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Perceptual Postural Imbalance and Visual Vertigo

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Abstract

Purpose of Review Disorders of posture and balance cause significant patient morbidity, with reduction of quality of life as patients refrain from critical activities of daily living such as walking outside the home and driving. This review describes recent efforts to characterize visual disorders that interact with the neural integrators of positional maintenance and emerging therapies for these disorders.

Recent Findings Abnormalities of gait and body position sense may be unrecognized by patients but are correlated with focal neurological injury (stroke). Patients with traumatic brain injury can exhibit visual vertigo despite otherwise normal visual functioning.

Summary The effect of visual neglect on posture and balance, even in the absence of a demonstrable visual field defect, has been characterized quantitatively through gait analysis and validates the potential therapeutic value of prism treatment in some patients. In addition, the underlying neural dysfunction in visual vertigo has been explored further using functional imaging, and these observations may allow discrimination of patients with structural causes from those whose co-morbid psychosocial disorders may be primarily contributory.

Keywords Postural instability · Vestibular imbalance · Hemianopia · Visual neglect · Optic flow · Visual vertigo

Introduction

Normal postural and balance control relies upon the integration of vestibulocerebellar and visual cues with proprioceptive input; disruption in any of these systems may cause static or dynamic abnormalities. The impact of visual or vestibulocerebellar dysfunction on quality of life in a heterogeneous group has not been measured directly, but over half of patients with even episodic vertigo have reported refraining from or fearing activities such as driving [1]. Disorders that cause postural instability often have associated eye movement abnormalities, and up to 70% of patients after stroke also may have problems with eye movements such as impaired saccadic initiation or accuracy, smooth pursuit, ocular alignment, or gaze stability [2].

It is more common that patients attempt to use their ocular motor system to compensate for cortical or brainstem dysfunction, rather than having the eye movement problems as the primary driver of postural problems. Indeed, vision is the primary means of establishing and optimizing static and dynamic equilibrium. Additionally, a rapidly changing visual environment, with movement of the observer, their surroundings, or both will stress the optokinetic system (OKS) by imparting a unique sensory load (optic flow) on central sensory integration and interpretation processes. Initially, large optic flow patterns impart an error signal on either the central or the peripheral retina and drive the optokinetic response. The error signal activates a negative feedback loop to reduce the error displayed on the central or peripheral retina. This ocular motor response consists of both a slow phase (pursuit) in the direction of the motion and a fast phase (saccade) acting to reset the eye in central position. Reduction of the error signal is

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accomplished when the velocity of the eye movement during the slow phase (output signal) approximates the velocity of the optic flow motion pattern [3].

In this context, it is not unexpected that patients who develop either monocular visual loss or homonymous visual field defects from cerebral injury may have impairment integrating visual information with input from other sensory modalities. Similarly, deficits in eye tracking or failure of external inputs to the ocular motor system may result in inappropriate position sense and perceived imbalance. In this review, we will consider evidence that loss of visual awareness, either through visual field loss or neglect, results in postural shifts and instability. Similar changes may occur through less well-characterized visual pathways that may be countered by optical correction. We will also consider how eye movement and visual processing disorders can affect the normal response to visual motion even in the absence of a discrete structural lesion.

Postural Instability

Postural Instability with Visual Deficits in Stroke

Recent interest in the effects of traumatic brain injury (TBI) on postural mechanisms and the relationship between vestibular dysfunction and eye movement abnormalities has a foundation in the study of patients after stroke. Particular attention has been given to patients with visual neglect, in which a fixed visual field defect is not present but visual information from one hemifield is ignored or deprioritized. In both the presence and absence of visual field abnormalities, patients may perceive a shift in the visual vertical or subjective straight-ahead position; persistence of a subjective vertical shift has been associated with reduced physical recovery at 6 months post-stroke [4]. A study of a cohort of subjects with right hemiparesis, in which about one-half had neglect and the remaining had a manifest right homonymous hemianopia, used structural MRI to identify anatomic lesions that corresponded to dysfunctional identification of the visual vertical or subjective straight-ahead [5]. In this retrospective analysis, it was neglect and not hemianopia that was correlated with exacerbation of postural dysfunction [5]. The authors also noted that there was some overlap between lesions associated with visual perceptual shifts and postural instability, with defects in the temporoparietal, superior temporal, and posterior insular cortices found in both conditions [5]. These results support the idea that processing of positional information from the visual, vestibular, and proprioceptive systems is integrated at a cortical level and points to the ability of individuals with "purely sensory" lesions (inner ear disease, occipital lobe injury, peripheral neuropathy) to use the remaining senses to compensate and maintain their equilibrium. From a clinical perspective, the results are consistent with observations that patients who think their eyes are the cause of their imbalance will find that their problems worsen with the eyes closed, suggesting that the visual system is being used to compensate for imbalance that arises from dysfunction elsewhere in the vestibular system. Furthermore, this compensatory behavior may exacerbate or even cause visual motion hypersensitivity (see below).

