Discussion about Visual Dependence in Balance Control: European Society for Clinical Evaluation of Balance Disorders

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INTRODUCTION
The ESCEBD has been meeting annually in Nancy, France, since 2005. It aims to discuss themes related to equilibrium that are not yet clearly defined or standardized [1, 2]. One of the latest discussions attempted to define and understand the concept of visual dependence and also attempted to develop parameters that could be used to diagnose this entity. The following is a brief report of our discussion.

BACKGROUND
Internal orientation in space and motion in humans are influenced by centrally integrated visual, vestibular, and proprioceptive inputs. On aging, the quality of all sensory inputs decreases. As a result, the elderly have a decreased ability to acquire and utilize each sensory input. Moreover, the senescence of somatosensory and vestibular information may make visual information more relevant (i.e., “preferred” input). Several studies have shown that aging is accompanied by an increased Romberg’s quotient; this indicates that a greater reliance or weighting is being placed on visual information [3-8]. However, the onset of in-
creased visual dependence or reliance does not suggest pathology by itself. This increased visual dependence can put individuals at risk of developing many individual symptoms and limitations, particularly in the group of patients we regarded as having “vestibular hypersensitivity.” The presence of vestibular hypersensitivity is not necessarily a pathological situation, but it renders a patient more susceptible to becoming symptomatic from a small difference in visual and vestibular inputs.

Another confounding factor is that in the presence of vestibular pathology, increased reliance on visual information is part of the compensation process; this can generate symptoms [3].

DEFINITION OF VISUAL DEPENDENCE
The concept of “visual vertigo” was first described by Erasmus Darwin in 1797 [7]. Since the 1950s, the term “visual dependence” has been used to describe inappropriate reliance on visual cues in postural behavior in situations where it might be better to use somatosensory or vestibular inputs for balance. In the literature, various terminologies that include hypersensitivity (intolerance) to visual stimulations were proposed. “Phobic postural vertigo” is the term that was originally described by Brandt [8]. In 1995, Bronstein [9] suggested that it was the expression of enhanced visual dependence in patients with an undetected vestibular problem and coined the term “visual vertigo.” Other terms that are clinically used include “space motion discomfort” and “visual vestibular mismatch” [10]. The term “visually induced dizziness” has recently been introduced as an all-encompassing term [10], but mechanisms underlining the concept of visual dependence and how and why it develops are still poorly understood.

Visual dependence induces symptoms generated by static or moving visual cues in the environment (i.e., related to spatial orientation). These symptoms can be present alone or together with intolerance to passive/active body movements. Visual dependence can also result in signs, such as measurable imbalance, which are often described by patients as an “inherent sense of instability” [11]. Visual dependence is associated with other symptoms or part of syndromes, including vestibular diseases, anxiety, motion sickness, and migraine. It can also be an accompanying element to disorders such as agoraphobia, but ascribing these symptoms as having psychiatric origin should be avoided [12].

It is unclear how “visual dependence” develops, but we define it as “reduced ability to disregard visual cues in complex or conflicting visual environments (e.g., height vertigo, crowd, traffic, supermarkets, etc.).” In brief, the reasons for the age-related development of visual dependence may correspond to the reduced ability to use visual inputs in a proper manner. Symptoms and signs can be provoked by an overabundance of visual cues in certain visual environments that was previously coped with.

FACTORS TRIGGERING VISUAL DEPENDENCE
Vestibular hypersensitivity by itself is an intolerance but instead is the inability to suppress stimuli that are environmentally inaccurate (e.g., moving train, 3D movies). The origin of visual dependence is unclear; symptoms can be generated by an assortment of pathological situations. The ESCEBD panel agrees that visual dependence is facilitated by the following:

1. vestibular end organ diseases, e.g., vestibular neuritis, that provoke an increase in visual contribution in the sensory integration process, often leading to difficulties in resolving various spatial daily situations where the whole body is implicated
2. vestibular migraine (which may be hypersensitivity) or conflicting visual input
3. psychiatric situations (i.e., environmental situations including panic and anxiety, which may be generated by stimuli such as watching the movement of water).
4. brain trauma (including blunt force trauma)

We feel that there are other non-pathological situations that can be implicated as triggering factors as visual dependence may also develop without any predisposing condition. In motion sickness (an inadequate response in certain individuals to environmental situations), vision is an important triggering factor and symptoms are facilitated in visually dependent subjects.

