Introduction

Human balance function depends on sensory inputs from the vestibular, proprioceptive, and visual systems as well as proper integration of those inputs in the central nervous system (CNS). Control of movements requires the motor centres to accurately process the sensory information and transmit the necessary commands to the appropriate muscles. Both structural and functional deteriorations in all of these systems have been reported with increasing age. The brainstem, cerebellum, and higher cortical structures within the CNS all undergo age-related degenerative changes 1. These include decrease in the number of neurons, loss of myelination, decrease in the number of Purkinje cells, and other neuronal changes. Vertigo, dizziness and imbalance are the main symptoms of vestibular disorders. These symptoms can lead to physical consequences such as reduced postural control and falls and to psychological and psychiatric consequences. Vertigo and dizziness are not synonymous with each other, although they are often used interchangeably. The two terms cover a number of multisensory and sensomotor syndromes of various aetiologies and pathogenesis disorders of perception (dizziness /vertigo), gaze stabilisation, postural control and the vegetative system are related to the main functions of the vestibular system. Vertigo refers to true spinning or movement of the environment and indicates an imbalance of tonic vestibular signals, either centrally or peripherally. Dizziness is one of the most frequent complaints in a medical office, for both primary and specialised care. The term dizziness encompasses a large spectrum of symptomatology dizziness is a nonspecific term that describes an unpleasant sensation of imbalance or altered orientation in space. Dizziness usually refers to lightheadedness, presyncope, vertigo, motion sensitivity, imbalance, anxiety, or just not feeling well. The prevalence...
of dizziness increases steadily with age, epidemiological studies suggest a prevalence of dizziness as height 30% in the general population, it rises to 39% in those over 80 years of age. For example considerable loss of proprioceptors and vestibular receptors is common in elderly people and may contribute to dizziness. For those older than 85, the number of adults with dizziness increases to about 50%. Furthermore, the prevalence of these symptoms is greater for women. For clinicians, dizziness often represents a diagnostic problem: it is a subjective sensation that depends on self-report, it may refer to several different and overlapping sensations, and it can be caused by a wide range of benign or serious conditions. Most dizziness and vertigo syndromes can be diagnosed with a careful history and bedside tests that do not require any special equipment. Older individuals who suffer from dizziness appear to be at significantly higher risk of accidental falls and consequent injuries. Falls are the leading cause of accidental death in persons older than 65 years, and are the number one reason for hospital admission for non fatal falls in this population.

**Define the symptom**

Obtaining an accurate description of the symptom in the patient’s words is the first step in the clinical evaluation. Violent rotatory vertigo as experienced when riding a merry-go-round (eg. vestibular neuritis), postural imbalance (eg. phobic postural vertigo), numbness (eg. medication, drug intoxication) or oscillopsia as the perception of objects bouncing while walking (bilateral vestibulopathy).

**The duration of the symptom**

Attacks of vertigo that last for seconds to minutes (eg. vestibular paroxysmia), over hours (eg. Menière’s disease), persistent lasting for days to few weeks (eg. vestibular neuritis), postural vertigo lasting for minutes to hours (eg. transient ischaemic attacks of the brainstem or cerebellar structures), episodes of vertigo lasting less than 1 minute (eg. benign paroxysmal positional vertigo).

**Trigger and exacerbation of the symptom**

Walking (eg. bilateral vestibulopathy), episodes of vertigo that triggered by head movements (eg. benign paroxysmal positional vertigo, until proven otherwise), head turning (eg. vestibular paroxysmia), coughing, pressing, loud sound of a certain frequency (eg. perilymph fistula).

**Patient’s medical history**

Comorbid conditions, drug-related problems (due in part to altered pharmacokinetics and pharmacodynamics), polypharmacy, larger number of doses of medications per day, low body weight, and a history of adverse drug reactions predispose older adults to dizziness. Many drugs can cause dizziness, some of which are used to treat dizziness. This includes some over the counter medications such as diphenhydramine and meclizine. The list of current medication is important to collect because details gathered can sometimes help make a diagnosis an even direct the management. Most medicines can cause dizziness as a side effect, and about 50% of community dwelling elderly are on five or more medications. Approximately 80% of
older people have at least one chronic medical condition, and most of those disorders can contribute to or even cause types of dizziness. Visual acuity, vestibulo-ocular reflex gain, and sensory nerve action potential amplitude inevitably decline with age. Probably resulting from these changes, measures of balance show gradual deterioration with age as well 15.

