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Chapter 4

Multisensory integration in balance control

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Abstract

This chapter provides an introduction to the topic of multisensory integration in balance control in, both, health and disease. One of the best-studied examples is that of visuo–vestibular interaction, which is the ability of the visual system to enhance or suppress the vestibulo-ocular reflex (VOR suppression). Of clinical relevance, examination of VOR suppression is clinically useful because only central, not peripheral, lesions impair VOR suppression. Visual, somatosensory (proprioceptive), and vestibular inputs interact strongly and continuously in the control of upright balance. Experiments with visual motion stimuli show that the visual system generates visually-evoked postural responses that, at least initially, can override vestibular and proprioceptive signals. This paradigm has been useful for the study of the syndrome of visual vertigo or vision-induced dizziness, which can appear after vestibular disease. These patients typically report dizziness when exposed to optokinetic stimuli or visually charged environments, such as supermarkets. The principles of the rehabilitation treatment of these patients, which use repeated exposure to visual motion, are presented. Finally, we offer a diagnostic algorithm in approaching the patient reporting oscillopsia – the illusion of oscillation of the visual environment, which should not be confused with the syndrome mentioned earlier of visual vertigo.

A GENERAL INTRODUCTION TO SENSORY INTERACTIONS IN BALANCE CONTROL

The main sensory inputs underpinning spatial orientation and balance control are provided by the visual, the vestibular, and the somatosensory, mostly proprioceptive, systems. The vestibular system is the more frequently discussed in this book because vestibular disorders are the main cause of dizziness and vertigo. However, from the point of view of spatial orientation under normal circumstances the main players are vision for spatial orientation and proprioception for balance. In this section we will use simple intuitive examples, albeit all backed up by experimental evidence, to introduce essential concepts of multisensory organization of the balance and spatial orientation systems. These principles are necessary not only for understanding how balance works but also for grasping the basic principles of balance rehabilitation.

There is a considerable degree of overlap and redundancy in the vestibular, visual, and proprioceptive sensory systems (Peterka, 2002). One can tell if one is upright by the pressure on the sole of the feet, the tension in the ankle muscles, the static gravitational vestibular input (otolith), or by viewing that objects such as buildings or trees in the environment look properly upright. Despite this overlap in the "message" conveyed by the various inputs there are major differences in the functions played by each of these systems. The vestibular apparatus is the only system solely dedicated to the detection of head motion (angular = semicircular canals; linear- = otoliths) and head position with respect to the gravitational vector (otoliths) (Fernandez and Goldberg, 1976). Its mechanical inertial properties, akin to those of engineering accelerometers, guarantee that only during real movements (accelerations) of the head will their sensory epithelium signal motion. These mechanical properties

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of the vestibular system are equally useful to signal absence of head movements, for instance, proprioceptive signals are virtually the same whether the head turns upon the trunk or the trunk turns under the head. It is the absence of dynamic vestibular signals during the latter that allows the brain to establish that the trunk, not the head, has turned. This may partly underlie the enhancement or disinhibition of neck reflexes, like the cervico-ocular reflex, when animals and humans lose vestibular function bilaterally (Bronstein and Hood, 1986; Popov et al., 1999; Yakushin et al., 2011).

In contrast to the vestibular system, the visual system is particularly susceptible to being "tricked" into interpreting that one is moving with respect to the environment whenever large portions of the visual environment move (Kleinschmidt et al., 2002). This visually elicited illusion of self-motion is called vection, as happens during the railway illusion, where we think our train has moved when in fact it is the train next to ours that has pulled out. Hence, from the point of view of spatial orientation, the visual input is said to be ambiguous as it can equally signal self or surroundings motion. In such circumstances of sensory ambiguity the inertially based sensory systems are required to "disambiguate" this situation by informing the central nervous system (CNS) that, despite the visual system signaling body motion, this could not be confirmed by the vestibular or proprioceptive systems. In this regard the CNS is said to have comparators, that is, neural mechanisms that bring together sensory signals from different receptors and examine how well matched these signals are (Wolsley et al., 1996). If these various inputs are not proportional or matched to each other, a "warning" signal is generated alerting the organism of unusual or unexpected sensory conditions, which in turn leads to further action via automatic or voluntary mechanisms, e.g., looking out of the train window at stationary objects to see whether we are moving or not.

