Etiology and Evaluation of Dizziness in Aging

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Dizziness is a frequent complaint in aging. Although a number of circumstances in everyday life may cause falls and injuries, such events are often due to dizziness induced by pathology. Dizziness as a consequence of more than one organic syndrome is not uncommon in the elderly. In addition, medication may further complicate the clinical picture. Such combinations may pose diagnostic challenges. This paper deals briefly with postural and gaze control in health and disease, how to take the history in dizziness, some peculiarities of the clinical examination, and the most common syndromes of brain stem or inner ear diseases that cause dizziness in general and, more specifically, in aging.

Key words: dizziness, vertigo, falls, vestibular dysfunction, brain stem disease.

Introduction

Dizziness may be defined as the distorted perception of space. Vertigo is a special kind of dizziness for which the predominant sensation is rotation that may be felt either as the patient rotating in relation to the environment or the other way around.[1] Patients describe dizziness differently depending on the predominant sensation, which may be imbalance, lightheadedness, instability of the visual world, sensation of rotation, a combination of various perceptions or even perceptions difficult to describe (Table 1).[2] Dizziness may either be due to an abnormal peripheral sensory input, such as in vestibular dysfunction, or an illusion in the presence of normal peripheral sensory inputs, such as in psychogenic dizziness.[3]

In addition to other abnormal sensations, dizziness also frequently induces disturbances of postural control. Loss of postural control is a serious problem in aging and may cause falls and injuries. Falls may be due to inattention, irregular or slippery floors, medication, frailty, reduced vision or a combination of various factors. In aging, these risk factors become significant due to reduced functional capabilities. In addition, in frail elderly persons reduced postural control may further deteriorate with the use of tranquilizers or sleeping pills.[4] These types of medications also may aggravate other conditions, such as postural hypotension induced by medication for high blood pressure. Such conditions are frequently described as dizziness.

Despite these eventualities, it is wrong to assume that these are the main causes of dizziness or to conclude that specific clinical syndromes that are known to cause dizziness are not common in aging.[5] In addition, "dizziness in aging" as a specific clinical entity does not exist.[6] Causes of dizziness are the same in all age groups, although one has to take into account the functional peculiarities in aging.[7]

What follows is a brief account of normal postural and gaze control, the history in dizziness, the clinical examination and some common syndromes that cause dizziness in aging.

Normal Postural and Gaze Control

Three main sensory modalities contribute to normal postural and gaze control:

- proprioception (skin, muscle and joint signals);
- vision (optokinetic information) and;
- vestibular information.

Hearing also may be included, which, if reduced in aging due to presbycusis, may at times contribute to space disorientation. This diverse sensory information reaches various parts of the central nervous system, where a response is formulated and appropriately executed under normal conditions.[8] Therefore, dizziness—a disruption of this process—may be caused by:

- abnormal or reduced sensory information (e.g., vestibular dysfunction, loss of visual acuity);
- abnormal central processing of the sensory information (e.g., due to tumours, or vascular or other degenerative CNS processes) or;
- abnormal response of the periphery to CNS signals (e.g., muscle weakness, stiff joints).

Proprioception is essential for normal postural control. Its importance becomes apparent in peripheral sensory neuropathies, such as in diabetes or advanced kidney disease, which may cause severe unsteadiness. Occasionally, the presence of vestibular neuropathy aggravates these conditions.[9]

Another important source of sensory information is vision, which becomes essential during motion. Vision, an optokinetic signal, also reaches the vestibular nuclei, which lack the capability to distinguish between self-motion and motion of the environment. However, this signal "supplements" vestibular

Table 1
The Predominant Sensations in Dizziness

Imbalance or the ground not being solid
Lightheadedness or pressure around the head
Visual world unstable (usually moving as a pendulum)
Sensation of rotation (horizontally or vertically)
Combination of various sensory perceptions
Sensation difficult to describe

information so that the eyes normally stay on the desired visual target during head motion.

The vestibular system is a major contributor to both postural control, which is achieved by the vestibulospinal reflexes (VSR), and gaze stabilisation, which is achieved by the vestibulo-ocular reflex (VOR). Due to the five sensors in the inner ear (three semicircular canals and two otoliths), these reflexes are activated during both angular and linear head motion.

In aging, simultaneous disruption of more than one process affecting postural control and gaze stabilisation is not uncommon. Frequently, such conditions become a diagnostic challenge.

The Clinical History

Despite recent technological developments, the history in dizziness is of paramount importance. The important points to define are the predominant sensation, the time and the circumstances of onset, the concomitant symptoms, if any, and the evolution of symptomatology (Table 2).[2] In other words, it is essential to garner whether the symptoms subside or are intermittent, and how the present condition differs from the initial symptoms. If the symptoms are intermittent, it is important to ask what triggers them, how long they last and whether the patient noticed something that aggravates or alleviates the symptoms. Finally, questions about the patient's other medical problems and medications should be included.

The Clinical Examination[10]

The clinical examination in dizziness includes the postural control and the oculomotor function, among others. Postural control will be tested in Romberg's position and during tandem gait, first with eyes open, then with eyes closed. If the patient is examined shortly after an acute loss of unilateral vestibular function, it is quite possible that nystagmus and past-pointing will be present in addition to other signs and symptoms. Past-pointing is the deviation of the body from midposition induced by the acute vestibular dysfunction in absence of vision. To test for past-pointing, the patient assumes the Romberg's position with eyes closed, and extends the arms forward. In this position there is a deviation of the extended arms towards the direction of the slow phase of nystagmus.