Therapeutic measures that could improve function in patients with neglect include prism adaption that shifts attention into the (left-sided) area of neglect. In a series of ten consecutive patients admitted to an inpatient rehabilitation facility, balance was evaluated on a Wii Balance Board after visual neglect was confirmed by testing. Visual attentional training and target pointing practice then was conducted with yoked prisms that shifted the visual environment 10° rightward. After a single training session, subjects were retested on the Wii board and were found to have improved measures with eyes open [6]. This finding suggests that establishing better visual orientation in space can improve posture control, but it again demonstrates that the visual system is being used in a compensatory manner that may or may not be sustainable in the long term. Cognitive rehabilitation has been tried as a means of improving spatial awareness in patients with neglect; however, a metanalysis of relevant studies demonstrated not only that cognitive therapy appeared to be ineffective, but that a lack of standardized endpoints (such as quality of life improvement and reduced falls) hampers interpretation of the existing literature [7].

Postural Instability and Traumatic Brain Injury

Questionnaires given to military service members after mild TBI indicate that the majority have balance or dizziness concerns and symptoms [8], and symptom reporting is enhanced by using self-reporting or screening rather than physician interviews [8]. In a cohort of young subjects (ages 8–20) who had suffered moderate to severe TBI, diffusion tensor imaging (DTI) abnormalities in the cerebellum, cerebral peduncle, corticospinal tracts, anterior and posterior limbs of the internal capsule, and posterior thalamic radiations were correlated with reduced postural control during sensory integration/balance platform testing [9]. Additional abnormalities of postural maintenance during a visual-spatial task (reaching for a virtual, projected, object) have been identified, with slower reaching velocity and more rapid failure of intentional body motion as an aid to the desired task [10]. In none of these cohorts [8–10] were visual field defects or possible neglect characterized. In TBI subjects with neglect, functional MRI suggests that prism adaptation training enhances activity in frontoparietal centers that may help to direct visual attention to the side of neglect (typically the left) [11]. However, a review of prism adaptation studies found that potential

confounding factors with respect to improvement after treatment including time since injury [12•], and spontaneous improvement must be considered as a potential explanation for the apparent therapeutic effect that has been seen.

Postural Instability Without Visual Deficits

A subset of patients who have suffered stroke or other neurological injury but without a measurable visual field defect will demonstrate perceptual abnormalities that have been termed the visual midline shift syndrome [13]. These patients should be differentiated from individuals with a perceptual shift of the visual vertical, which can be seen with medullary lesions and is part of the Wallenberg (lateral medullary) syndrome [14]. Patients with Wallenberg syndrome will have abnormal eye movements including saccadic dysmetria and accompanying ocular lateropulsion toward the affected side. Patients may perceive that their vision is tilted several degrees off axis, and in some cases, they may even report inversion of the visual environment. In general, the patients are aware of the fact that their perception is incongruous with reality. In contrast, subjects with the visual midline shift syndrome may be unaware of their condition, reporting difficulties with activities of daily living but not vision. Observations that subjects would veer to the left or right when attempting to walk in a straight line led to the hypothesis that the perceived visual midline was displaced away from the normal egocenter. Initial studies of this condition relied upon a diagnostic method that was also felt to be therapeutic, namely, the placement of yoked horizontal prisms to induce an optical shift of the environment that would compensate for the visual misperception [15]. Criticism was leveled at this diagnosis and its putative treatment, especially as many subjects were seen to improve in a time frame that could be consistent with natural history. Thus, in an effort to find an objective means of studying the condition and its treatment, gait mat testing was introduced into the diagnostic protocol. A group of stroke patients with neither hemiparesis nor visual field defects were found to walk favoring the side ipsilateral to their stroke (i.e., unweighting of the contralateral body). In these patients, yoked prisms that shift the visual midline away from the favored side (toward the true middle) resulted in a more neutral posture and balanced stride [16•]. Although this series did not include a sham treatment group (i.e., placement of base down prism or prism oriented in the opposite direction), the findings are promising and suggest that screening for gait alteration in stroke or TBI patients with incomplete recovery and/or persistent neurological symptoms may be helpful in identifying treatable pathology.