This leads to the concept of sensory weighting, namely how much "weight" the CNS places on each individual system at any one time (Asslander and Peterka, 2014). In most natural environments the three sensory inputs are synergistic and congruent. For instance, if you are standing upright and somebody pushes sideways from your right, the CNS will be informed by the visual system that you have moved to the left because you see the world shift to your right by the proprioceptive system, because muscles on the right side of your body have stretched, and by the vestibular system because your head has accelerated towards the left. However if you were standing in total darkness when the push occurs then you can only rely on the inertial systems (vestibulo-proprioceptive) which then become upregulated; that is, they are given more functional weight by the CNS. Similar mechanisms also operate in disease, therefore allowing for central compensation of sensory deficits; for instance, in a patient who suffers bilateral vestibular failure the remaining sensory inputs (vision and proprioception) also become upregulated (e.g., vision: Bronstein et al., 1996; proprioception: Bronstein and Hood, 1986).

Despite the considerable degree of overlap amongst these three sensory systems, their functional specificity remains. This is provided by their intrinsic anatomic structure which in turn determines the optimal operational frequency range of their peripheral receptors. The best examples are the high-frequency preference of the vestibular apparatus and the lower-frequency preference of the visual system (Barnes, 1983) and this will be illustrated yet again with the railway illusion. If you have experienced the sensation that your own train pulled off when in fact it didn't, you will have noticed that this illusion (vection) is only induced when you are exposed to a very gentle visual acceleration provided by the neighboring train. At low frequencies and accelerations levels, that is, when outside its optimal frequency range, the vestibular system is unable to confirm whether there is or there isn't head motion. In such situations, the CNS accepts or "trusts" the visual signals indicating self-motion because they have been collected at the visual system optimal-frequency range. In contrast, we don't experience vection when a fast train passes by because, at such high-frequency/acceleration range, the vestibular system is "trustworthy" and the CNS accepts the absence of vestibular signals as indicative that no self-motion has occurred. Experimental and modeling approaches supported these conclusions (Carver et al., 2006).

The frequency specificity of the visuovestibular systems is further illustrated by this simple experiment. Hold the page that you are reading at arm's length and read it. Then oscillate your head side to side ("no-no") at approximately 1–2 Hz (5–10 cycles in 5 seconds) and continue to read. You will notice that you can still read pretty well. Then keep the head stationary and oscillate the page in front of you at the same rate – now you will notice that you are no longer able to read. This illustrates that the vestibulo-ocular reflex (VOR) is capable of operating at high frequencies of motion but that visual pursuit, like all visually driven systems, is not – it only works well at lower frequencies of target oscillation. Figure 4.1 presents quantitative data of the original experiments proving this.

These differences in the frequency capacity of the visual and vestibular systems are partly due to the longer latency of the visual pathways (many synapses) than the VOR (two synapses), but also to the biophysical characteristics of the peripheral receptors. Think of the canal-endolymph system as a bowl filled up with soup. If you rotate the bowl extremely slowly, the soup and bowl will move together with no differential velocity between

