HOW TO MANAGE DIZZINESS*

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INTRODUCTION

In the words of W.B. Matthews, there are few physicians so dedicated to their art that they do not experience a slight decline in spirits on learning that their patient's complaint is of dizziness. It seems as if there is a multitude of potential causes, many of which are difficult to diagnose and treat. The aim of this paper is to provide a simple, structured approach to the assessment of this challenging problem.

Acute dizziness is relatively straightforward. Occasionally it will be due to sudden hypotension in the context of an acute MI or arrhythmia, or significant blood loss. Other common causes include an acute cerebellar or brain stem stroke in older age groups and, in younger patients, vestibular neuronitis or possibly a first episode of Ménière's disease. The presentation with chronic episodic symptoms is a more difficult problem and it is this that will be explored in more detail.

Dizziness is a symptom which covers a vast range of sensations, many of which are hard for patients to describe, making assessment very difficult (Box 1). However most of these sensations fall into one of three categories: a sensation of movement or vertigo; lightheadedness and pre-syncope, or loss of balance or unsteadiness. It may help patients to be offered these phrases as possible descriptions when they are struggling to explain what they feel. This then helps to decide what approach to take in their further investigation and management.

Box 1

Terms patients use to describe dizziness:

- **Woozy**
- **Muzzy**
- **Giddy**
- **Spinning**
- **Burling**
- **Staggering**
- **Veering to the side**
- **Tottery**
- **Shaky**
- **Just not right**
- **All queer**

VERTIGO

Vertigo is defined as sensation of movement of either oneself or the surroundings. It can be a rotatory or a to and fro sensation, and can occur spontaneously or only on change of position or posture. It may be caused by peripheral vestibular disease or disease of the brain stem. It is difficult to distinguish these causations from the history alone but it is important to enquire about hearing loss or other neurological symptoms. Common disorders include Benign Positional Vertigo and Ménierè's disease in younger and middle-aged patients, and posterior fossa disease of vascular origin in older patients. Space occupying and demyelinating lesions are rare.

Benign Positional Vertigo

Benign Positional Vertigo (BPV) is a condition in which clusters of calcium carbonate crystals move freely within the posterior semicircular canal. This can occur as a result of utricular degeneration associated with ageing, head trauma or viral infection. Whenever the head is moved these clusters cause turbulent flow within the endolymph. As a result, the patient experiences transient vertigo for a few seconds every time they turn, often most particularly in bed.

The Hallpike test is a manoeuvre to establish the diagnosis of BPV.[1] It involves sitting the subject on a couch and then tipping them backwards, so that their head hangs over the end of the couch, first rotated to the right and then the manoeuvre is repeated with the head rotated to the left (Figures 1 and 2). A positive response is associated with a latent period of a few seconds, followed by severe vertigo and torsional nystagmus which lasts up to one minute. Both the symptoms and the vertigo fatigue on repeat testing. The test can also induce nystagmus in those with posterior fossa lesions, but in these cases there is no vertigo and no latent period, and the nystagmus is vertical and does not fatigue. If any of these features are present, radiological imaging is required. There is no other diagnostic test for BPV.

A series of manoeuvres has recently been described which are curative in BPV and can be performed in the clinic at the time of diagnosis[2] (Figures 3-6). The theory behind these manoeuvres is that they cause the clusters of crystals which are loose within the posterior semicircular canal to be dispersed. When a positive Hallpike response is obtained, the patient should then have their head turned to the opposite side. They should then be rotated so that they are lying on their side with their head facing obliquely downwards. They should then be sat up and their head held slightly flexed to 45°. Each position should be held for at least 30 seconds and the patient should be advised to keep their head upright for the next 48 hours, and to try to sleep propped up as much as possible. This has been shown to be effective in 77% of patients after one treatment session, and in another 20% after a second trial a week later.[2]

