Dizziness in the Elderly and Age-related Degeneration of the Vestibular System

Anna J. Matheson
Dept. of Psychology & Neuroscience Research Center, University of Otago
Dept. of Pharmacology, School of Medical Sciences, University of Otago Medical School
Cynthia L. Darlington
Dept. of Psychology & Neuroscience Research Center, University of Otago
Paul F. Smith
Dept. of Pharmacology, School of Medical Sciences, University of Otago Medical School

The peripheral and central vestibular systems exhibit an age-related structural deterioration which may be responsible for vestibular reflex deficits and dizziness in the elderly. However, it seems likely that the central nervous system is capable of compensating for a certain degree of decline in function, since not all elderly people are impaired to the extent that the clinical signs of vestibular dysfunction are apparent. Dizziness and other vestibular disorders may develop only when the degree of deterioration of the vestibular system exceeds the ability of the nervous system to compensate. If dizziness does eventuate, it can have profound psychological consequences, particularly in terms of loss of confidence in independent activity, and may lead to the development of anxiety disorders. Vestibular rehabilitation programs may help to minimise the effects of age-related deterioration of the vestibular system and its psychological impact.

Age-related dizziness and its psychological consequences

Dizziness, often described by patients as a feeling of light headedness or a spinning sensation, is a common complaint amongst elderly people (Sloane, Blazer & George, 1989). Clinically, the term 'dizziness' has no specific definition, except that it refers to some kind of altered orientation in space (Baloh, 1996a). Often 'dizziness' represents a complex combination of overlapping symptoms and it is notoriously difficult to assess (Yardley & Luxon, 1994; Baloh, 1996a). Because orientation in space relies on the integration of visual, vestibular and proprioceptive sensory information, damage to any of these systems, either at the level of the sensory receptors, or in the central nervous system itself, can potentially result in the phenomenon of dizziness. If dizziness is caused by the illusion of movement, either rotatory (ie. angular) or linear, then a diagnosis of 'vertigo' is appropriate and an imbalance in the peripheral and/or central vestibular systems, or an abnormality in their interactions with other sensory systems, is likely.

However, dizziness can also be caused by cerebrovascular, cardiovascular and psychological dysfunction, and most studies suggest that more than 50% of patients complaining of dizziness have a non-vestibular problem (see Baloh, 1996a for a review). A differential diagnosis to exclude these possibilities must be performed before a diagnosis of vestibular dysfunction can be reached (Sloane, Blazer & George, 1989; Baloh, 1995). Presyncopal dizziness, often associated with a feeling of light-headedness and impending collapse, can be caused by hyperventilation due to chronic anxiety (Baloh, 1996a). Orthostatic hypotension (ie. reduced blood pressure), caused by rapid movement to a standing position, is also a common cause of dizziness. Dizziness can be caused by vasovagal attacks, in which heart rate and blood pressure decrease, as a result of fasting, heat or fear and anxiety. Reduced cardiac output as a result of cardiac arrhythmia is another possible cause. Dizziness is commonly associated with hypoglycemia (ie. low blood glucose), drug intoxication and motion sickness (Baloh, 1996a). Psychological dysfunction, as a result of generalized anxiety disorder, panic disorder or phobias, also frequently results in dizziness (Baloh, 1995; Sloane, Blazer & George, 1989).

An increasing number of people, particularly those aged 75 and over, report experiencing frequent dizziness. A recent Dunedin-based study reported that approximately 14% of elderly subjects (aged 60 and over) living in the community experienced dizziness of a recurrent nature which was not obviously attributable to a cerebrovascular or cardiovascular cause (Matheson, Darlington, & Smith, in press(b)). This may be an underestimation, since other studies report figures as high as 30% (Colledge, Wilson, Macintyre & MacLennan, 1994).

Dizziness has been considered as a cause as well as a symptom of anxiety disorders (Yardley & Luxon, 1994). While psychological factors are thought to play a major role

in causing dizziness, these are more prevalent in young and middle-aged individuals (Drachman & Hart, 1972; Davis, 1994). In elderly people with dizziness, psychological problems are more commonly a secondary symptom of vestibular dysfunction, and may include anxiety, depression and a fear of falling. Elderly people with dizziness experience far more psychological distress than their younger counterparts (Sloane, Hartman & Mitchell, 1994). Fear of falling can place constraints on the quality of life enjoyed by elderly people and compound the feelings of anxiety and depression. As a result, daily activities may be reduced, leading to isolation, lack of confidence and lost independence (Burker, Wong, Sloane, Mattingly, Preisser & Mitchell, 1995). The consequences of falls in the elderly can be extremely serious and sometimes life-threatening (Boult, Murphy, Sloane, Mor & Drone, 1991). Dizziness may be exacerbated by increased anxiety, leading to greater chances of falling, creating a vicious cycle.