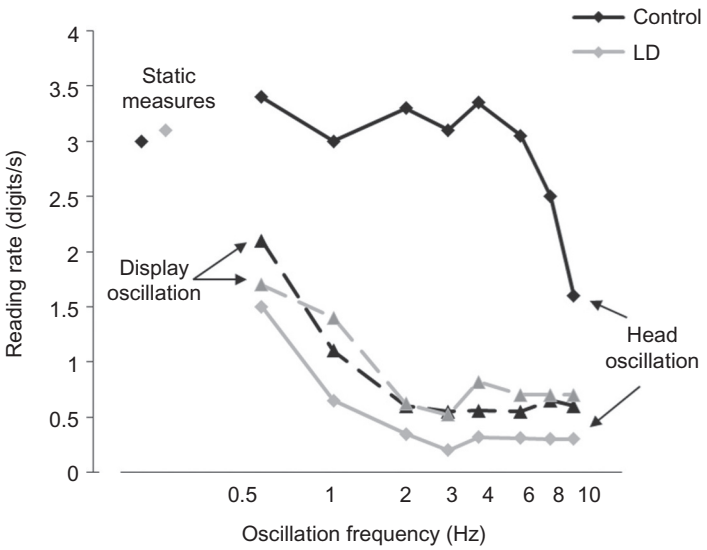

Fig. 4.1. This figure compares reading ability (*y*-axis) as a function of oscillation frequency (*x*-axis) in two experimental conditions: during oscillation of the visual display (eliciting pursuit eye movements) and during head oscillation (eliciting vestibulo-ocular eye movements). Note the considerably higher visual performance during head oscillation except for one labyrinthine-defective subject (LD, illustrated in light gray). In this subject, reading performance during head oscillation and target oscillation are the same because, in the absent of vestibulo-ocular reflex, he can only follow the visual display with pursuit eye movements. In the group of normal subjects, the frequency response of the vestibulo-ocular reflex (head oscillation) is much higher than that of pursuit movements (visual display oscillation). (Courtesy of Graham Barnes, reconstructed from Barnes, 1983.).

the two – cupular deflection would not occur in this case. If you turn the bowl faster you will be able to see the soup rotating slower than the bowl and this differential velocity would induce cupular deflection. In summary, the canal-endolymph system is not very efficient at signaling low-frequency or acceleration motion. More generally, frequency content is partly relevant to a fundamental question of vestibular physiology: how does the brain distinguish linear acceleration, which might require a compensatory eye movement response, from gravity, which usually does not require an eye response (Merfeld et al., 1999; Kingma and Janssen, 2013; Clark et al., 2015).

Finally, we will illustrate the concept of sensory conflict, which was introduced above with the example of the railway illusion. In this example it can be said that the visual input (the train moving on the next track) is in conflict with the vestibular and proprioceptive signals (which signal no real body or train motion). However, the most common example of a specific visuovestibular conflict is illustrated by Figure 4.2, showing two passengers seated on a bus. When the bus is turning, a passenger reading a newspaper will experience sensory conflict as the visual system will not confirm the head-turning input provided by the vestibular system. Such conflict situations, as many of you would have experienced personally, often lead to nausea and motion sickness feelings. If the passenger is however looking outside the bus the visuovestibular conflict is resolved because both sensory inputs are now congruent. Hence the advice given to passengers to prevent or delay motion sickness by seeking view of the horizon looking out of windows or going up on deck if on a ship (Murdin et al., 2011).

So far, most concepts discussed have been kept within the boundaries of the sensory systems. However, even for low-level brainstem reflexes, sensory explanations alone are insufficient to understand the general functional principles of the balance system. The motor system is inextricably linked to balance and postural control and we must remember that descending modulatory inputs from higher levels in the brain are at least as important as local or segmental reflexes. The head–neck system can illustrate this point. Neck and head stability are subserved by two reflexes, the neck-afferent based cervicocolic reflex or CCR, which stabilizes the head upon the trunk, and the vestibulocolic reflex or VCR, which stabilizes the head in space. How do these two reflexes interact during body movements? In certain conditions both these reflexes are synergistic. For instance, if somebody pushes your head forwards, the stretching of the neck extensors will elicit the CCR and the head will realign with the body. In parallel, the head push will also result in head rotation and tilt, eliciting simultaneous semicircular canal- and otolith-mediated vestibulocolic reflexes (VCRs), restoring the head to its upright position. In this example both VCR and CCR work in agreement or synergistically. Think now of a person tripping on the road

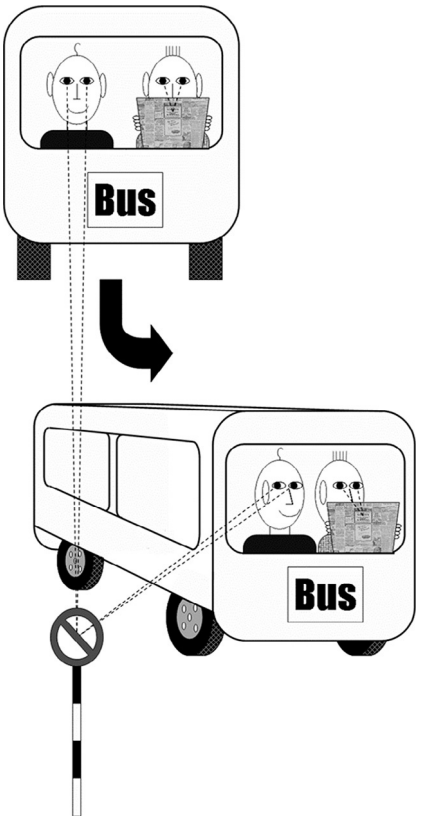

Fig. 4.2. The subject sitting on the right-hand side of the bus is looking outside and thus, as the bus turns, he senses the turn both by the vestibular system and the visual system – these two sensory signals are congruent. The subject sitting on the left of the bus is reading the newspaper which moves with him and so, when the bus turns, his vestibular system indicates a turn but his visual system does not – the visual and vestibular inputs are now said to be in conflict. (Modified from Bronstein and Lempert (2007), with permission from Cambridge University Press.)