**Physical examination**

All components of the balance system should be examined, including the peripheral vestibular system, central vestibular system, visual system, proprioception, and the interactions between these systems. Physical examination is particularly important in the evaluation of dizziness in the elderly because in some settings such as nursing homes, extensive laboratory tests may not be available. An otoscopic examination is important. Abnormalities possibly encountered include middle ear effusions, tympanic membrane perforations, cholesteatoma, and neoplasms. A typical physical examination consists of neurologic, neurotologic, and general medical examination 10. Neurological examination: any clearcut central neurologic signs in the presence of a neuro-otological syndrome make lesion localisation relatively easy, particularly when there is brainstem involvement. Nystagmus can be defined as periodic, most often involuntary eye movements that normally consist of a slow (causative or pathological) phase and a quick eye phase, which brings the eye back to the initial position. In the clinical examination the physician should at first try to differentiate between peripheral vestibular and central vestibular forms of vertigo. The first step is clinical examination of the eyes in nine different positions to determine ocular alignment, fixation deficits, nystagmus, range of movement, and disorders of gaze-holding ability. In primary eye position, one should first look for periodic eye movements such as nystagmus or saccade intrusion. An important pattern of spontaneous nystagmus to recognize is peripheral vestibular nystagmus (eg. patients with vestibular neuritis in the acute phase, they will exhibit a spontaneous nystagmus with the fast phase beating toward the opposite ear), which is readily apparent in an incautious disorders as a horizontal greater than torsional, unidirectional nystagmus that can suppressed with fixation. Nystagmus in vestibular neuritis is spontaneous (i.e., present in primary gaze) for at least the first several hours of symptoms. Following this initial time period, the nystagmus may only be identified during gaze testing (i.e., having the patient look to each side) or if visual fixation is blocked. The examiner should closely inspect saccade velocity and accuracy. First it is necessary to observe spontaneous saccade triggered by visual or auditory stimuli. Then the patient is asked to glance back and forth between two horizontal or vertical targets (eg. slowing of horizontal saccade is generally observed in brainstem lesions; these is most often a dysfunction of ipsilateral paramedian pontine reticular formation, and slowing vertical saccades indicates a midbrain lesion in which the rostral interstitial medial longitudinal fascicle is involved. In the course of the clinical examination of eye movement with an examination flashlight, one should ensure that patient has full ocular ductions and determine the effect of gaze on the spontaneous nystagmus, or if no spontaneous nystagmus is present then look gaze-evoked nystagmus. Gaze-evoked nystagmus can only be clearly identified when the patient fixates with both eyes (eg. horizontal gaze-evoked nystagmus can indicate a structural lesion in the area of the brainstem or cerebellum, and vertical gaze-evoked nystagmus is observed in midbrain lesions involving the interstitial nucleus Cajal). Restricted upgaze is common in the elderly subjects but complete loss of up gaze o slowed vertical saccades indicate pathology within the midbrain. The head impulse test is a test for the vestibulo-ocular reflex (VOR). To test the horizontal VOR, the examiner holds the patient’s head between both hands, asks him to fixate a target (on examiner nose) in front of his eyes, and rapidly turns the patient’ head horizontally on the left and then to the right. When the VOR is intact, thrusting the head to the side will cause an immediate compensatory conjugate eye movement in the opposite direction such that the patient maintains fixation on the examiner at full time. The head impulse test is particularly helpful in identifying lateral or bilateral impairment of the VOR at bedside 17. The peripheral sensory examination is important because a peripheral neuropathy can cause non specific dizziness or imbalance. Finger pointing test, and rapid alternating movements will assess extremity coordination. Indeed, coordination in patients with dizziness, because disorders char-
acterized by ataxia can present with the clinical symptom of dizziness.

