CHAPTER 1: NATURE AND CAUSES OF VERTIGO

> I was standing in my bedroom, brushing my hair in front of the mirror, and suddenly it was as if I had two heads -- me looking at the mirror and knowing that I was, but the inner head spinning round and everything going with it.
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> When you're not near anything you feel as though you don't know whether you're standing up or laying down or what, because there's such a vast area around you ... it was all as though I was standing in the sea on my own with miles and miles all round me, and there was nothing to hang on to.
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> I wake up and everything's black, the room's going round, the bed's like a boat tossing about, and I'm feeling sick. I usually have to go to the toilet, be sick, I go all weak and limp, cold. It's the weakness afterwards too, you feel as though you've been seriously ill, I'm exhausted, limp, I can't pick up a cup sometimes for a few hours ... I get them [attacks] about every 5 to 6 weeks, I never know just when ... When I have one of my severe turns I don't do anything for 4 or 5 days, I sit like a zombie.

Vertigo is a sensation that is very difficult to describe, according to those unfortunate individuals who suffer from attacks. Nevertheless, the preceding accounts vividly convey the experience of people with symptoms which doctors label "vertigo", but which sufferers more often refer to using expressions such as "dizziness", "whirling", "a swimming sensation", "a feeling of unsteadiness or falling". The medical definition of the term "vertigo" (which doctors pronounce as "ver-tie-go") is quite different from the lay usage of this word (ordinarily pronounced "ver-tee-go"). In everyday use, the word vertigo most commonly describes a fear of heights, although it is sometimes also used to refer to generalised feelings of giddiness, faintness, confusion, anxiety or insecurity, regardless of the precise nature and cause of these sensations. However, the term vertigo is strictly defined medically as an illusion of movement of the self or of the environment. This illusion of movement can be caused by any disorder or injury which disrupts the functioning of the multisensory balance system, which controls the perception of orientation and self-motion relative to the external environment. In the medical context, the term vertigo is therefore simply a technical label for the symptom of perceptual disorientation, which can be due to a wide variety of causes.

Although vertigo is a common health problem which can cause quite severe disability and distress, there has been surprisingly little research into the impact it has on people's lives. This may be partly due to the perplexity and confusion that surrounds the topic. A firm diagnosis of the organic cause for any particular case of vertigo is frequently difficult to achieve, since there are so many possible aetiologies. Indeed, often it is not possible to even confirm that some physical dysfunction exists, as the functioning of the balance system is extremely complex and difficult to test. The sensations of perceptual disorientation can be vague and difficult to describe, and so symptoms of balance system dysfunction may be mistaken for signs of other physical disorders which can cause dizziness, ranging from hypotension to epilepsy.

Alternatively, since dizziness is one of the most common sensations experienced during a panic attack and is included as a symptom in the clinical descriptions of several psychiatric disorders, complaints of dizziness are often interpreted as a sign of underlying anxiety or psychological disturbance. Consequently, there is no neatly defined and circumscribed population of vertigo sufferers available for study, and any attempt to analyse the problem of vertigo is beset by the difficulties pertaining to uncertain diagnoses and diverse aetiologies.

Owing to these diagnostic problems, the population of people who complain of vertigo undoubtedly encompasses a variety of individuals, ranging from those with severe vestibular disorders to those with no clinically significant physical abnormalities. It is possible that the experience of vertigo may be similar, whether the origin of the feelings of disorientation is primarily physical or psychological. The fears, uncertainties, social embarrassment, occupational problems, and the form and extent of the malaise and handicap resulting from spells of dizziness caused by anxiety may be comparable in many respects to the difficulties encountered by people suffering from vestibular disorders. Nonetheless, the scope of this book is deliberately limited to consideration of the experience of individuals who are believed to have some organic balance system dysfunction. In the studies upon which much of this book is based, this criterion was met by including only those individuals who had received a firm diagnosis of peripheral vestibular dysfunction from an experienced hospital clinician. In addition, the homogeneity of the samples obtained was assured by comparing the characteristics of the people who exhibited definite signs of balance system dysfunction upon examination and testing (usually slightly more than half of the sample) with those who did not. Since absolutely no differences in diagnosis, symptoms, handicap or psychosocial profile were found between people who did or did not show objective signs of organic dysfunction, it seems reasonable to conclude that the failure to obtain evidence of balance system dysfunction in some cases was simply due to the limitations of the available tests (discussed in the following chapter).