Visual Motion Hypersensitivity

When afferent visual function is intact but an inappropriate ocular motor response (from the OKS) to real or perceived movement (ocular flow) occurs, a feedback loop of heightened stimulation may result. Activation clusters within the subcortical/infratentorial structures of the brainstem, primarily pontomedullary and hemispheric and midline cerebellar substructures [17], are the primary sensory integration hubs that control and modulate the OKS-mediated ocular motor phases (Fig. 1). The cascade of neuronal activity begins with vestibular nuclei receiving simultaneous acceleration-related afferent end organ data signaling from the inner ear (e.g., ipsilateral head rotation or stability) and eyes (e.g., contralateral visual field) regarding OKS-related motion [18]. The outgoing subcortical signals from this initial integration projects to multiple cortical regions for further processing and modulation [19••]. In order to generate effective optokinetic ocular motor phases, these cortical areas (e.g., corticovestibular, corticocerebellar, corticovisual) are separate from each other, leading to a deactivation-dominate reciprocal inhibition process within the cortical network system proportional to the level of incongruent visual-vestibular interaction and ensuing perceived mismatch [18, 20–23]. For example, it has been reported that OKS-related error signals lead to overall deactivation of the vestibular cortices while simultaneously exciting bilateral medial parieto-occipital areas [18].

Postural Control

As reviewed above, postural instability has been studied extensively with respect to sway during static and dynamic tasks. However, the illusion of self-motion as it relates to postural control has received much less research attention. Mal de debarquement syndrome (MdDS) is a chronic (e.g., > 1 month) symptomatic condition of perceived self-motion (e.g., rocking, swaying, bouncing) while walking or in a resting state. Historically, MdDS has been recognized as a residual effect of prolonged exposure to external movement patterns of the head and body, typically at the 0.2–0.3 Hz range, that is experienced after "disembarking" from a boat or car, hence the term "debarquement." However, more recent reports acknowledge the presence of MdDS in non-motion-mediated situations, including psychopathologies (e.g., depression, stress, anxiety), which have been recognized as both causative and as resultant disorders of MdDS [24, 25]. The suggested neuronal underpinning of MdDS resides in the vestibulocerebellar circuitry, with inappropriate activity of the prolonged velocity storage integrator initiated by repeated motion stimuli. Moreover, it is also reported that OKS optic flow pattern exposure also may be linked to MdDS, especially as it relates to spatiotemporal processing and orientation [26•].

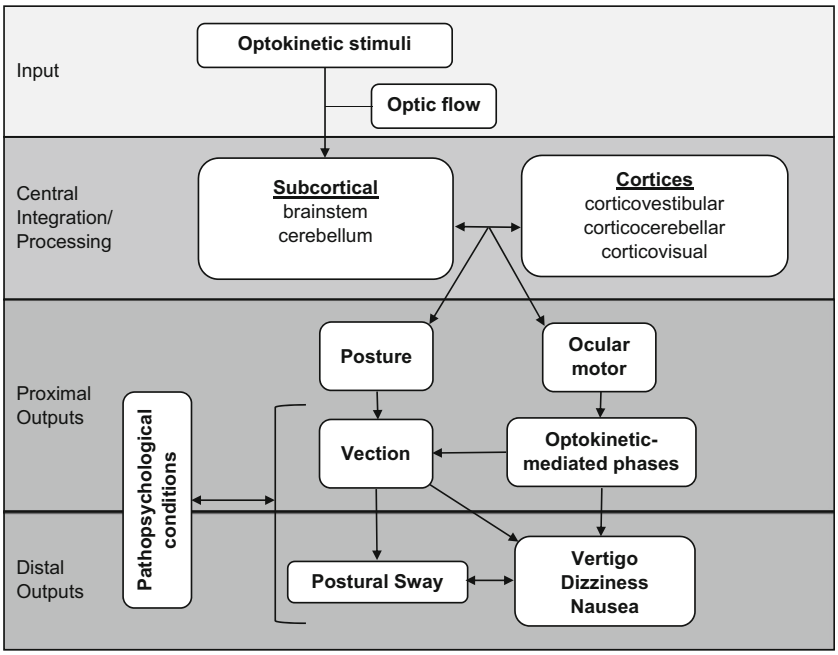

Fig. 1 Flow diagram of the system by which visual motion stimuli are processed and the pathological conditions (such as postural sway) that may occur when inappropriate processing and efferent output occurs

