single-case experiment. *Brain Inj.* 1997;11:629–641.

96 Smith-Wheelock M, Shepard N, Telian S. Physical therapy program for vestibular rehabilitation. *Am J Otol.* 1991;12:218–225.

97 Schubert M, Migliaccio A, Della Santina C. Modification of compensatory saccades after a VOR gain recovery. *J Vestib Res.* 2006;16:1–7.

98 Herdman S, Tusa R, Zee D, et al. Single treatment approaches to benign paroxysmal positional vertigo. *Arch Otolaryngol Head Neck Surg*. 1993;119:450–454.

99 Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. *Adv Otorhinolaryngol.* 1988;42:290–293.