and falling forwards. As the trunk tilts forwards, the VCR will tend to realign the head to the gravitational vector, whereas the CCR will tend to work in the opposite direction and realign the head with the trunk. As the net head motion cannot be resolved as a "tug of war" between CCRs and VCRs, central descending inputs will modulate the final pattern of neck muscle contraction according to general context ("what is needed now?") and constraints such as avoiding a head injury. Another example of VCR and CCR top-down modulation takes place during voluntary head movements when descending influences will have to switch off both CCRs and VCRs so that the head can actually turn (Peterson et al., 1985; Peng et al., 1999). Such internal neural actions are thought to be mediated by efference copy mechanisms and, indeed, brainstem neurons coding for such elusive internally generated signals have been found (Cullen, 2014).

Finally, the influence of context in balance control can be linked to cognition and the tuning of a particular movement response to a specific environment, internally represented in our brains on the basis of multiple sensory inputs. In the area of balance, context can modify vestibular reflexes, for instance, otolith-ocular responses operating at short latency which, in order to be functionally powerful, require appropriate visual context and convergence cues (Gianna et al., 1997; Misslisch et al., 2001). Other examples of clinical relevance are the profound modifications, both in simple vestibular reflexes and complex balance tasks, induced by fear and postural threats, where vestibular reflexes become weighted up if a risk of fall is perceived (Horslen et al., 2014). It is easy to see how, through this top-down modulation, high-order, cognitive, and emotional influences are capable of interacting with balance and postural function in health and disease.

We will now apply some of the concepts briefly presented in this introduction to a more specific, clinically relevant example, in order to understand how visual input interacts with balance in health and disease.

VISUAL–VESTIBULAR INTERACTIONS IN HEALTH AND DISEASE

The more studied and clinically useful form of multisensory interaction is the specific modulation of vestibular responses by visual input termed VORs. When we rotate in the dark, or in the light whilst gazing at the surroundings, a strong vestibular nystagmus is induced – the slow phase affords visual stability of the earth-fixed surroundings and the quick phases afford refixation upon different objects in the environment. However, when we focus on an object that rotates with us, the vestibular nystagmus induced by the rotation has to be suppressed to allow effective visual fixation. This is what allows us to read a newspaper on a bus, as shown above in Figure 4.2. VOR is driven by visuomotor structures and mechanisms that are closely related to smooth pursuit, as established by animal and human research. In the clinic, patients with CNS lesions disrupting pursuit usually have commensurate disruption in VOR function (Halmagyi and Gresty, 1979; Waterston et al., 1992). In contrast, patients with peripheral vestibular disease show preserved VORs and this feature is a useful pointer for separating central and peripheral vestibular lesions in the clinic. This is why the nystagmus in a patient with a peripheral vestibular lesion is much larger in amplitude, frequency, and slow-phase velocity in the dark than whilst fixating in the light.

More generally, visual suppression of vestibular imbalance is also the first line of defense against an acute vestibular disorder, and this applies not only to

nystagmus but also to postural stability. It is well known that in patients with an acute vestibular disorder eye closure, as during the Romberg test, can induce significant instability, veering or even falling towards the lesion side. This can be regarded as an initial phase of central vestibular compensation, which is visually mediated and starts to work immediately after the lesion. Gradually, brainstem and cerebellar mechanisms become dominant in vestibular compensation as the degree of asymmetry in the vestibular system is redressed (Dutia, 2010).