There is, at present, a lack of research on the adverse psychological consequences of dizziness in community-dwelling elderly. The true incidence may be underestimated. Elderly people may be less likely to seek medical help concerning episodes of dizziness or may feel that it is a consequence of aging that they have to live with. Boult, Murphy, Sloane, Mor & Drone, (1991) have shown that people suffering from dizziness have a higher probability of becoming disabled. However, this is dependent on age, race and level of sensory impairment, underlying the importance of adequate screening and treatment.

Katsarkas (1994) has argued that the increase in dizziness seen in the elderly may be causally related, either directly or indirectly, to vestibular dysfunction with advancing age. Since structural deterioration of the peripheral and central vestibular systems with increasing age has been documented (see below), the consequent physiological dysfunction may be contributing to dizziness and postural instability exhibited by a significant proportion of elderly.

Elderly people with recurrent dizziness may benefit from a vestibular rehabilitation program (Darlington & Smith, 1996a; Herdman, 1996). People with vestibular lesions respond well to a 10 to 12 week rehabilitation program designed to teach them ways to cope with dizziness and instability. Most rehabilitation programs involve a graded series of eye, head and body movement exercises designed to train the patient's nervous system to compensate for the impairment of normal vestibulo-ocular and vestibulo-spinal reflexes (Herdman, 1996). At each stage, the patient is asked to work only to the limit of their ability. Nonetheless, at first, the exercises may actually exacerbate the patient's vertigo, since they are essentially designed to challenge the nervous system to develop an alternative means of achieving the same ocular motor and postural stability which is usually afforded by an intact vestibular system (Yardley & Luxon, 1994). However, gradually, voluntary (saccadic and smooth pursuit) eye movements are generated which substitute, to some extent, for the deficient vestibulo-ocular reflex, and visual and proprioceptive feedback are used to develop compensatory postural responses (Herdman, 1996). Animal studies suggest that this process of compensation and adaptation involves functional and structural changes in the central nervous system (see Leigh & Zee, 1991 for a review). The rehabilitation may be coupled with relaxation training, reducing symptoms of anxiety and/or panic (Herdman, 1996). The opportunity to gain an understanding of their symptoms may allay the patient's fears and resolve any misconceptions. The overall goal is to build confidence and to allow a gradual return to normal functioning (Yardley & Luxon, 1994).

For many elderly people, dizziness of vestibular origin is not simply a fact of aging that they have to learn to live with. Clinicians should be aware of the concordance of secondary problems associated with recurrent dizziness, and of the factors linked to an increased probability of disability. There also needs to be more awareness of interventions which may improve the quality of life.

The vestibular system

The peripheral vestibular system consists of two labyrinths, each of which contains three semi-circular canals (the horizontal, anterior and posterior canals), a utricle and a saccule. While receptor hair cells in the ampullae of the semi-circular canals respond to rotatory or angular acceleration of the head, the utricle and saccule (collectively referred to as the 'otoliths') respond to linear acceleration (including linear acceleration by gravity). Sensory information from the receptor hair cells leaves each labyrinth via the vestibular nerve (ie. VIIIth cranial nerve), the cell bodies of which form Scarpa's ganglion, and vestibular nerve axonal fibres project into the brainstem vestibular nucleus complex (VNC) on the same side and also into the cerebellum (see Leigh & Zee, 1991 for a review). In the process of transducing the acceleration stimulus, the mechanical apparatus in the labyrinth converts the acceleration signal into one that is approximately in phase with head velocity (ie. a mathematical integration or a 90° phase lag on acceleration). Neurons in the various subnuclei of the VNC (ie. the medial, lateral, superior and inferior vestibular nuclei) receive different amounts of synaptic input from specific parts of the labyrinth: for example, while medial vestibular nucleus neurons receive more input from vestibular fibres which innervate the horizontal canal ampulla, neurons in the lateral vestibular nucleus receive more input from fibres which innervate the otoliths. Many physiologists and anatomists also include prepositus hypoglossi as a subnucleus of the VNC, since it receives input from other vestibular subnuclei and is intimately involved in the control of the vestibulo-ocular and optokinetic reflexes (see Leigh & Zee, 1991 for a review). Since the signal which is conveyed to the VNC is approximately in phase with head velocity, it must undergo a second mathematical integration (ie. a 180° phase lag on acceleration) in order to achieve the position signal which the ocular motoneurons require to generate a vestibulo-ocular reflex eye movement which is equal and opposite to head movement. Many researchers believe that prepositus hypoglossi is critically involved in providing this second integration (see Leigh & Zee, 1991 for a review).