There are two principal reasons why I have chosen to focus selectively upon the experience of disorientation caused by physical disorder, despite the possibility that there may be important parallels with the experience of people whose dizziness is due primarily to psychological factors. The first is that feelings of disorientation are all too readily categorised as "all in the mind" by professionals working within both medicine and psychology. The psychological explanations for symptoms of vague dizziness are so well established that the sometimes subtle and complex perceptual causes for disorientation may be overlooked. Moreover, there is a prevalent (albeit unproven) hypothesis, familiar to all clinicians who treat vertiginous patients, that some forms of vertigo due to organic dysfunction may have a partly psychosomatic aetiology and that vertigo sufferers are therefore likely to have neurotic personalities, or a predisposition for anxiety and hypochondria. Consequently, the reported psychosocial problems of even those people who obtain a diagnosis of physical disorder may be regarded as signs of emotional distress or personality disorder. One of the purposes of this book is to present an alternative to these interpretations of the link between anxiety and vertigo, by describing precisely how and why people who were previously psychologically healthy and emotionally stable may become seriously distressed and handicapped because of recurrent attacks of vertigo due to disordered balance system functioning.

The second reason for basing this book entirely on studies of people with a diagnosis of balance system dysfunction is to illustrate the way in which the activities, attitudes, intentions and environment of the individual fundamentally affect the experience of vertigo, even when it is clearly due to some organic disorder. The importance of cognitive and behavioural responses to events and environments is readily accepted in the context of "non-organic" complaints of disorientation, such as the dizziness and confusion which are often experienced during a panic attack. However, the essential role of such processes is seldom fully appreciated in the case of individuals diagnosed as suffering from vertigo caused by organic disorder; in the attempt to identify and remediate the pathophysiological features of these patients' problems, other equally important and intrinsic features of their experience of illness are often ignored.

The following chapters detail the way in which physiological processes mutually affect and are affected by attitudes, activities and environments, and demonstrate how the experience and course of vertigo emerges from combined and reciprocal influences, both physical and psychosocial, and both internal and external to the individual concerned. However, in order to appreciate how environmental, cognitive, emotional and behavioural factors can affect the course of vertigo, a basic understanding of the nature of vertigo and of the balance system is required. The remainder of this chapter therefore provides an elementary description of the balance system, and of the characteristics and causes of vertigo. (For more detailed descriptions of the pathophysiology and differential diagnosis of vertigo, the reader is referred to Brandt, 1991; Dix and Hood, 1984; Wright, 1988).

The balance system

The perception and control of orientation and self-motion is achieved by integrating information from three primary sources: the visual, somatosensory (or proprioceptive) and vestibular sensory systems. The perceived motion of the visual scene provides constant feedback about self-motion; for example, turning the head to the left produces a simultaneous movement of the entire visual scene to the right. The somatosensory system comprises all the sensors in the body (skin, muscles, joints etc.), and provides information about internally controlled movements, as well as direct contact with the environment. For instance, when an individual turns to the left the somatosensory system monitors the joint and muscle activity involved in making the turn, while information about contact with the ground is derived from the soles of the feet. Finally, the vestibular system directly senses the momentary acceleration and orientation of the head. Although the vestibular system is not the most important source of information relating to self-motion -- in most situations, the visual and somatosensory systems actually provide more precise and reliable information -- disorders of the vestibular system are the most common organic cause of illusions of movement. The functioning of the vestibular system will therefore be described in more detail below.

The peripheral vestibular sensory organ forms part of the inner ear, and consists of a tiny bony structure (the "labyrinth") filled with two types of fluid, known as perilymph and endolymph (see Figure 1). The vestibular organ comprises the semi-circular canals, which detect angular acceleration, and the otoliths, which monitor linear acceleration and the orientation of the head relative to gravity. The otoliths contain "cilia", which are similar to stiff hairs

which stick out of the sensory cells of the otolith. When the cilia are in their resting position, the sensory cells give out a constant signal, or "resting discharge" of neural activity. However, when the cilia are bent this signal changes, as the neural activity either increases or decreases, depending upon the direction in which the cilia are deflected. At the opposite end from the sensory cells, the cilia are attached to a membrane in which are embedded minute but relatively heavy crystals ("otoconia"). When the orientation of the head changes (see Figure 2), the sensory cells move with the head, to which they are firmly attached, but the heavy, free-floating membrane containing the otoconia lags behind. Consequently, the cilia are bent, and a change in the sensory signal is produced. These alterations in the sensory signal can be produced either by a change in the position of the head relative to the force of gravity, or by the force of linear acceleration or deceleration, such as that produced by stopping in a car (horizontal force) or in a lift (vertical force). The otoliths consist of two structures -- the utricle and saccule -- which are set approximately at right angles, so that both vertical and horizontal forces can be detected.