This early compensatory phase after vestibular lesions, where visual input is dominant, has been demonstrated in experiments with devices, as shown in Figure 4.3, which allow subjects to be exposed to large or full-field visual stimuli (Bles et al., 1983). Bles et al. found that patients with bilateral vestibular failure show enhanced responses to movements of the visual surround, particularly when tested soon after the lesion. These enhanced visually evoked postural responses gradually normalize as patients improve and adapt to their lack of vestibular input. This indicates that patients progressively attempt to downregulate the visuopostural loop and rely more on proprioception. Indeed, if similar full-field optokinetic stimulation is delivered to patients with profound proprioceptive loss but preserved vestibular function, the effects are devastating, with a consistent tendency to fall in the direction of the visual motion with virtually no adaptation to the moving visual surroundings (Bronstein, 1986).

Visual vertigo (visually induced dizziness)

Techniques using large-field visual motion stimuli have been particularly useful to understand visually related symptoms observed in dizzy patients, in particular the syndrome variably called visual vertigo (VV) (Bronstein, 1995; Guerraz et al., 2001), visuovestibular mismatch (Longridge et al., 2002), space and motion discomfort (Jacob, 1988; Jacob et al., 2009) or, as recently defined by the Bárány society, visually induced dizziness (Bisdorff et al., 2009).

This syndrome refers to those vestibular patients who report worsening or triggering of dizziness and imbalance in specific and challenging visual environments, as encountered in traffic, crowds, disco lights, and car scenes in films. Typically, dizziness and discomfort develop when walking in busy or complex visual surroundings such as supermarket aisles. The triggering or worsening of dizziness by visual stimulation in some patients with vestibular disorders has long been recognized (Hoffman and Brookler, 1978; Hood, 1980), but only in the last 20 years some research has characterized its physiologic basis (see Bronstein, 2002, for review). For a clinically oriented textbook like this one, we should

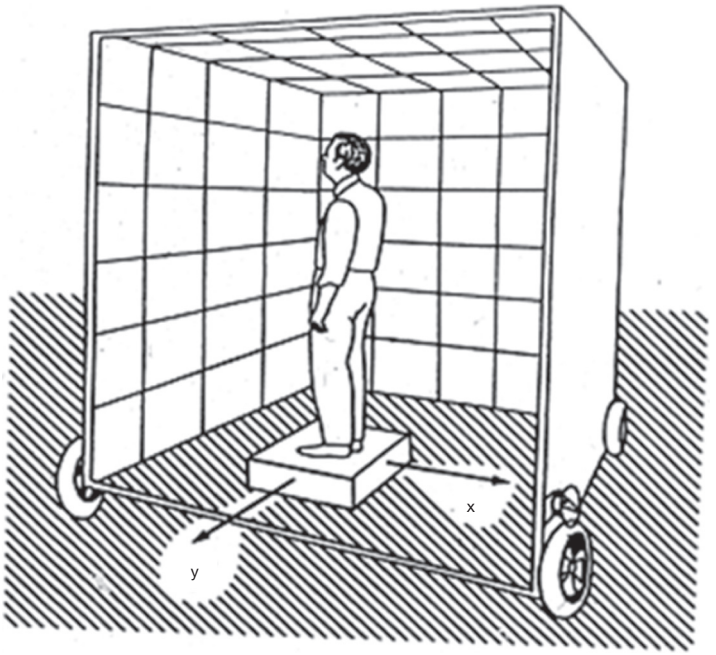

Fig. 4.3. An "optokinetic room" (modified from Bronstein et al., 1991), used for the study of visually evoked postural responses. Large-field visual motion stimulation with devices of this kind, projected images, head-mounted displays, or rotating discs, as shown in Figure 4.4, induce powerful postural sway in patients with the syndrome of visual vertigo. In turn, these devices are useful for desensitization treatment of these patients, as shown in Figure 4.5.

Table 4.1

Oscillopsia diagnostic algorithm

When does the oscillopsia occur?