**Positional testing**

Testing can help identify peripheral or central cause of dizziness. The so-called Dix-Hallpike manoeuvre (Fig. 1) is performed especially to determine whether benign paroxysmal positioning vertigo (BPVV). During this maneuver, the patient's head is turned 45° to one side while he is seated. The patient is then moved quickly to a supine position with the neck slightly extended and the head remaining turned. The characteristic ipsidirectional torsional nystagmus is seen when the undermost ear is affected. The characteristics of the nystagmus include onset after several seconds, a decline after 10 to 30 seconds, and diminished effect with repeated positional testing in the same sitting. If a BPVV of the left posterior semicircular canal, for example, is present, this manoeuvre will induce, with a certain latency, a crescendo-decrescendo like nystagmus, which from the patient's viewpoint beats counterclockwise toward the left ear and to the forehead. The most common type of positional vertigo, BPVV, is caused by free-floating calcium carbonate debris usually in the posterior semicircular canal, but occasionally in the lateral canal, or rarely the anterior canal. BPVV is the most common cause of vertigo, not only in the elderly. Positional testing can be difficult in the elderly patients because of pain or reduced range of motion of the neck. In cases of severe neck or back problems, specialized equipment may be needed to safely perform the procedure. Positional testing can also trigger central type of nystagmus, usually persistent downbeating (eg. Chiari malformation or cerebellar ataxia).

**Gait examination**

Gait is an important part of the neurologic examination in patients with dizziness. Unsteadiness of the gait is increased in the dark and on uneven or inability to stand with feet apposed with eyes closed. (eg. bilateral vestibular neuropathy). Patients with acute vestibular loss are unsteady and often veer or fall toward the side of the affected ear. A wide-based gait with inability to tandem walk is characteristic of truncal ataxia. Classifying patients with vertigo and imbalance A careful history that accurately details the important features of the vertigo (acute-onset, recurrent spontaneous episodes, or recurrent positional episodes), and patients with only imbalance should be distinguished from those with non-specific types of dizziness.

**Acute-onset vertigo**

The elderly patient presenting with new onset vertigo probably has stroke or vestibular neuritis. Although ischemia to any portion of the vestibular pathway can cause vertigo, infarcts in the territory of the anterior inferior cerebellar artery (AICA) and the medial branch of the posterior inferior cerebellar artery (mPICA) are the most likely to be confused for inner ear disorders. An acute-onset with accompanying focal neurologic symptoms, particularly when they correspond to the posterior circulation, suggests an ischemic stroke. AICA territory infarct should be considered whenever a patient presents with the hyperacute onset of unilateral deafness and vertigo (AICA supplies the inner ear), central abnormalities can occur because the AICA serves the middle cerebellar peduncle, anterior cerebellar hemispheres, and lateral inferior pons. On the other hand, cerebellar infarction in the distribution of the mPICA can lead to isolated vertigo without auditory symptom (the infarct is in the caudal cerebellum just off the midline). Red flags include hyperacute onset vertigo, occipital headache or profound gait ataxia. A key point to note is that the head impulse test is intact in cerebellar strokes.
head impulse or head thrust test: the examiner holds the patient's head and asks him to fixate on her nose (Fig. 2). The examiner then delivers a discrete, low amplitude (15-20 u), but very fast, head thrust to one side. If the patient's vestibular-ocular reflex (VOR) is intact then, when the head is rotated, the eyes will remain fixated on the examiner's nose. If the VOR is unilaterally impaired then, when the head is rotated the patient's eyes will momentarily lose their fixation on the examiner's nose (Fig. 2). Indeed, in the acute setting head impulse test (eg, no catch-up saccade) is the only bedside test that can reliably distinguish an mPICA infarct from vestibular neuritis when the cerebellar examination is otherwise normal 23-25. Although itself not life threatening, distinguishing vestibular neuritis from stroke for example, is essential not only to avoid missing a serious diagnosis, but also to avoid over-investigation and inappropriate lifelong treatment for secondary stroke prevention. Large PICA territory cerebellar infarction can cause brainstem compression, hydrocephalus, cardiorespiratory complications, coma, and death. Vestibular neuritis is presumed to be due to reactivation of latent herpes simplex 1 in the vestibular ganglion 26. Vestibular neuritis affects adult most often between 30 and 60 years of age. Symptom and clinical sign of the acute unilateral labyrinthine deficit are spontaneous nystagmus, with quick phase and rotatory vertigo to unaffected side, and deviation by a tendency to fall, ocular torsion, and deviation of the subjective visual vertical and the subjective straight-ahead to affected side. Additionally, it is possible that some practitioners may confuse vestibular neuritis with other diagnoses, including migraine-associated vertigo (MAV). MAV is most often seen in patients having some prior history of headaches or a family history of migraines 27. The variability of symptom duration in patients with MAV, with dizziness lasting from several hours to even a whole day, may explain why there might be some difficulty in properly distinguishing between this entity and cases of vestibular neuritis.