The semi-circular canals are three ring-like bony tubes protruding from the utricle. A small swelling ("ampulla") at one end of each semi-circular canal contains sensory cells and cilia similar to those in the otoliths, but in this case the cilia extend towards the "cupula", a piece of tissue which virtually fills the ampulla. When the head accelerates in the same (angular) direction as the plane of the semi-circular canal (see Figure 2), all the structures of the semi-circular canal naturally move with the head. However, due to inertia the fluid (endolymph) which fills the canal lags behind the head movement, and therefore flows against the cilia, bending them and thereby producing a change in the signal emitted by the sensory cells. The three semi-circular canals are positioned at right angles, so that between them they can detect acceleration in each of the three possible planes of motion, both lateral (spinning) and vertical (somersaults and cartwheels).

Signals relating to linear and angular accelerations detected by the two otoliths and three semi-circular canals pass via the vestibular nerve to the vestibular nuclei in the brainstem. The central inter-connections within the balance system are extremely complex; information from the vestibular organs in both ears is combined with information derived (mainly) from vision and the somatosensory system at various levels within the brainstem, cerebellum and cortex. This integrated information provides the basis not only for the conscious perception of orientation and self-motion, but also the pre-conscious control of eye-movements and posture, by means of what are known as the vestibulo-ocular and vestibulo-spinal reflexes. The purpose of the vestibulo-ocular reflex is to maintain a stable point of visual fixation during head movement by automatically compensating for the head movement with an equivalent eye movement in the opposite direction. The vestibulo-spinal reflexes contribute to postural stability and balance.

Balance system dysfunction and vertigo

In normal circumstances, the perceptual information about orientation derived from the vestibular, visual and somatosensory systems is congruent; at the same time that the visual field sweeps past our eyes, the vestibular system signals angular acceleration, and activity in our neck muscles and joints confirms that we are turning our head. Indeed, when the balance system is functioning

properly there is no conscious awareness of "sensations" corresponding to the perception of orientation -- we just automatically register our precise orientation and self-motion and utilise this information to maintain balance and well-coordinated activity. However, when an apparent mismatch occurs between the different sensory inputs to the balance system, the perceptual uncertainty this creates is itself experienced as a sensation, which may be described as dizziness, disorientation or vertigo. The perceptual disorientation can be caused either by a failure of sensory processing, or by man-made environmental conditions which our balance system has not been equipped by evolution to cope with, such as passive transport by car, boat, airplane, or even spaceship (the space sickness caused by floating around in conditions of weightlessness causes significant problems for astronauts, particularly when wearing their helmets!). If the disorientation is attributable to internal dysfunction it is labelled "vertigo", whereas disorientation caused by external perceptual conditions is known as "motion sickness" (or more specifically, car-sickness, sea-sickness etc.).

The physiological causes of vertigo include dysfunction of any of the sensory systems contributing to orientation perception, or of the central interconnections of the balance system; these disorders and their effects are described in more detail in the following section. The environmental causes comprise any situation characterised by an unusual combination of visual, vestibular and somatosensory information. For example, when a person in a ship travelling across rough seas sits in a cabin with no portholes, the vestibular system will signal constant motion, but this information is contradicted by the inability of the visual system to detect any corresponding movement of the visual field, since the visual environment (the cabin) moves with the ship and passenger. These relatively uncommon perceptual conditions are experienced as disorientation and sea-sickness, since on dry land vestibular signals are usually accompanied by visual field motion. (Of course, there are many other combinations of perceptual information that can result in motion sickness at sea, on land and in the air; for a more detailed discussion of the factors contributing to motion sickness see Money, 1990; Reason and Brand, 1975; Yardley, 1992).