1. During movements of the head?
   Absent vestibulo-ocular reflex: bilateral loss of vestibular function
   - Postmeningitic
   - Ototoxicity
   - Idiopathic
   - Miscellaneous
2. Triggered by movements of the head
   Positional nystagmus: brainstem-cerebellar disease
3. At rest (not significantly associated to movement)
   Paroxysmal
   - Sound-induced: Tullio phenomenon (superior canal dehiscence)
   - Vestibular paroxysms
     - VIIIth nerve: vestibular paroxysmia
     - Vestibular nuclear lesions
   - Ocular flutter
   - Microflutter
   - Voluntary nystagmus
   - Monocular: superior oblique myokimia

   Continuous
   - Nystagmus (brainstem-cerebellar lesion)
     - Pendular
     - Down/upbeat
     - Torsional
   - Others
   - Pseudonystagmus (head tremor + absent vestibulo-ocular reflex)

Modified from Bronstein (2004).

clarify that VV or visually induced dizziness should not be confused with oscillopsia. Oscillopsia means oscillation of the visual world – the symptom is visual. In VV, the trigger is visual but the symptoms have a vestibular flavor, such as dizziness, vertigo, disorientation, or unsteadiness. Table 4.1 provides a practical algorithm to diagnose oscillopsia at the bedside (Bronstein, 2004).

The symptoms of VV develop after a vestibular insult. A typical patient is a previously asymptomatic person who suffers an acute peripheral disorder (e.g., vestibular neuritis or benign paroxysmal positional vertigo (BPPV)) and, after an initial period of recovery of a few weeks, he/she discovers that the dizzy symptoms do not fully disappear and begin to be aggravated by looking at visually challenging surroundings. Patients may also report anxiety or frustration not only because the symptoms do not go away, but also because medical practitioners tend to disregard the symptoms or tell patients that "they are just in your mind."

The origin and significance of the symptoms of VV in vestibular patients have been the subject of research. We know that tilted or moving visual surroundings have a pronounced influence on these patients' perception of verticality and balance, over and above what can be expected from an underlying vestibular deficit (Bronstein, 1995; Guerraz et al., 2001). For instance, as Figure 4.4 shows, a rotating optokinetic disc induces more unsteadiness in patients with VV than in patients with long-term bilateral absence of vestibular function (Guerraz et al., 2001). The term used to describe an increased responsiveness to orientational or moving visual stimuli is "visual dependency." Of practical interest, patients with central vestibular disorders and patients combining vestibular disorders and congenital squints or squint surgery often report VV, show enhanced visuopostural reactivity (Bronstein, 1995), and respond less well to visuovestibular rehabilitation (Pavlou et al., 2015).

The general interpretation of these findings is that the combination of a vestibular disorder and visual dependence in a given patient is what leads to the VV syndrome. Visual input is inherently ambiguous for balance and spatial orientation because visual motion can be the result of self or surroundings motion. For this reason, relying excessively on vision for balance is never very useful, but the situation will get even worse if the person is both visually dependent and has a vestibular lesion. Ultimately, what makes some patients with vestibular disorders develop excessive visual dependence is not known. Indeed, it is possible that the syndrome

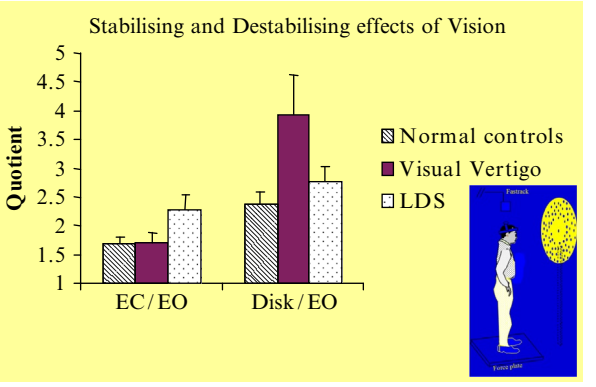

Fig. 4.4. The bars on the left show the amount of body sway induced by closing the eyes (sway with eyes closed/sway with eyes open (EC/EO), or "stabilizing effects of vision," as in the Romberg test) in three group of subjects: normal controls, bilaterally labyrinthine-defective subjects (LDS), and visual vertigo. The bars on the right show the amount of body sway induced by an optokinetic rotating disc, as shown in the inset, expressed as a ratio, sway during disc rotation/sway with eyes open with the stationary disc, disc/EO (or "destabilizing effects of vision"). The data show the disproportionate increase in body sway induced by the rotating disc in the visual vertigo patients. (Modified from Guerraz et al., 2001.)

of VV develops in vestibular patients who were visually dependent beforehand, given that visual dependence is a trait variably expressed in the general population (Witkin and Goodenough, 1981).