The examination of oculomotor function includes the testing of the pursuit function (following an object moving slowly in front of the eyes) and the saccadic eye movements (moving the eyes quickly from one visual target to another in all gaze directions). Determining the presence of nystagmus, and its type, is essential. The search for nystagmus is first attempted in upright position and all gaze directions (spontaneous nystagmus). Then the patient assumes the supine position and the eye movements are observed in this and the two lateral head positions, and in both right and left gaze directions, in relation to patient (positional nystagmus). Due to compensation, absence of postural disturbances or nystagmus does not necessarily indicate normal vestibular function.

The history and the physical examination are the main diagnostic tools at hand, while more specific tests, such as electronystagmography, audiology or CNS imaging techniques, may be employed to confirm or clarify the obtained information.

Frequent Syndromes that Induce Dizziness in Aging[3,10,11]

Central Nervous System Syndromes

CNS pathology may induce dizziness in slowly progressing diseases, for which the patient may never experience vertigo and may just complain of increasing loss of postural control, or in diseases with acute onset, for which vertigo may be the prominent sensation. Thus, the symptomatology depends on whether CNS functions, which contribute to postural control and gaze stabilisation, fail slowly or acutely. It is also possible for symptoms to be intermittent.

Table 2
The Pertinent Questions in Clinical History

Predominant initial sensation
Concomitant initial symptoms
Onset acute or progressive
Duration of initial sensation(s), if of acute onset
Time of onset
Circumstances at onset
Evolution of dizziness – subsided? when? – intermittent? how often?
What triggers the dizziness, if anything?
What stops the dizziness, if anything?
Other diseases
Medications

As CNS abnormalities are revealed with increasingly accurate imaging techniques, one has to be careful not to indiscriminately attribute dizziness to such findings. In other words, any correlation between dizziness and CT, MRI or even angiographic findings must be firmly established. It is also possible that such imaging findings detect CNS diseases that may aggravate dizziness due to other causes, such as CNS vascular disease, Meniere's disease, multiple sclerosis and benign paroxysmal positional vertigo.

In most cases of brain stem involvement, there are concomitant symptoms, such as distinct double vision, vertical or horizontal (caution: not hazy) vision, facial numbness, slurred speech or hemiparesis. However, in rare cases persistent positional nystagmus—that is, nystagmus present in head positions other than upright—has been found to be the only initial clinical sign of brain stem pathology.[12] Rarely, persistence of acute symptoms, which indicates lack of compensation, may also be the only sign of such pathology. Dizziness due to CNS

disorders seems to be less frequent than dizziness due to inner ear pathology, even in aging.[7]

Peripheral (Inner Ear) Syndromes

What complicates the clinical picture of inner ear disease in aging is the possible presence of concomitant CNS pathology (cerebro- or cardiovascular disease, Parkinson's disease), non-CNS pathology (arthritis, muscle weakness, reduced vision) or other factors (medication).

By far the most common syndrome of peripheral origin is benign paroxysmal positional vertigo (BPPV).[7] Occasionally, this may be confused in the elderly with CNS disease, particularly with vertebrobasilar insufficiency. The clinical examination confirms the diagnosis of BPPV, as the observed provoked nystagmus during positional testing is characteristic of the involved semicircular canal.[13] In the great majority of cases, BPPV is due to pathology of the posterior semicircular canal. In such cases, if the patient is examined during the active phase of the syndrome, the nystagmus induced in the provocative head position is torsional in gaze towards the lowermost ear and linear upward oblique in gaze towards the opposite direction. Post-traumatic cases, even after minor head injuries, are not rare. Occasionally, the "liberatory maneuvers" may help in reducing the active time period of the syndrome.[10]

Another syndrome that may be confused with brain stem pathology is sudden (sensorineural) hearing loss.[14] In this condition, the patient suddenly loses hearing in one ear with the severity of loss varying from case to case. Concomitant acute vertigo is a frequent event and may indicate loss of vestibular function in the affected ear. There are a variety of causes, but cases in which no apparent cause can be identified are attributed to viruses affecting the inner ear. These cases are called idiopathic. In rare cases with cardiovascular disease, however, the cause may be a thromboembolic event of the labyrinthine artery or one of its branches. In cases of acute bilateral loss of sensorineural hearing, the etiology is due to autoimmune processes in most cases.

Meniere's disease is another inner ear syndrome that is not a rare event in aging.[14,15] Although in most cases the onset occurs in early adult life, cases with onset in advanced age have been encountered. The diagnosis may become more challenging and the patient's symptoms more difficult to control in this age group due to the frequency with which concomitant syndromes are present. In absence of contraindications, the treatment of choice may be infusion of small dosages of gentamycin in the middle ear.

Psychogenic dizziness[3] also is part of the pathology in aging,[2] which may be induced either by the fear of impending disease or the fear of living alone. It may also be due to various functional syndromes. The diagnosis becomes frequently clear from the history, which is neither compatible nor suggestive of organic syndromes. The negative clinical examination or the inconsistency of alleged postural disturbances on a day when symptoms are supposed to be present confirms the diagnosis. Occasionally the diagnosis is difficult, especially in patients who had previous experience of dizziness due to organic disease and who relate a history based on their previous experience. In persistent cases, psychological counseling may become necessary.

It is also worth mentioning multifactorial conditions, for which dizziness may be due to:

- concomitant pathologic processes;
- slowly failing functions;
- diseases with no CNS or inner ear involvement (e.g., cardiovascular, respiratory diseases) or;
- various drugs, especially those acting on the CNS.

A combination of such conditions is not unusual. Occasionally, this may become a diagnostic challenge, especially in elderly patients. ◆

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