The symptoms of vertigo are very similar to those of motion sickness, as one might expect in view of their shared causal origin. The defining symptom is, as previously stated, an illusion of movement. However, the subjective experience of people who receive a diagnosis of "vertigo" can be extremely varied, as the following descriptions illustrate:

> The floors start to come up and things revolve and you feel nausea ... you just entirely lose your balance and sort of reel about.
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> Every time I looked down all I could see was a black hole in front of me -- everything seems to come pushing me back.
>
> I was beginning to sweat, would feel cold, shivery, I would physically begin to look pale, drained.
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> I go on the tilt, it washes over my head, surges through -- a bit like if you went up one of these loop things, Alton Towers [a

roller-coaster ride] or something like that.

The classic symptom of acute vestibular imbalance is a strong sensation of spinning, or of the environment whirling around. However, there may be simply a momentary feeling of being pushed to one side, an impression that the world appears to be rocking or moving about, or just a vague consciousness of giddiness or unsteadiness. The perceptual disorientation also results in two constellations of secondary symptoms. The first group of symptoms are directly attributable to the disruption of ocular and postural control caused by balance system dysfunction. Disordered vestibulo-ocular reflexes can result in a blurred or flickering visual image and difficulty in focusing, while abnormal vestibulo-spinal reflexes may cause staggering, loss of balance and falling, or a tendency veer to one side when walking. In addition, disorientation triggers a range of autonomic changes. The principal symptoms are nausea, vomiting, pallor and cold sweating, but other common physiological concomitants of vertigo and motion sickness include salivation, flatulence or diarrhoea, sighing or yawning, a feeling of warmth, an increase in heart rate and respiration rate, headache, drowsiness and fatigue, apathy, anxiety and depression. Many of these secondary physiological changes are undoubtedly triggered directly via the central pathways shown in Figure 2, although anxiety may contribute to or exacerbate some of this autonomic symptomatology (see Chapter 4).

While perceptual disorientation and failure of vestibulo-ocular and postural coordination are simply the inevitable consequence of a disruption of balance system functioning, the adaptive significance or "survival value" of the ancillary autonomic symptoms remains the subject of speculation. Triesman (1977) has proposed an "evolutionary hypothesis" to account for motion sickness which may also be considered relevant to the autonomic symptomatology provoked by vertigo. He suggested that motion sickness is simply an accidental manifestation of what originally evolved as an adaptive response to ingesting poisons which disrupt coordination and perception through their effects on the central nervous system. The vomiting would expel the poison, and the nausea would cause aversion to the poisonous substance; indeed, sensitivity and aversion to sights and smells associated with periods of disorientation has been observed in both motion sickness (Lawther & Griffin, 1988) and vertigo (Grisby & Johnston, 1989). The general lethargy and malaise associated with both motion sickness and vertigo would tend to limit the amount of activity undertaken while the animal was dangerously uncoordinated.

However severe the symptoms initially provoked by peripheral sensory dysfunction, neurophysiological and sensorimotor adaptation to this dysfunction can gradually be achieved by means of a process of "habituation" or "compensation", provided that the central structures and inter-connections within the balance system are functioning normally. At first, the change in the sensory signal resulting from, say, the sudden complete destruction of the vestibular organ in the left ear will result in a strong sensation of spinning to the left, because the resting discharge from the right vestibular organ is no longer counterbalanced by signals from the left vestibular organ. Similarly, there will be a tendency to veer or fall towards the left side, and the eyes will tend to drift over to the left. As an "emergency" measure, central processes automatically attempt to partially suppress the remaining vestibular input. In the longer term, compensation is achieved by a process of perceptual re-learning; the balance

system adjusts to the fact that there is now no vestibular input from the left side, and utilises information from the healthy vestibular organ and from vision and the somatosensory system to substitute for the missing vestibular signal. This process of re-learning can only occur through repeated experience of the new pattern of sensory information that is now provoked by each head movement and orientation.

The subjective experience of this process of compensation is that the person who suffers unilateral vestibular failure will immediately learn to lie down and keep absolutely still, in order to minimise the changes in vestibular activity which provoke dizziness and nausea. After several hours, the disorientation when lying still will lessen, but the slightest head movement will provoke fresh symptoms. Over time the person will be forced to make some repeated head movements (e.g. lifting or turning the head), and eventually the disorientation provoked by these movements will become less severe, as the balance system learns the new pattern of information associated with these movements. However, new movements, such as bending down or nodding quickly, will continue to provoke dizziness until they have been repeated enough times for the process of adjustment to occur. In this sense, the process of adaptation to sensory dysfunction is very similar to the acquisition of "sea-legs". During a short sea passage there is insufficient time for the balance system to adjust to the new pattern of perceptual information experienced on a ship, and the individual will tend to stagger as the ship heaves, and may feel very ill. However, over the course of a longer voyage the continued exposure to these perceptual conditions eventually results in complete adaptation, and the same individual is able to move around freely and without sickness.