Recent data have shown that visual dependence is enhanced even in patients with BPPV (Agarwal et al., 2012). Interestingly, in patients with just a past history of acute vestibular neuritis (i.e., not selected for VV or poor clinical outcome), visual dependence is significantly higher in those with worse clinical outcome (Cousins et al., 2014). Visual dependence, vestibular disorders, and VV is therefore a three-way "chicken and egg"-type problem. Pragmatically, however, simple ways of identifying visual dependency in chronic dizzy patients are available, such as laptop versions of the rod and disc test (Cousins et al., 2014) and questionnaires (Pavlou et al., 2006), and this has clinical value for patient management because, as we will discuss below, specific treatments for visually induced dizziness are available.

The role of the associated anxiety/depression, often observed in patients with chronic dizziness and VV, and whether this is a primary or secondary phenomenon is not fully established either. The evidence so far is somewhat contradictory. As expected, VV is more prevalent in patients attending dizziness clinics than patients attending other clinics (Dannenbaum et al., 2011). Some studies show higher levels of anxiety in VV patients than in vestibulopathy patients without VV (Zur et al., 2015) whereas other studies report that anxiety or depression levels, as measured with established questionnaires, are not higher in unselected VV patients than in other patients seen in dizzy clinics (Guerraz et al., 2001). However, the opposite does not seem to hold true, in that one of the frequent problems reported by patients with psychogenic dizziness is VV (see Chapter 24). It is likely that pre-selection of patients in these studies, some coming from psychiatry clinics but others from neurology and neuro-otology services, is playing a part in these differences. That the psychologic and visuovestibular components in patients with dizziness are intertwined is undeniable, both as witnessed by clinical experience and by recent research from our group. In a prospective study following up patients with vestibular neuritis from the acute to the chronic stages we carried out factor analysis of many psychologic, psychophysical, and vestibular variables. The data have shown that poor clinical outcome is largely dictated by a single statistical factor which combines the results of the rod and disc test (visual dependence) with questionnaires measuring health anxiety and psychosomatic traits.

Another predisposing factor or comorbidity in patients with VV is migraine (Lempert, 2013), and the available evidence suggests that increased visual dependency, as measured with an optokinetic rotating disc, is also a feature of these patients (Furman et al., 2005b; Agarwal et al., 2012). A syndrome bringing together the individual components of migraine, anxiety, and dizziness (migraine anxiety-related dizziness (MARD)) has been described (Furman et al., 2005a), and it is a useful concept when discussing multifactorial, multisensory dizzy symptoms with patients in the clinic.

DIFFERENTIAL DIAGNOSIS

Occasionally, a hyperacute VV syndrome can arise in a patient with an acute brainstem lesion in the vestibular nuclei area, including vomiting in response to visual motion (Khan et al., 1995). However, for the reasons just mentioned above, the more important differential diagnosis in these patients is one of a purely psychologic disorder or panic attacks (Furman and Jacob, 1997). An accepted set of criteria to distinguish between psychologic and vestibular symptoms is, however, not complete at this stage (Brandt, 1996; Bronstein et al., 1997; Furman and Jacob, 1997; Staab et al., 2014). Indeed, it may be argued that such distinctions are academic because, in line with treatment of functional disorders in other areas of neurology (Gelauff et al., 2014), visually aggravated symptoms such as VV need to be treated and rehabilitated in their own right regardless of a "physical or psychologic" origin (Chapter 24; Thompson et al., 2015).

Notwithstanding these general considerations, a patient who has never had a history of vestibular disease, no findings on vestibular examination, and with visual triggers restricted to a single particular environment (e.g., only supermarkets) would be more likely to have a primary psychologic disorder. Reciprocally, a patient with no premorbid psychologic dysfunction who after a vestibular insult develops car-tilting illusions when driving (Page and Gresty, 1985) and dizziness when looking at different moving scenes (traffic, crowds, movies) is more likely to have VV secondary to vestibular disease than a psychiatric disorder. The presence of clearcut abnormalities on vestibular testing can be valuable in understanding the initial trigger that led to the secondary syndrome of VV and, as will be mentioned below, for treatment.

TREATMENT OF VISUAL VERTIGO

There are three aspects in the treatment of patients with the VV syndrome. The first is specific measures for the underlying vestibular disorder, e.g., Menière's disease, BPPV, migraine, and these will be found elsewhere in this book. However, a specific vestibular etiologic diagnosis cannot be confirmed in many patients with chronic dizziness with or without VV as, by definition, this will have to be done retrospectively.