Ménière's disease

Ménière's disease is associated with dilatation of the

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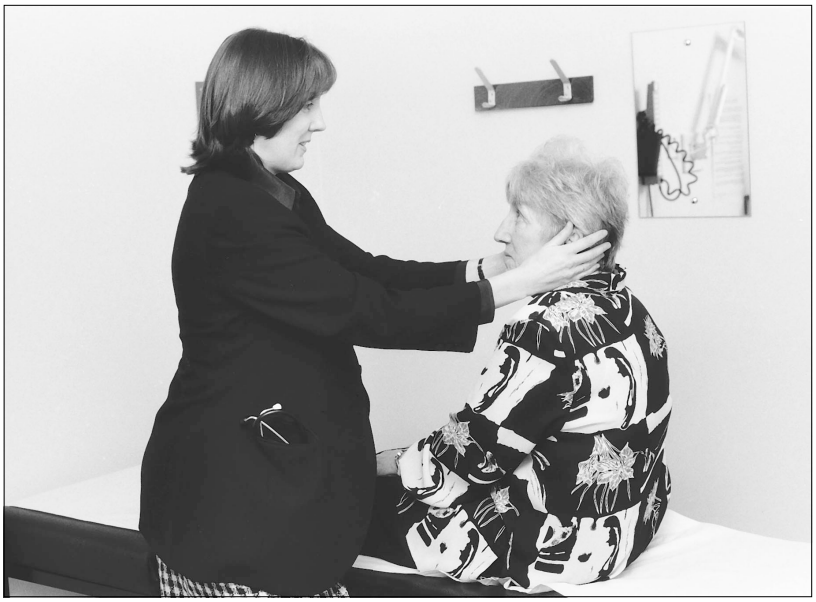

FIGURE 1

FIGURE 2

FIGURES 1 AND 2
The Hallpike test: The subject is seated on a couch (Figure 1) and then tipped backwards, so that their head hangs over the end of the couch (Figure 2), first rotated to the right and then repeated with the head rotated to the left. See text for further details.

FIGURE 3

FIGURE 4

FIGURE 5

FIGURE 6

FIGURES 3-6

Treatment of Benign Positional Vertigo: When a positive Hallpike response is obtained, the patient should be maintained in position for at least 30 seconds after the symptoms and nystagmus have resolved (Figure 3). The patient should then have their head turned to the opposite side (Figure 4). They should then be rotated so that they are lying on their side with their head facing obliquely downward (Figure 5). They should then sit up and with their head held slightly flexed to 45° (Figure 6). Each position should be held for at least 30 seconds.

membranous labyrinth, the cause of which is uncertain. It is usually unilateral and results in insidious loss of hearing and the development of tinnitus. Vertigo occurs at first as acute attacks, which are often preceded by an increase in the level of tinnitus and pain or fullness in the affected ear. The vertigo tends to be of prolonged duration lasting several minutes, hours or even for a whole day. It can be associated with pallor, nausea and vomiting, and varies in intensity from slight unsteadiness to severe, prostrating vertigo.

The onset of Ménière's disease is more common in middle than old age and initially the diagnosis is made on clinical grounds. In the later stages of the disease, fluctuating sensorineural deafness can be detected on audiometry, particularly for lower pitched sounds. In the later stages, these fluctuations stop, and deterioration becomes progressive. In cases where there is doubt, electrocochleography may give a highly characteristic waveform, but it can be negative in the early and later stages of the disease.

Acute attacks are treated with vestibular sedatives such as prochlorperazine, cinnarizine or promethazine. Few controlled trials of maintenance therapy have been carried out and most treatment is empirical. Dietary salt restriction and diuretics can be used in an attempt to modify the dilatation of the membranous labyrinyth: the use of the histamine analogue, betahistine, has been found to be beneficial.[3] Other treatments, including surgery, are beyond the scope of this paper.

Disorders of the posterior cranial fossa

Where there is any doubt about the diagnosis, posterior fossa disease has to be excluded by imaging. Formal otolaryngological assessment to identify at-risk patients may be more appropriate than performing an MR scan at once, unless there are obvious neurological abnormalities or the patient is at high risk of vascular disease by virtue of their age or other risk factors, or they have an atypical Hallpike response. The incidence of cerebellopontine angle masses in patients presenting with dizziness is in fact very low, and it has been calculated that 2,500 imaging studies would have to be performed to identify one cerebellopontine angle mass.[4] Even in high-risk patients with dizziness and assymetric hearing loss, 638 imaging studies would have to be performed to identify one such mass.