DISCUSSION
As outlined by several groups [13-16], visual dependence can suggest the presence of vestibular pathology. However, on aging, the level of visual dependence also increases [17]. It was initially suggested by Paige [18] that visual dependence resulted from a differing signal between two sensory inputs, visual senescence and adaptive plastic mechanisms, which normally maintain the vestibulo-ocular performance under conditions of head movement. If the senescence of these two systems did not occur in parallel, a resulting mismatch (and the possible development of visual dependence) in elderly patients might occur. Paige supported this by showing that the rate of caloric abnormalities was similar in elderly and younger patients with documented vestibular abnormalities. He suggested that the developed mismatch in both groups was similar (i.e., nonparallel senescence in elderly patients “mimicked” vestibular loss in younger patients). We agree that this decrease in the quality of sensory input is a factor that increases reliance on visual cues.

CLINICAL DIAGNOSIS AND ASSESSMENT OF VISUAL DEPENDENCE
As visual dependence creates symptoms and signs resulting from overweighting (or over-reliance) of visual inputs in the balance maintenance strategy, quantifying visual dependence should be possible using standardized assessments that employ visual stimuli such as optokinetic stimulation during standing eyes open, the rod-and-frame test, computerized dynamic posturography (e.g., conditions 3 and 6 of Equitest™ or similar testing on other equipment), or referenced visual stimuli in virtual reality conditions. However, currently, there is no correlation between symptoms and test results and only minimal correlation between the various testing methods reported in the literature. ESCEBD members involved in this discussion agree that no specific test for visual dependence exists to date and that this condition would be best evaluated using questionnaires.

There are three important points that we feel need to be emphasized:
1. Visual dependence frequently occurs in patients with a history of past vestibular disease but pathology does not have to be present as motion sickness and many other factors can generate these symptoms. A neurovestibular examination can display a past (inactive) peripheral vestibular deficit but is most often normal.
2. The symptoms of visual dependence can be generated by the actual compensation process (i.e., a conscious increase in reliance on visual stimuli can elicit symptoms).
3. The symptoms of visual dependence are often part of “chronic subjective dizziness” syndrome (15), which is a physical and not a psychiatric entity.

TREATMENT OF VISUAL DEPENDENCE
There is currently no standardized rehabilitation technique for visual dependence. Vestibular exercises are useful and help improve balance control. Optokinetic stimulation and virtual reality are tools that can often help, and it is thought this is accomplished by inducing symptoms and “habituation” a patient (16). Both may be used as therapies, and both aim to lower visual sensitivity and restore central visuo-vestibular integration. Improvement in symptoms can be consolidated with a program of progressive confrontation to disabling situations. Learning from fighter pilots or rally car navigators might open new perspectives (17).

CONCLUSION
Visual dependence corresponds to hypersensitivity to moving or conflicting visual stimulations that can produce disabling vertigo/dizziness symptoms in triggering situations. Mechanisms underlining the symptomatology are unclear but likely imply the overweighting of vision in the equilibrium multisensory integration strategy. Symptoms frequently occur as part of syndromes including past vestibular disease and certain anxiety-related disorders, but the symptom set is not a psychiatric diagnosis or condition. In patients with visual dependence, an extensive history has to be obtained by a clinician familiar with the symptom set. These patients have a genuine disease, and their symptoms should not be regarded as having psychiatric origin. Standardized testing and pertinent questionnaires need to be developed to help us understand how to help such patients cope.

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