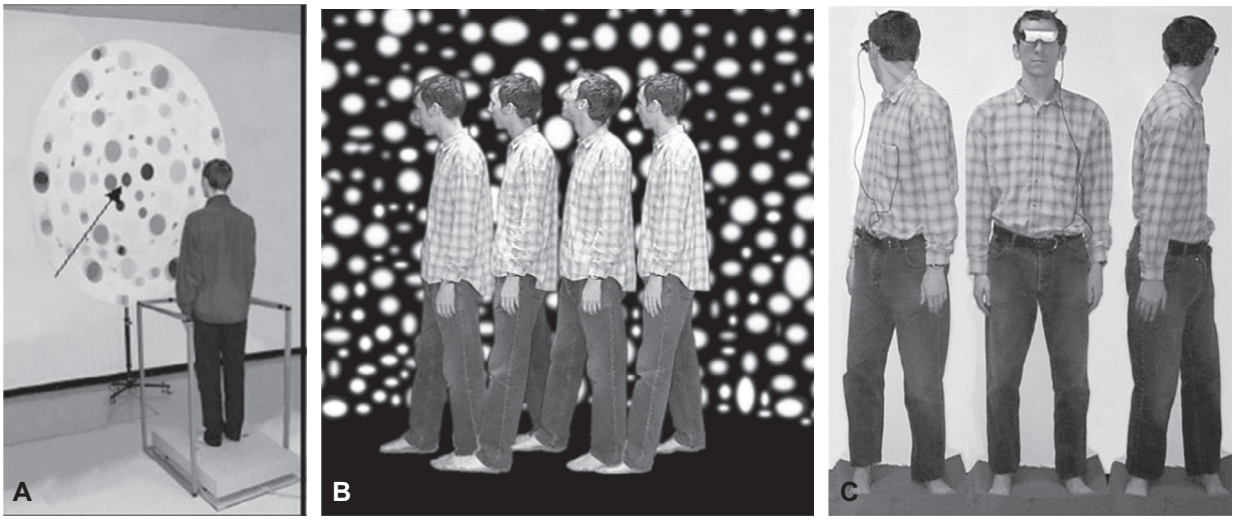

Fig. 4.5. Techniques for promoting visual motion desensitization when treating patients with visual vertigo. Patients may (**A**) fixate the center of rotation (arrow) of the rotating disc, (**B**) walk amidst moving blobs projected by a planetarium, or wear (**C**) goggles projecting moving visual senses. (Modified from Pavlou et al., 2004, with permission from Springer Science and Business Media.)

Second, patients benefit from general reassurance and vestibular rehabilitation with a suitably trained audiologist or physiotherapist. These exercise-based programs can be either generic, like the original Cawthorne–Cooksey approach (Cooksey, 1946) or, preferably, customized to the patient's needs. All regimes involve progressive eye, head, and whole-body movements (bending, turning), as well as walking exercises (Black and Pesznecker, 2003; Pavlou et al., 2004; video-demonstrated in Bronstein and Lempert, 2007).

Critically, specific measures should be introduced in the rehabilitation program in order to reduce the patient's hyperreactivity to visual motion. The aim is to promote desensitization and increase tolerance to visual stimuli and to visuovestibular conflict. Patients are therefore exposed, under the instruction of the balance therapist, to optokinetic stimuli which can be delivered via projection screens, head-mounted virtual reality systems, video monitors, ballroom planetariums, or optokinetic rotating systems (Vitte et al., 1994; Pavlou et al., 2004). The treatment approach is one of progressive difficulty both for the visual stimuli selected and for the more or less challenging postural setting adopted during the visual stimulation. Initially patients watch these stimuli whilst seated, then standing, walking, initially without and then with head movements, in a progressive fashion (Fig. 4.5). Research has shown that these patients benefit from repeated and gradual exposure to such visual motion training programs; both the dizziness and associated psychologic symptoms improve over and above conventional vestibular rehabilitation (Pavlou et al., 2004).

Finally, attention should be paid to the psychologic and psychiatric aspects of these patients. This may involve a variable range of treatments, all the way from simple physician-led reassurance and explanations on the origin of the symptoms up to antidepressant drug treatment and psychotherapy (Chapter 24).

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