Many VNC neurons exhibit a convergence of input from different parts of the labyrinth (ie. they may respond to both angular and linear head acceleration), and many also respond to movement of the visual field ('optokinetic stimuli') in a way that is complementary to their response to vestibular stimulation (ie. if a neuron responds to horizontal angular acceleration to the right, it may respond to optokinetic stimulation in a leftward direction). Visual input to the VNC arises from a subcortical pathway which includes the accessory optic tract, the medullary reticular formation and prepositus hypoglossi; however, visual information also converges on the VNC via the cerebellar flocculus and inferior olive, and a diversity of other sensory information (e.g, proprioceptive and somatosensory input from the spinal cord) is also integrated within the VNC. In addition to these inputs, VNC neurons also receive disynaptic or monosynaptic input from the contralateral VNC via the brainstem commissures, and the functional effect of this input is usually inhibition; therefore, there exists a lateral inhibitory network between the bilateral VNC which enhances the dynamic sensitivity of the response of vestibular neurons to head movement (see Leigh & Zee, 1991 for a review). The complexity and convergence of inputs to the VNC demonstrates that, even at the level of the brainstem, extensive processing of vestibular information, and integration with other sensory information, has already taken place (see Wilson and Melvill Jones, 1979; Baloh and Honrubia, 1990; Leigh and Zee, 1991, for reviews).

It is also well demonstrated that some neurons in the VNC project disynaptically to the visual and parietal cortices, and these cortical projection areas are presumed to be primarily responsible for the sensation of self-motion (see Smith 1997; Guldin & Grusser, 1998 for recent reviews). Although there may be a vestibulo-cerebello-cortical pathway in addition, the four major vestibulo-cortical pathways all involve the thalamus: either the lateral posterior nucleus, the ventral posterior nucleus, the medial geniculate nucleus, or the ventrolateral geniculate nucleus (see Berthoz, 1996; Smith, 1997 for reviews). Demonstrations of vestibular responses in the thalamus were reported as early as 1965, when Spiegel and colleagues reported vestibular-responsive neurons in the midbrain, thalamus and basal ganglia. Since these original studies, numerous others have confirmed that the thalamus is a critical relay site in ascending vestibular projections, and recent imaging studies in humans have provided additional confirmation (Smith, 1997). Whether afferents carrying vestibular information to the hippocampus arise from the thalamus, the presubiculum, the cingulate cortex, or some other area of the cerebral cortex (e.g, the parietal cortex), is still a matter of controversy (Berthoz, 1996; Smith, 1997). However, most reseachers believe that vestibular input to the hippocampus is derived from thalamic projections to the parietal cortex, which then projects fibres back to the hippocampus via the perirhinal and entorhinal cortices. However, other, more direct, connections are possible (see Berthoz, 1996; Smith, 1997; Guldin & Grusser, 1998). The precise function of vestibular projections to the hippocampus is not yet clear; however, it is hypothesised that the hippocampus may use vestibular, as well as other sensory information, in the development of place cell response patterns (see Smith, 1997 for a review).

There is increasing evidence to suggest that the vestibular receptor hair cells in the inner ear, the vestibular nerve which carries vestibular information to the brain, and the brainstem VNC itself, all undergo a certain amount of age-related deterioration which is variable between different individuals but which may contribute to the development of dizziness in the elderly.

Anatomical degeneration of the vestibular system with age

Degeneration of the sensory hair cells

Prior to the 1970's, there was some controversy regarding whether the vestibular system degenerates with age. Researchers found changes in the cochlea that were attributable to aging; however, there appeared to be no age-related changes in the vestibular labyrinth (Jorgensen, 1961; Reske-Nielsen & Hansen, 1961). This may have been due to the different techniques used by different researchers and a lack of histological consistency in the handling of post-mortem material (Jorgensen, 1961). However, by the early 1970's, Rosenhall (1973) had shown that degeneration of the vestibular system did occur with aging, reaching an approximately 40% loss by the 9th decade.

It is now known that the vestibular sensory epithelia undergo distinct changes in individuals over 70 years of age, with some changes occurring as early as 50-60 years (Engstrom, Bergstrom & Rosenhall, 1974; Rosenhall, 1973; Rosenhall, 1974; Rosenhall & Rubin, 1975). Hair cell degeneration is dispersed over the sensory epithelium, although hair cell loss is more concentrated in the central region of the crista, which is the part of the semicircular canals that contains the sensory hair cells (Rosenhall, 1973). Whereas hair cell loss in the semicircular canals would be expected to result in deficits in the sensation of angular motion of the head, hair cell loss in the otoliths (the utricle and saccule) would be expected to cause deficits in the sensation of linear motion. Unfortunately, few comparisons of vestibular reflex deficits arising from semicircular canal versus otolithic sources of vestibular information have been carried out in elderly populations; therefore, the functional consequences of these different kinds of receptor degeneration are not well understood.