Incidence and common organic causes of vertigo

The precise incidence of vertigo due to balance system dysfunction is very difficult to ascertain because of the problems associated with defining and diagnosing the condition. The exact prevalence of cases of vertigo within the community, and the proportion of these that are actually seen in general practice or referred to hospital, is therefore unknown. It is also difficult to establish the relative prevalence of the various causes of vertigo; although some clinicians have detailed the incidence of various disorders within their specific clinic populations (e.g. Drachman & Hart, 1972), these figures are likely to be heavily influenced by local customs relating to referral and diagnosis. Nevertheless, it is known that each year five out of every thousand people in the U.K. population seek consultations with their doctor because of symptoms which are classified by their general practitioner as true vertigo, and a further ten people are seen by the G.P. on account of a complaint of dizziness or giddiness (RCGP/OCPS). Although vertigo can affect people of any age, the incidence rises with advancing age owing to the greater prevalence in older people of disorders which can give rise to vertigo (such as cardiovascular and cerebrovascular disease). In a community survey of people aged fifty to sixty-five, a quarter of the sample stated that they currently suffered from giddiness or dizziness (Stephens, 1990), while by the age of eighty years two-thirds of women and one-third of men report having experienced episodes of vertigo (Luxon, 1984). Baloh (1992) notes that dizziness is the most common presenting complaint in primary care among people aged over seventy-four.

The symptoms and prognosis in cases of vertigo vary according to the

aetiology. A brief overview of the major causes of vertigo, together with typical symptoms and prognoses, is therefore given below. However, the distinctions between these disorders are not always entirely clearcut and there can be a considerable overlap in symptomatology. For example, episodic vertigo may be associated with a feeling of fullness in the ears but no hearing loss or tinnitus, Meniere's disease is sometimes preceded by accident or infection and accompanied by signs of benign paroxysmal positional vertigo, while a vague dizziness may persist in the intervals between attacks of definite positional vertigo.

Peripheral vestibular causes of vertigo. One of the most common types of vertigo is known variously as "vestibular neuronitis", "labyrinthitis", or sometimes "epidemic" or "idiopathic" vertigo, and is characterised by the classic symptoms of vestibular dysfunction described in the previous section, often preceded by a viral infection. The exact definition of the disorder and its precise cause, or causes, are not fully established. Another very common vestibular disorder is known as "benign paroxysmal positional vertigo". This is thought to occur when heavy debris (otoconia), dislodged from the otoliths as a result of age-related degeneration or head injury, comes to rest in one of the semi-circular canals. The affected canal therefore starts to register changes in orientation relative to gravity, but continues to signal angular acceleration. The subjective experience is that changes in orientation, such as lying down or rolling over in bed, can provoke a brief but extremely powerful sensation of spinning (and accompanying nausea). Both vestibular neuronitis and benign positional paroxysmal vertigo usually clear up spontaneously over a period of weeks or months, but an unfortunate minority of individuals find that they have repeated attacks or persistent symptoms for many years.

The major cause of recurrent attacks of severe vertigo is a syndrome known as "Meniere's disease" characterised by a unique combination of symptoms: severe bouts of vertigo lasting several hours; fluctuating tinnitus (a noise in the ear or head) -- generally a low-pitched buzzing or roaring in one ear, which at first accompanies attacks of vertigo but may later become continuous; an intermittent feeling of pressure in the ear; fluctuating, progressive unilateral hearing impairment, which interferes predominantly with hearing for low-pitched sounds initially, but may eventually result in complete loss of hearing in the affected ear. These symptoms are generally thought to arise as a result of an imbalance of fluid pressures in the inner ear known as "endolymphatic hydrops", but although evidence of a link between Meniere's disease and hydrops has been found at *post mortem* (Rauch et al., 1989) the association is not clearcut since many people with signs of hydrops are asymptomatic. The prognosis for people with Meniere's disease is uncertain, although Browning (1991) calculates that in the long-term (over more than a decade) 98% of those seen in hospital clinics recover from their vertigo. Some people have only a few attacks, followed by complete remission. Often acute attacks occur several times a year, and unless complete compensation for the change in the vestibular signal caused by these acute attacks is achieved, the individual may experience considerable residual dizziness and movement-provoked vertigo in the intervening months. The disorder eventually seems to "burn itself out", usually leaving the sufferer with a permanent unilateral hearing loss and tinnitus but no vertigo, but sometimes the disease spreads later to the previously unaffected ear. Meniere's disease is most common among people aged between 30 and 50 years, with a slight

predominance of females, and prevalence estimates range from 0.1 to 1% of the population.