LIGHTHEADEDNESS

Lightheadedness can be a very difficult symptom to untangle. Cardiovascular disease is an important possibility in this instance, and there can be a large overlap with the causes of syncope. Some 'specific' types of syncope are obvious from the history such as cough, micturition and defaecation syncope: these result from a variety of autonomic reflexes such as the Vasalva manoeuvre whilst straining, vagal stimulation by a full bladder, reflex vasodilatation by bladder emptying and reflex heart rate changes with cough. Drugs may also cause vasovagal symptoms as a result of volume depletion or altered vascular tone, the best known example being GTN syncope.

Cardiac causes

These include outflow obstruction due to aortic stenosis or cardiomyopathy, and arrhythmias, of which the most common causing dizziness or syncope in old age are sick sinus syndrome and ventricular tachycardia, and in younger patients, paroxysmal supraventricular tachycardia. It can be difficult to interpret 24-hour ambulatory ECG abnormalities in older people as the vast majority of detected arrhythmias are brief and produce no symptoms.[5] Thus any arrhythmia on 24-hour ambulatory ECG must be associated with a documented episode of symptoms before it can be assumed the cause of a patient's presentation rather than an incidental finding.

Postural hypotension

This is a common cause of dizziness and its precipitants are shown in Table 1. There is poor correlation between postural symptoms and postural hypotension, symptoms being much more common and more commonly associated with subsequent falls in old age.[6] Some argue that this is a reflection of those with abnormalities of cerebral rather than peripheral autoregulation.[7] However others have noted that postural hypotension may be missed if routine blood pressure equipment rather than beat-to-beat measurement of blood pressure is used.[8]

TABLE 1
Causes of postural hypotension.

Physiological changes of ageing
- Reduced baroreceptor sensitivity
- Excessive venous pooling
- Autonomic dysfunction

Pathology
- Dehydration
- Anaemia
- Cardiac failure
- Diabetic autonomic neuropathy
- Multiple Systems Atrophy
- Shy-Drager syndrome
- Addison's disease

Drugs
- Anti-hypertensive agents
- Diuretics
- Anti-depressants including tricyclics and SSRIs
- Phenothiazines
- Anti-Parkinsonian therapy

Treatment includes advice to rise carefully and to avoid heavy meals. Head-up tilt of the bed can also help. Any medication which could be contributing should be stopped or reduced. TED stockings can be recommended though few elderly patients can get these on. Caffeine is said to help by causing vasoconstriction of capacitance vessels. A small dose of a non-steroidal anti-inflammatory agent can be useful as it causes mild salt and water retention, but GI side-effects can be limiting. In severe cases, fludrocortisone can be used but it may cause excessive fluid retention and overload. Midodrine, an alpha blocker, is another option but it is not yet licensed in the UK.

Carotid sinus sensitivity and vasovagal syncope

These are much more common causes of syncope (and possibly dizziness) in old age, than previously appreciated:[9] both are the consequences of exaggerated baroreceptor reflexes which can cause bradycardia, hypotension or both.

Carotid sinus sensitivity occurs in response to pressure on the carotid sinus with stimulation of the baroreceptors in its wall with impulses being transmitted via the glossopharyngeal nerve to the brain stem. This in turn generates a response via the vagus nerve to the sinoatrial and atrioventricular nodes, and the sympathetic chain to peripheral blood vessels. A transient slowing in the sinus rate occurs as well as a minor drop in blood pressure. In those with carotid sinus sensitivity, reactions are exaggerated and include a positive cardio-inhibitory response (which is defined as a period of asystole for longer than three seconds), or a positive vasodepressor response (which is defined as a drop in systolic blood pressure of more than 50 mmHg, in the absence of a bradycardia). A mixed response can also occur. These responses can be reproduced clinically by carotid sinus massage which is performed by rubbing longitudinally over each carotid pulse in turn for five seconds with continuous ECG and blood pressure monitoring. Full resuscitation facilities should be available. The pick-up rate is increased by performing massage in the head-up tilt positon as well as in the supine position. Massage should **not** be performed in those with a carotid bruit, known cerebrovascular disease or recent myocardial infarction.