In individuals over 70 there is a significant decrease in the number of hair cells in the utricle, when compared to fetuses, infants and young adults (Rosenhall, 1973). Engstrom, Bergstrom & Rosenhall (1974) studied the sensory cells of the vestibular sensory epithelia in humans at various ages. They found that, normally, there were approximately 33,100 hair cells in the utricle; by comparison, at 70-95 years of age there were only 26,100 remaining. This constitutes a reduction in hair cells of about 20% by the ages 70-95.

The saccule also shows a significant degeneration in the number of hair cells, comparable to that of the utricle,

of approximately 25% (Rosenhall, 1973). Normally, the human saccule contains approximately 18,800 hair cells; however, an elderly human has on average, 14,200 hair cells (Engstrom, Bergstrom & Rosenhall, 1974). In individuals over 60 years, the degeneration seen in the sensory epithelia of the saccule is matched by a reduction in the calcium carbonate crystals ('otoconia') which exert an inertial force on the hair cells, resulting in an otolithic membrane which is bare. This loss of otoconia occurs in the utricle but to a lesser degree than the saccule; this may be because the saccule is more susceptible due to its vertical position (Johnsson & Hawkins, 1972).

The semicircular canal cristae exhibit a larger reduction in hair cells than either the utricle or saccule (approximately 40% reduction in individuals over 70 years) (Rosenhall, 1973). This degeneration of hair cells is already distinguishable in 50-60 year olds. Hair cell loss is concentrated in the central rather than the peripheral region of the crista. Each of the three cristae average approximately 7,600 hair cells normally, compared to 4,600 in an elderly human (Engstrom, Bergstrom & Rosenhall, 1974).

Degeneration of the vestibular nerve

The reduction in the number of hair cells is paralleled by a degeneration of the vestibular nerve fibres which transmit vestibular information to the VNC in the brainstem (Engstrom, Bergstrom & Rosenhall, 1974). This reduction in nerve fibres begins in the 5th decade (Bergstrom, 1973a; Bergstrom, 1973b). A reduction of approximately 40% of fibre counts was found in individuals aged 75-85 years when compared to younger subjects (Bergstrom, 1973a; Bergstrom, 1973b). A person aged between 10-20 years had an average of 18,000 fibres compared to approximately 11,000 in a person over 80 years of age. In the elderly there is also a loss of innervation of the sensory epithelium. The diameter of the vestibular nerve fibres increases with age, from newborn to adult; however, the elderly exhibit a thinning of the fibre diameter. There is also a loss of thick fibres, in particular thick myelinated fibres, which innervate type I hair cells of the crista (Bergstrom, 1973a; Bergstrom, 1973b). The number of vestibular ganglion cells also decreases with increasing age, with a steep decline in the average number of cells at age 60 (Richter, 1980).

The presence of amyloid bodies has been detected in the vestibulocochlear (VIIIth) nerve root (Fujii, Goto, Okada, Kida & Kikuchi, 1996). The mean area of these amyloid bodies tends to increase with increasing age; their functional significance is unknown.

Large cysts containing basophilic material have also been reported within the cristae of elderly individuals. Rosenhall (1974) found that these cysts were confined to the cristae of the two vertical semicircular canals and were not present in the cristae of the horizontal canals or the utricle or saccule. He suggested that the cristae are more vulnerable to the effects of aging than the sensory epithelia (macula) of the utricle and saccule. Richter (1980) found intraepithelial basophilic inclusions in the horizontal canal cristae and the utricular macula and larger basophilic cysts

in the cristae. There was an increase in the density of these inclusions with increasing age and a decrease in the density with increasing numbers of hair cells. Richter (1980) suggested that degeneration of the hair cells creates these inclusion bodies within the nerve endings of the sensory epithelium; however, this hypothesis has yet to be confirmed.

Degeneration of the brainstem vestibular nucleus

Lopez, Honrubia and Baloh (1997) have reported a neuronal loss within the brainstem VNC of approximately 3% per decade, beginning at age 40 years. This loss was most extensive in the superior vestibular nucleus, and least in the medial vestibular nucleus. This decrease in cell number was coupled with a significant decrease in volume and neuronal density within the vestibular nucleus. An accumulation of lipofuscin deposits in the cell bodies also resulted in an increase in the number of giant neurons (>500μm2) in older people (Lopez et al., 1997).

More recently, Alvarez, Diaz, Suarez, Fernandez, Delrey, Navarro and Tolivia (1998) have reported more extensive neuronal loss within the human medial vestibular nucleus. Comparing total numbers of neurons in a 35 year-old to an 89 year old, they observed a decrease of almost 40%, as well as a decrease in the diameter of cell nuclei.

Other studies in animals suggest that aging is associated with the development of swollen, abnormal axons within the VNC, which contain high densities of neurofilaments and granular deposits (Takeuchi, Takeuchi, Muashima & Setoohshima, 1997). The functional significance of these abnormalities is unknown at present.