Postural control-related clinical manifestations are also generated during OKS-mediated visual-vestibular acceleration conflict while maneuvering in open environmental space (e.g., walking) or at a physiological static postural state. Vection, the illusion of self-motion, is frequently endorsed when subjects are exposed to large optic flow OKS-related motion patterns; this illusory movement subsequently acts as the precursor to quantifiable directional posture sway patterns. Optokinetic-induced vection (OKV) is when the observer of motion in the field of vision perceives body movement and falsely concludes that visual field environment is stationary. In concert with OKS-mediated ocular motor phases, visual motion stimuli on the peripheral retina principally dictate direction or type of OKV [21]. Although this retinal coordinate modeling has been consistently supported as the main driver of optokinetic response and vection, other spatial coordinates such as that may be derived from proprioception (motion of self during observed OKS) have been reported as an additive factor [27]. There are three types of OKV. Circular OKV is induced by large visual field motion moving around the observer, typically in horizontal (H-OKV) or vertical (V-OKV) cardinal directions. H-OKS patterns rotate about the vertical axis and V-OKS patterns rotated about the interaural axis. Linear vection is generated by optic flow stimuli approach or receding within the observer's visual field. Roll vection is initiated from visual motion stimuli rotating around the observer's center visual point.

The perceived direction of vection by the observer is dependent on OKS-mediated optic flow direction. When exposed to upward or downward circular flow patterns, the directional sense of the observer is initially in the same direction as the pattern flow without reversal. Thus, when viewing upward optic flow patterns, V-OKV results in a sensation of backward vector push or leaning. Downward pattern V-OKV causes the observer to feel a forward vector pull or lean. However, for horizontal circular flow patterns, the direction of H-OKV initially is in the same direction of the optic flow pattern; however, reversal of this preliminary H-OKV dominates with the observer perceiving a postural lateral vector pull opposite of the direction of the flow pattern. Quantifiable changes in postural control due to OKS exposure are commonly measurable, and Hoppes and colleagues reported recently that optic flow exposure elicited increased postural sway in participants with and without OKS-related hypersensitivity. However, those subjects endorsing hypersensitivity had approximately 38% greater sway than those who did not [19••].

Non-pathological influencers of other vection characteristics have different origins. Active suppression of OKS-mediated ocular motor phases through visual fixation on a stationary point (OK nystagmus suppression or cancelation) within the visual field by the observer reduces the latency of vection and lessens the time between initial OKS exposure and onset of vection [21]. Increased velocity [28] and density or complexity [29, 30] of the OKS augments vection intensity and/or duration as do the additional demands of central sensory integration from other end organs including the auditory system [31].

Pathology

In response to OKS, non-diseased persons can operationally re-weight sensory input from the somatosensory and/or vestibular end organ systems to optimize central sensory

integration processing, imparting effective and efficient asymptomatic place-in-space homeostasis. However, various peripheral and central pathologies disrupt this required functional adjustment, often leading to persistent visually mediated vertiginous outputs including impaired correction and adaptation to postural control perceptions of vection and OKS-mediated ocular motor phases. This proximal impairment in turn leads to the advancement of the distal outputs of large postural sway patterns and symptoms of dizziness, vertigo, or nausea. Peripheral abnormalities, especially involving the vestibular end organ, may result in visual motion hypersensitivity; individuals become abnormally dependent upon the visual system for posture and balance control, leading to an excessive response to variations in visual field motions. Central vestibular abnormalities also are associated with sensitivity to OKS. Central origin of OKS-related hypersensitivity has also been reported in those without a diagnosis of a central disease. Pollak and colleagues reported 37.5% of patients without neurological, psychiatric, cardiovascular, or pharmacological etiology of dizziness and OKS-related hypersensitivity presented with scattered cortical hemispheric white matter abnormalities compared to 7% in controls without OKS-related hypersensitivity ($p = 0.009$) [32]. A potential confounder in this study was an age-related increase in prevalence of white matter lesions in the study population (31% for age 40–49 vs 83% for age > 70), as 67% of the OKS-related hypersensitivity group was thought to have symptoms related to a vascular cause [32]. Furthermore, it was theorized that a global multifocal distribution of white matter abnormalities was likely more predictive of OKS-related hypersensitivity than the specific etiology of the white matter lesions themselves. However, these findings were not replicated in a recent imaging trial comparing patients with OKS-related hypersensitivity to healthy controls [33]. OKS-related hypersensitivity is found in defined central disorders, especially when related to conditions that cause multifocal brain dysfunction such as traumatic brain injury [34, 35•]. Wright and colleagues found OKS-related symptoms were greater in patients with mild traumatic brain injury compared to healthy controls ($p = 0.020$) [36••]. Similar to MdDS, psychopathologies such as depression and anxiety [37, 38] have been associated with OKS-related hypersensitivity.