In those with vasovagal syncope, prolonged standing can lead to similarly defined hypotension or profound bradycardia associated with syncope. In this case, the abnormality lies in the baroreceptor responses within the ventricles to reduced venous return. This can be reproduced by tilting the patient to 70° for 30 minutes, again with continuous monitoring.

Carotid sinus sensitivity is found in approximately 5% of asymptomatic elderly people, but in a highly selected group of elderly people with falls, syncope or dizziness unexplained after initial investigation, nearly half were found to have carotid sinus sensitivity.[9] As up to a third of elderly people have retrograde amnesia for syncope, it has been suggested that the condition may also be present in a proportion of those who deny any loss of consciousness, and that there is therefore a place for routine carotid sinus massage in any elderly patient with unexplained dizziness or falls. The picture is less clearcut for vasovagal syncope as it occurs in many more of the asymptomatic elderly at around 11%.[10]

Treatment for these conditions can be difficult. Those with a positive cardio-inhibitory response to carotid sinus massage respond well to permanent pacing.[9] It is much more difficult to treat the vasodepressor response, but ephedrine and fludrocortisone can be helpful. Treatment for vasovagal syncope is even more difficult but some success has been reported with beta blockers, disopyramide and theophylline, although their mechanisms of action are poorly understood, and there have been no placebo-controlled trials.[5] In those with significant bradycardia on tilting in whom medical therapy has failed, insertion of a permanent pacemaker may be considered.

To summarise, the appropriate investigations in those with lightheadedness would be:

- A 24-hour ambulatory ECG which may have to be repeated several times until a definite episode of symptoms occurs.
- Echocardiography can confirm the diagnosis of outflow obstruction if suspected from the presence of a murmur.
- Carotid sinus massage.
- Tilt testing should be performed in patients who are having recurrent episodes of dizziness or collapse for which no other explanation has been found.

UNSTEADINESS

Some patients, particularly older people, use the term 'dizziness' to describe symptoms of poor balance and unsteadiness. A full discussion of the physiology of balance and locomotion is beyond the scope of this paper, but clearly any pathology that impairs sensory input from vision, proprioception or the vestibular system, or impairs rapid righting reflexes, central processing or the effector response, has the potential to cause imbalance. In addition, pathology affecting the mechanical support system of bone and joints will adversely affect balance. Exercise tolerance is also important, and may be impaired by disease of the cardiorespiratory system.

Nutt *et al.*[11] have suggested a useful classification for balance and gait disorders in terms of the level of the sensori-motor disturbance (See Table 2). These can usually be identified by a thorough neurological examination and observation of the patient's gait.

Lowest level disorders encompass peripheral sensori-motor problems, while middle level disorders include gait abnormalities due to disease of the pyramidal, cerebellar and basal ganglia motor systems. The highest-level gait disorders are not well understood, and can be difficult to define clinically.

Cautious gait is often a response to real or perceived dysequilibrium and is not specific to any pathology. It is often seen amongst older people anxious after a fall. Patients walk with a widened base with body, hips and knees bent to place their centre of gravity forward. The arms are sometimes held abducted and flexed.

TABLE 2
Classification of Gait disorders (after Nutt *et al.*)[11]

Lowest level

Peripheral musculoskeletal disease
- Arthritis
- Myopathy
- Peripheral neuropathy

Peripheral sensory disease
- Sensory ataxia
- Vestibular ataxia
- Visual ataxia

Middle level
- Hemiplegia
- Paraplegia
- Cerebellar ataxia
- Parkinsonism

Highest level
- Cautious gait
- Subcortical dysequilibrium
- Frontal dysequilibrium
- Isolated gait ignition failure
- Frontal gait disorder

Subcortical dysequilibrium results in severe abnormalities of postural reflexes so that the patient has great difficulty standing or walking with a tendency to fall backwards, despite relatively preserved strength and sensation. This may be associated with vertical gaze palsies, dysarthria and extra-pyramidal signs, and has been reported after thalamic, basal ganglia and mid-brain stroke. It is also seen early in the course of Progressive Supra-nuclear Palsy and Multiple Systems Atrophy.