Vascular disease and vestibular dysfunction

One possible explanation for the structural degeneration of cells in the peripheral or central vestibular systems is that their blood supply has been compromised. The arterial blood supply to the membranous labyrinth, which contains the vestibular receptor hair cells, is provided by the internal auditory or labyrinthine artery which arises from the vertebrobasilar arterial system (Baloh, 1996b). Inside the inner ear, the labyrinthine artery divides into the anterior vestibular artery and the common cochlear artery: the former provides blood to the ampullae of the anterior and horizontal semicircular canals, the utricle and a small part of the saccule; the latter branches into the posterior vestibular artery and the main cochlear artery, the first of which supplies blood to the remainder of the saccule and the posterior semicircular canal ampulla (Baloh, 1996b). Interruption of the blood supply through the labyrinthine artery for longer than 15 seconds results in hair cell and VIIIth nerve dysfunction which, if continued, is irreversible (Baloh, 1996b). Possible causes include vertebrobasilar insufficiency due to atherosclerosis of the subclavian, vertebral or basilar arteries, or an embolism in the posterior vestibular artery. Transient ischemia (reduced blood supply) in the vertebrobasilar arterial system often causes episodic

vertigo in elderly patients (Baloh, 1996b) and recurrent ischemia could be expected to result in both hair cell and vestibular nerve degeneration. However, vertebrobasilar insufficiency is usually associated with vertigo of abrupt onset and both nausea and vomiting; therefore, the severity of the symptoms would normally result in the patient seeking medical advice. If occlusion of the labyrinthine artery occurs ('labyrinthine infarction'), the symptoms will be much more severe and cell death will occur rapidly (Baloh, 1996b).

It is also possible for vertigo to develop as a result of interruption of the blood supply to the brainstem VNC or cerebellum, which arises mainly from the posteroinferior cerebellar artery, the anteroinferior cerebellar artery (VNC and cerebellum), and the superior cerebellar artery (cerebellum) (Baloh, 1996b). Lateral medullary, lateral pontomedullary or cerebellar infarction will result in severe vertigo (amongst other symptoms) and neuronal loss; however, as with labyrinthine infarction, these kinds of major neurological events are unlikely to account for more subtle vestibular dysfunction in elderly patients without a history of neurological disorders.

Age-related changes in vestibular neurophysiology and neurochemistry

Despite the well documented age-related changes in vestibular anatomy, there is a lack of research on age-related neurophysiologial and neurochemical changes within the vestibular system. However, changes in monoamine neurotransmitters have been observed in the aged rat medial vestibular nuclei (Cransac, Peyrin, Cottet-Emard, Farhat, Pequignot & Reber, 1996). In elderly rats there is a decrease in noradrenaline; however, there is a compensatory increase in the ratio of its metabolite, 3-methoxy,4-hydroxyphenylglycol (MHPG), to noradrenaline, which might suggest that the remaining noradrenergic neurons have become hyperactive (Cransac, Peyrin, Cottet-Emard, Farhat, Pequignot & Reber, 1996). No age-related changes were found with dopamine and its metabolite, 3,4,-dihydroxyphenylacetic acid (DOPAC). Elderly rats exhibited an increase in the levels of serotonin and its metabolite, 5-hydroxyindolacetic acid (5-HIAA). The authors suggest that increased serotonin is compensating for age-related deficits in other monamine neurotransmitters (Cransac, Peyrin, Cottet-Emard, Farhat, Pequignot & Reber, 1996).

Effect of age on vestibular reflex function

Vestibulo-ocular reflexes

Responses to caloric testing of vestibular function (ie. using cold water irrigation of the external ear) have shown no consistent trend with aging (Peterka, Black & Schoenhoff, 1990). However, vestibulo-ocular reflex (VOR) testing, using a rotating chair to deliver rotatory or angular acceleration, has shown that the amplitude of VOR nystagmus (ie. repetitive activation of the VOR) is smaller and of lower frequency in elderly subjects. The VOR gain (ie. the ratio of eye velocity to head velocity, a measure of

the efficacy of the VOR) is reduced in elderly people and this reduction becomes more pronounced as the stimulus amplitude increases (Paige, 1992; Paige, 1994). There is an increase in the phase lead of eye velocity to head velocity, resulting in the eyes moving too quickly to properly compensate for head movement and stabilize the retinal image (Baloh, Jacobson & Socotch, 1993; Paige, 1992; Paige, 1994; Peterka, Black & Schoenhoff, 1990). The functional consequence of this reduction in VOR gain and increased phase lead is that 'oscillopsia', or an apparent movement of the visual world, is likely to occur whenever the head is moved. This very distressing symptom may be caused even by the small amount of head movement produced by the pulse beat. Oscillopsia is often so debilitating that those afflicted find it difficult to stand upright and have particular difficulty in any situation in which other objects are moving within their visual field (e.g, an approaching bus) (see Leigh & Zee, 1991 for a review).