Miscellaneous additional causes of vertigo of peripheral vestibular origin include a variety of disorders and diseases of the middle ear, ototoxic drugs (used only in medical emergencies), obstruction of the peripheral blood vessels supplying the inner ear, syphilis, herpes zoster, or fracture of the temporal bone. Occasionally, a small hole ("fistula") in the membrane of the vestibular organ can be caused by middle ear disease, surgery, head injury, or the abrupt pressure changes which may be induced by diving, flying, or strenuous physical activity. The hole will allow the fluid inside the organ to leak out ("perilymph leak"), resulting in symptoms of sudden vertigo and unilateral hearing loss, often exacerbated by additional pressure changes (e.g. blowing the nose, straining when constipated) which force the fluid out of the inner ear. Very rarely, vertigo may be caused in the early or later stages of the growth of a benign tumour ("acoustic neuroma") which can develop on the audiovestibular nerve, affecting not only the nerve function but also the blood-supply to the inner ear, and eventually pressing upon the brain-stem.

Non-vestibular peripheral causes of vertigo. Disorders of the neck, which may alter the somatosensory information relating to head movement or interfere with the blood-supply to the vestibular system, are believed by many clinicians to be a cause of vertigo; common aetiologies include whip-lash injury and cervical spondylosis. Sometimes unsteadiness is related to a loss of feeling in the feet and legs due to "neuropathy", which may caused by diabetes, alcohol abuse, vitamin deficiency, damage to the spinal cord, or a number of other disorders. Occasionally the origin of feelings of giddiness, unsteadiness or illusory movement of the environment can be traced to some distortion of the visual input. An abnormal visual input may be caused by weakness of the eye muscles, or may be experienced when adjusting to powerful lenses (such as those worn after an operation to remove cataracts) or to bifocal glasses.

Central causes of vertigo. Although most cases of vertigo are attributable to peripheral (mainly vestibular) pathology, symptoms of disorientation can be caused by a wide range of central disorders or injuries, at the level of the brainstem, cerebellum or cortex. Vertigo of central origin is almost always accompanied by some other symptom of central neurological disorder, such as sensations of pain, tingling or numbness in the face or limbs, difficulty speaking or swallowing, headache, visual disturbances, and loss of motor control or loss of consciousness. The more common central causes of vertigo include disorders of the blood supply to the brain (ranging from migraine to strokes), epilepsy, multiple sclerosis, alcoholism, and sometimes tumours. Dizziness and imbalance due to central causes have been reported as a potential side-effect of a vast array of drugs, including widely-used analgesics, contraceptives, and drugs used in the control of cardiovascular disease, diabetes and Parkinson's disease, and in particular the centrally-acting drugs such as stimulants, sedatives, anti-convulsants, anti-depressants and tranquillisers (Ballantyne & Ajodhia, 1984).

Age-related causes of vertigo. The incidence of vertigo, as noted previously, is age-related; there is a progressive growth in the number of reported and diagnosed cases with increasing age. As in the case of age-related hearing loss, it is possible that vertigo in the elderly may be partly attributable to non-specific degeneration within the peripheral and/or central levels of the vestibular system.

In addition, many of the disorders which can give rise to vertigo, such as cerebrovascular disease or cervical damage, are more common in the elderly. Often people in the older age-groups are obliged to take a number of medications which can cause dizziness. Finally, disorientation and unsteadiness may result from what is known as "multisensory dysfunction" -- a combination of minor defects in the various sensory systems contributing to orientation. For example, failing eyesight alone may not be sufficient to cause imbalance, but the addition of slightly reduced sensation in the lower limbs and intermittent positional vertigo may result in a dangerous and severely handicapping degree of postural instability. Since compensation for vestibular dysfunction requires intact central neurological functioning, alternative sources of sensory input (to substitute for absent or distorted vestibular signals), and plenty of active sensorimotor experience, the process of compensation may sometimes be retarded in the elderly as a result of minor central dysfunction, multisensory impairment, or inadequate physical activity.