OKS-mediated hypersensitivity, also referred to as visual vertigo (VV), is often confused with other ocular motor-related symptoms including oscillopsia; differentiating the two is crucial since their diagnosis and management are quite distinct [39]. Oscillopsia is also an illusionary disorder; however, instead of the perception of vection (self-motion) experienced for patients with VV, oscillopsia is the sense that the visual field is in motion or unstable. The onset of oscillopsia most often is while body or head is in motion, however it may also occur while at rest. Though VV and oscillopsia share similar symptom outputs (e.g., dizziness, vertigo, postural sway, anxiety), the neuronal mechanisms of oscillopsia are somewhat more specific as compared to VV. Abnormal ocular motor and vestibulo-ocular reflex function leading to impaired suppression of visual afferent signaling are the main physiological causative factors of oscillopsia [40]. As such, it is important for the clinician to differentiate oscillopsia and VV and to understand the underlying causes of the symptomatology in order to accurately prescribe best intervention approaches.

Treatment

Pharmacological approaches to VV treatment are limited. Recently, acetazolamide was shown to improve a single VV environmental stimulus-related symptom, "riding as a passenger in car," compared to patients who did not initiate the drug therapy [41]. However, significant differences were not found for eight other VV symptoms, and the authors could not exclude a placebo effect [41]. Indirectly, drug therapy aimed at suppressing symptoms (e.g., vertigo, depression) due to a variety of vertiginous disorders can also be considered; however, their efficacy is less predictable for patients with VV and their specific etiologies. Physical medicine approaches have some demonstrated efficacy as well. Stationary visual fixation within the OKS visual field is a strategy that reduces symptoms such as nausea [21] and OK-mediated ocular motor response and vection gain or intensity [42•]. OKS optic flow exposure as an additive component of habituation vestibular rehabilitation has been shown to be efficacious for patients with VV. These additional habituation exercises have been shown to decrease VV symptoms when incorporated into rehabilitation regimens [43, 44]. Immersive virtual reality approaches and technology have also resulted in improved VV symptoms [45]. Furthermore, the task-specific, experiential approaches of standardized or customized vestibular rehabilitation training similarly fosters the plausibility of central adaptation of the subcortical-cortical network. Such modified constraint-induced, forced-use concepts (e.g., eyes closed, compliant support surfaces), similar to what is reported in stroke rehabilitation [46], may lead to improved vestibulocerebellar and vestibulo-proprioceptive responses that will improve the ability to override visual error signals and oppose vection. To this point, OKS exposure should be combined concurrently with upright postural control challenges progressing from static (standing) to dynamic (walking), a varied base of support alignment, and with and without static visual fixation.

Due to importance of visual sensory input for effective central sensory integration and body coordination and postural control, vision therapy may be useful to remediate or enhance ocular motor-mediated visual accuracy and endurance. Convergence function for near and far point fusion can be trained using such readily accessible equipment such a Brock String and beads, along with the use of Marsden Balls, which can also be used for visual tracking training.

Dynamic visual acuity training to improve the functional capacity of the vestibulo-ocular reflex is a common task in vestibular rehabilitation but should be considered as an important function in the arena of vision therapy for patients with VV and vection. Compensatory approaches may be required as well. Adjustments of daily activities to reduce chronic exposure to conflicting or complicated visual tasks may be required. For example, the requirement of binocular function during prolonged static near-point work (e.g., computer screens, handheld devices) results in accumulative contraction-related strain on the vergence system to hold fixation, leading to ongoing break of visual function and error signals [47]. Overall, coupling vestibular and vision therapies appears to be the most effective strategy to yield the greatest benefit for patients with chronic VV and vection. We are employing this methodology in a cohort of patients with mild traumatic brain injury and are in the process of determining efficacy and durability of the training.

Conclusions

Static and dynamic postural control may be impaired when visual information is either unavailable or processed inappropriately. Afferent visual dysfunction, in which part of the visual field is either absent (scotomatous) or ignored (neglect), may lead to perceived shifts in the visual environmental position and/or spatial orientation and cause inappropriate alteration of gait and station. When the visual field and acuity are intact but visual fixation is disrupted, or the normal feedback systems that dampen real or perceived motion are inappropriately activated, patients may experience an illusory sense of body movement in response to a moving visual milieu. In both situations, postural unsteadiness, vertigo, and even falls may occur. Because a specific peripheral or CNS lesion may not be identified, rehabilitative strategies have been designed to retrain patients in their visual environments and thus overcome the drivers of postural and gait dysfunction.

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Compliance with Ethical Standards

Conflict of Interest Jeffrey R. Hebert and Prem S. Subramanian each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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