Optokinetic reflexes

Relatively few studies have examined the optokinetic reflexes (OKRs) and the associated illusion of self rotation, circularvection, in elderly subjects. The OKRs are normally elicited by movement of the entire visual field, when the head is moved relative to it. The OKRs then work with the VORs to maximally compensate for movement of the visual field. One effective way of testing the OKRs in the absence of the VORs, is to place a person inside a rotating visual field (an 'optokinetic drum') which consists of vertical black and white stripes. Although the person remains stationary, within seconds they begin to feel that they are moving in the opposite direction to the movement of the visual field. This illusion is known as 'circularvection'; the corresponding illusion of linear self-motion is known as 'linearvection' (Darlington and Smith, 1996b; see Leigh & Zee, 1991 for a review).

Of those studies that have examined OKRs in elderly subjects, sinusoidal (ie. back and forth) horizontal oscillations of the optokinetic drum, rather than unidirectional drum rotation, have been used to produce optokinetic stimulation in most studies (Baloh, Jacobson & Socotch, 1993; Paige, 1994). Optokinetic reflexes have been measured by comparing the gain of optokinetic nystagmus (OKN), and slow phase eye velocity, of young and elderly subjects. Baloh, Jacobson & Socotch (1993) measured optokinetic responses to sinusoidal drum rotation at 0.05 Hz and peak velocities of 60°/s and 120°/s. Elderly subjects displayed significantly lower OKN gain than younger subjects (aged 18-39), for both stimulus velocities. Similarly, the time constant (ie. the time to decay to 66% of the peak response) of slow phase OKN velocity during optokinetic afternystagmus (OKAN) was shorter in elderly subjects. Paige (1994) used sinusoidal oscillations at a frequency of 0.025 Hz to determine subjects' ability to experience circularvection. The experience of circularvection was measured by the proportion of subjects who experienced the sensation of self-rotation over each cycle of the 21/2 cycle trial. An increased likelihood and intensity of

circularvection occurred with increasing age. However, recently Matheson, Darlington & Smith (1998) used horizontal unidirectional drum rotations at 50°/s to measure latency to circularvection with increasing age; by contrast with the results of Paige (1994), a significant increase in latency to experience the illusion occurred with increasing age, suggesting reduced sensitivity to optokinetic stimulation.

Posture

Current research agrees that loss of postural control, exhibited as amount of sway, increases with aging (Norre, Forrez & Beckers, 1987; Baloh, Fifie, Zwerling, Socotch, Jacobson, Bell & Beykirch, 1994; Baloh, Spain, Socotch, Jacobson, & Bell,1995; Matheson, Darlington & Smith, in press(a); Teasdale, Stelmach & Breunig, 1991; Whipple, Wolfson, Derby, Singh & Tobin, 1993). Changes in the control of posture in the elderly can be attributed to changes in two areas: the integration of sensory information, including vestibular information, under altered conditions, and the coordination of muscle responses (Woollacott, Shumway-Cook & Nashner, 1986). Older people demonstrate impaired balance under conditions which cause a conflict of sensory input, in particular conflicting visual and vestibular or somatosensory conditions. Elderly subjects demonstrated the greatest sway when reliant on their vestibular system alone (Matheson, Darlington & Smith, in press(a); Teasdale, Stelmach & Breunig, 1991; Woollacott, 1993). When reliant on their vestibular system in isolation, they demonstrated an inability to compensate for the removal of other sensory information, to ensure postural stability. However, some elderly people may be able to adapt to difficult sensory conditions, as balance improves on subsequent exposure to altered sensory conditions (Woollacott, Shumway-Cook & Nashner, 1986). It is likely that elderly people may also become more reliant on their visual system to control balance.

Conclusions

The peripheral and central vestibular systems exhibit an age-related structural deterioration which may be directly responsible for vestibular reflex deficits, and consequently dizziness, in the elderly. However, it seems likely that the central nervous system is capable of compensating for a certain degree of decline in function, since not all elderly people are impaired to the extent that the clinical signs of vestibular dysfunction are apparent (Paige, 1992). Dizziness and other vestibular disorders may develop only when the degree of deterioration of the vestibular system exceeds the ability of the nervous system to compensate. For those who experience dizziness as a result of age-related deterioration of the vestibular system, the psychological consequences can be devastating, leading to loss of independence, isolation and anxiety disorders related to the fear of falling. However, vestibular rehabilitation programs can be used to reduce the impact of vestibular dysfunction and in so doing can be used to minimise its psychological effects.

References

Alvarez, J.C., Diaz, C., Suarez, C., Fernandez, J.A., Delrey, C.G., Navarro, A. & Tolivia, J. (1998) Neuronal loss in human medial vestibular nucleus. *Anatomical Record, 251,* 431-438.