Recurrent spontaneous vertigo

Ménière's disease is the prototypical disorder characterized by recurrent spontaneous episodes of dizziness. Ménière's disease is clinically characterised by recurrent spontaneous attacks of vertigo, fluctuating hearing loss, tinnitus and aural fullness. Its incidence varies between 7.5 per 100,000 to 160 per 100,000 persons 28. Patients reporting recurrent vertigo attacks lasting hours. Transient ischemic attacks (TIA) should be a concern in the patient who presents with new onset recurrent spontaneous attacks of dizziness. TIA generally lasts for minutes, less than is typical for Ménière's disease. Recurrent spontaneous attacks of dizziness is often the initial symptom of an impending basilar artery occlusion 29. Transient ischemia should be a leading concern when the patient reports recent onset brief attacks, particularly if the attacks are increasing in frequency (i.e., a crescendo pattern). In patients with recurrent attacks of vertigo, rotational vertebral artery occlusion, migrainous vertigo and vestibular paroxysmia syndromes should be suspected.

Recurrent positional vertigo

The patient complaining of recurrent episode of vertigo triggered by certain head movements likely has BPPV, but this is not the only possibility. In acute presentations, patients are often more frightened by symptoms than debilitated by them. It is important to recognize this cause because it can be readily treated at the bedside and because identification of the key features is the most effective way to exclude a central nervous system cause of positional dizziness. The most common triggers for BPPV episodes are extending the head back to look up (top shelf vertigo), turning over in bed, or getting in and out of bed. The BPPV is particularly common in older people patient, characteristics associated
with BPPV are older age, history of head trauma, inner ear surgery, other inner ear disease and prolonged recumbency.

**Dysequilibrium**

Dysequilibrium is an imbalance or unsteadiness while standing or walking and it is caused by a variety of factors. Elderly walking performance then starts to decline and the elderly slow down gradually. Many individuals suffer from imbalance, rather than spinning or an abnormal “head” sensation. If the patient is complaining of balance problems, the single-leg stance (SLS), Romberg, and tandem Romberg tests are also performed. Bohannon and associates have found that SLS times significantly decrease as people get older. When imbalance is acute, stroke of cerebellum is the leading concern. Moreover, patients with cerebellar hemispheric strokes not also involving the brainstem may complain of vertigo without any other symptoms. Episode of imbalance may be caused by vertebro-basilar insufficiency (VBI). VBI results in transient ischemia of the posterior cerebral circulation, resulting in vertigo usually lasting minutes. Additional neurolologic signs, including dysarthria, numbness of the face, hemiparesis, headache, diplopia, visual field defects, blindness, dysphagia, ataxia, and drops attacks, may also be present. When symptoms are subacute but rapidly progressive, an autoimmune ataxia, post-infectious cerebellitis paraneoplastic disorder, or even the Brownell–Oppenheimer variant of Creutzfeldt–Jakob disease should be considered. The most common type of imbalance in older people is a gradual onset with slow progression (eg. cerebellar ataxia, extrapyramidal features, spastic or peripheral neuropathy). Other disorders that cause imbalance are leukoaraiosis, normal-pressure hydrocephalus (NPH), and large-fiber peripheral neuropathy. Some older people with confluent white matter hyperintensities (leukoaraiosis) describe lightheadedness in addition to imbalance while upright. In NPH, there is a triad of symptoms consisting of dementia, imbalance, and urinary incontinence, with communicating hydrocephalus found on computed tomography (CT) or MRI of the head. Non-vestibular imbalance, defined as imbalance that is not due to an inner ear or vestibular nerve disorder, can be very frustrating to the clinician, because the symptoms are often vague and the vestibular test results are normal. In elderly individuals, there is progressive decline in muscle bulk, joint range of motion, and reflex time. Increased exercise can reduce the rate of this decline.

**REFERENCES**