Baloh, R. W. (1995) Approach to the evaluation of the dizzy patient. *Otolaryngol.-Head and Neck Surg., 112,* 3-7.

Baloh, R.W. (1996a) History I. Patient with dizziness. In R.W. Baloh and G.M. Halmagyi (Eds.), *Disorders Of The Vestibular System,* Oxford University Press, New York-Oxford, pp. 157-270.

Baloh, R.W. (1996b) Vestibular disorders due to cerebrovascular disease. In R.W. Baloh and G.M. Halmagyi (Eds.), *Disorders Of The Vestibular System,* Oxford University Press, New York-Oxford, pp. 418-429.

Baloh, R.W. & Honrubia, V. (1990) *Clinical Neurophysiology Of The Vestibular System.,* F.A Davis Company, Philadelphia.

Baloh, R.W., Jacobson, K. M. & Socotch, T. M. (1993) The effect of aging on visual-vestibulo-ocular responses. *Exp. Brain Res., 95,* 509-516.

Baloh, R. W., Fifie, T. D., Zwerling, L., Socotch, T., Jacobson, K., Bell, T. & Beykirch, K. (1994) Comparison of static and dynamic posturography in young and older normal people. *J. Am. Geriatric Soc., 42,* 405-412.

Baloh, R. W., Spain, S., Socotch, T. M., Jacobson, K. M. & Bell, T. (1995) Posturography and balance problems in older people. *J. Am. Geriatric Soc., 43,* 638-644.

Bergstrom, B. (1973a) Morphology of the vestibular nerve. 2. The number of myelinated vestibular nerve fibers in man at various ages. *Acta Otolaryngol. (Stockh.), 76,* 173-179.

Bergstrom, B. (1973b) Morphology of the vestibular nerve. 3 Analysis of the calibers of the myelinated vestibular nerve fibers in man at various ages. *Acta Otolaryngol. (Stockh.), 76,*173-179.

Berthoz, A. (1996) How does the cerebral cortex process and utilize vestibular signals? In R.W. Baloh and G.M. Halmagyi (Eds.), *Disorders Of The Vestibular System,* Oxford University Press, New York-Oxford, pp. 113-125.

Boult, C., Murphy, J., Sloane, P., Mor, V. & Drone, C. (1991) The relation of dizziness to functional decline. *J. Am. Geriatric Soc., 39,* 858-861.

Burker, E. J., Wong, H., Sloane, P. D., Mattingly, D., Preisser J. & Mitchell, C. M. (1995) Predictors of fear of falling in dizzy and nondizzy elderly. *Psychol. Aging, 10,* 104-110.

Colledge, N. R., Wilson, J. A., Macintyre, C. A. & MacLennan, W. J. (1994) The prevalence and characteristics of dizziness in an elderly community. *Age and Aging, 23,* 117-120.

Cransac, H., Peyrin, L., Cottet-Emard, J. M., Farhat, F., Pequignot, J. M. & Reber, A. (1996) Aging effects on monoamines in the rat medial vestibular and cochlear nuclei. *Hear. Res., 100,* 150-156.

Darlington, C.L. & Smith, P.F. (1996a) Oscillopsia and dizziness due to gentamicin antibiotic treatment: A clinical note on the benefits of vestibular rehabilitation therapy. *NZ J. Psychol. 25,* 24-28.

Darlington, C.L. & Smith, P.F. (1996b) Circularvection: visually-induced illusion of self-motion. *NZ J. Psychol. 25,* 20-23.

Davis, L. E. (1994) Dizziness in elderly men. *J. Am. Geriatric Soc., 42,* 1184-1188.

Drachman, D. A. & Hart, C. W. (1972) An approach to the dizzy patient. *Neurol., 22,* 323-324.

Engstrom, H., Bergstrom, B. & Rosenhall, U. (1974) Vestibular sensory epithelia. *Acta Otolaryngol. (Stockh.), 76,* 331-338.

Fujii, M., Goto, N., Okada, A., Kida, A., & Kikuchi, K. (1996) Distribution of amyloid bodies in the aged human vestibulocochlear nerve. *Acta Otolaryngol. (Stockh), 116,* 566-571.

Guldin, W.O. & Grusser, O-J. (1998) Is there a vestibular cortex? *Trends Neurosci., 21,* 254-258.

Herdman, S.J. (1996) Vestibular rehabilitation. In R.W. Baloh and G.M. Halmagyi (Eds.), *Disorders Of The Vestibular System,* Oxford University Press, New York-Oxford, pp. 157-270.

Johnsson, L.G. & Hawkins, J.E. (1972) Sensory and neural degeneration with aging, as seen in microdissections of the human inner ear. *Ann. Otolaryngol. Rhinol. Laryngol., 81,* 179-193.

Jorgensen, M. B. (1961) Changes of aging in the inner ear. *Arch. Otolaryngol., 74,* 56-62.

Katsarkas, A. (1994) Dizziness in aging: A retrospective study of 1194 cases. *Otolaryngol. Head-Neck Surg., 110,* 296-301.

Leigh, R.J. & Zee, D.S. (1991) *The Neurology Of Eye Movements,* 2nd Edition, F.A. David Company, Philadelphia.

Lopez, I., Honrubia, V. & Baloh, R.W. (1997) Aging and the human vestibular nuclei. *J. Vest. Res., 7,* 77-85.

Matheson, A.J., Darlington, C.L. & Smith, P.F. (1998) Age-related changes in human optokinetic function. *NeuroReport, 9,* 2175-2177.

Matheson, A.J., Darlington, C.L. & Smith, P.F. (in press(a)) Further evidence for age-related deficits in human postural function. *J. Vest. Res.*

Matheson, A.J., Darlington, C.L. & Smith, P.F. (in press(b)) Incidence of dizziness in the elderly: Correlation with ocular motor, optokinetic and postural function. *NZ J Psychol.*.

Norre, M.E., Forrez, G. & Beckers, A. (1987) Vestibular dysfunction caused by instability in aged patients. *Acta Otolaryngol. (Stockh), 104,* 50-55.

Paige, G.D. (1992) Senescence of human visual-vestibular interactions: I. Vestibular - ocular reflex and adaptive plasticity with aging. *J. Vest. Res., 2,* 133-151.

Paige, G.D. (1994) Senescence of human visual-vestibular interactions: II. Smooth pursuit, optokinetic, and vestibular control of eye movements with aging. *Exp. Brain Res., 98,* 355-372.

Peterka, R. J., Black, F. O. & Schoenhoff, M. B. (1990) Age related changes in human vestibulo-ocular and optokinetic reflexes: Pseudorandom rotation tests. *J. Vest. Res., 1,* 61-71.

Reske-Nielsen, E. & Hansen, C. C. (1961) Pathological studies in perceptive deafness. Four patients with presbyacusis. *Acta Otolaryngol. (Stockh.), 188,* 327-333.

Richter, E. (1980) Quantitative study of human Scarpa's ganglion and vestibular sensory epithelia. *Acta Otolaryngol., 9,* 199-208.

Rosenhall, U. (1973) Degenerative patterns in the aging human vestibular neuroepithelia. *Acta Otolaryngol., 76,* 208-220.

Rosenhall, U. (1974) Epithelial cysts in human vestibular apparatus. *J. Laryngol. Otol., 12,* 105-112.

Rosenhall, U., & Rubin, W. (1975) Degenerative changes in the human vestibular sensory epithelia. *Acta Otolaryngol., 79,* 67-80.

Sloane, P., Blazer, D. & George, L.K. (1989) Dizziness in a community elderly population. *J. Am. Geriatric Soc., 37,* 101-108.

Sloane, P., Hartman, M. & Mitchell, M. (1994) Psychological factors associated with chronic dizziness in patients aged 60 and older. *J. Am. Geriatric Soc., 42,* 847-852.

Smith, P.F. (1997) Vestibular-hippocampal interactions. *Hippocampus, 7,* 465-471.

Takeuchi, Y.K., Takeuchi, I.K., Muashima, Y. & Setoohshima, A. (1997) Age-related appearance of dystrophic axon terminals in cerebellar, and vestibular nuclei of Mongolian gerbils. *Exp. Animals, 46,* 59-65.

Teasdale, N., Stelmach, G E. & Breunig, A. (1991) Postural sway characteristics of the elderly under normal and altered visual and support surface conditions, *J. Gerontol. Biol. Sci., 46,* 238-244.

Whipple, R., Wolfson, L., Derby, C., Singh, D. & Tobin, D. (1993) Altered sensory function and balance in older persons, *J. Gerontol., 48,* 71-76.

Wilson, V. J. & Melvill Jones, G. (1979) *Mammalian Vestibular Physiology,* Plenum Press, New York.

Woollacott, M.H., Shumway-Cook, A. & Nashner, L. M. (1986) Aging and posture control changes in sensory organisation and muscular coordination. *Int. J. Aging Hum. Dev., 23,* 97-114.

Woollacott, M.H. (1993) Age-related changes in posture and movement. *J. Gerontol., 48,* 56-60.

Yardley, L. & Luxon, L. (1994) Treating dizziness with vestibular rehabilitation. *British Medical Journal, 308,* 1252-1253.

Address for correspondence:
Dr. C. L. Darlington
Department of Psychology and the Neuroscience Research Centre
University of Otago
PO Box 56
DUNEDIN

Email: cynthia@otago.ac.nz