Lesions in the frontal lobe can lead to a variety of gait abnormalities. *Frontal dysequilibrium* results in bizarre gait patterns, often with crossing of the legs and ineffective propulsion. This pattern is seen with a wide variety of structural lesions of the frontal lobes.

Isolated gait ignition failure appears very similar to a Parkinsonian gait but with only initial hesitation and freezing, and no other clinical signs of Parkinsonism. The underlying problem is not clear, but it has been suggested that it may be due to an isolated focal degenerative cortical condition. However in some cases, patients progress to frontal gait disorder, in which case cerebrovascular disease or rarely a frontal lobe tumour may be responsible.

Frontal gait disorder is characterised by difficulty in starting to walk, short steps, shuffling and hesitation on turns, with freezing (coming to a sudden stop) and poor balance. Patients frequently also have cognitive impairment, pseudobulbar signs and urinary incontinence. Vascular disease is the most common cause, particularly subcortical arteriosclerotic encephalopathy (Binswanger's disease).

DIZZINESS IN OLD AGE

The most common presentation in old age is the patient who feels unsteady and lightheaded most of the time, with occasional more severe episodes during which the patient has to hold on to something to prevent a fall. Multiple pathology is often present, and a thorough assessment is required to identify all the contributing factors. It can sometimes be difficult to decide what further investigations are useful.

Evidence in this respect comes from a study which set out to identify the causes of dizziness in elderly people recruited from the community, and to identify the clinical findings and investigations that best discriminated subjects from non-dizzy controls of the same age.[12] Significant differences between dizzy and control subjects were identified on clinical examination, particularly neurological signs suggestive of small vessel cerebrovascular disease, impaired visual acuity and degenerative joint disease of the cervical spine and hip. Anxiety scores were much more frequently abnormal or borderline in dizzy subjects than controls. In addition specific provocation tests for dizziness including hyperventilation, head turning, Romberg testing and blood pressure measurement on postural change with any associated symptoms noted, all caused symptoms more frequently in the dizzy group. As previously discussed, significantly more subjects had symptoms on postural change without than with a blood pressure drop. The Hallpike manoeuvre was also assessed but was positive in relatively few, and carotid sinus massage was never positive.

In contrast to the clinical findings, formal investigations such as blood profile, ECG, electronystagmography and MRI of head and neck failed to distinguish dizzy from non-dizzy subjects largely because of the frequency of asymptomatic abnormalities in controls. The diagnoses underlying the symptoms of dizziness (Table 3) were designated using clinically-based criteria. Central vascular disease and cervical osteoarthrosis were the most common causes, with only 9% having no evidence of either. In 88% of the patients there were multiple diagnoses, perhaps confirming that dizziness is a common manifestation of chronic ill-health. Poor vision and anxiety usually occurred in association with other pathology.

TABLE 3
Diagnoses in dizzy subjects (after Colledge *et al.*)[12]

Diagnosis	n	%
Central vascular disease	105	71%
Cervical osteoarthrosis	98	66%
Anxiety/hyperventilation	48	32%
Poor vision	23	15%
Postural hypotension	10	7%
Benign postional vertigo	6	4%
Other	38	26%
No diagnosis	6	4%
Number with >1 diagnosis	126	88%
Number with both cervical and central vascular disease	68	46%
Number with no cervical or central vascular disease	14	8%
Poor vision only	0	0
Anxiety/hyperventilation only	3	2%

The results of this study suggest that a thorough clinical assessment will identify a cause or several causes for chronic dizziness in the majority of elderly individuals, so that specialist investigations should only rarely be required. Figure 7 shows an algorithm for this purpose.

Only in those in whom a clinical diagnosis cannot be made should investigation prove necessary. Some may have one of the more exotic causes of dizziness, such as acoustic neuroma or normal pressure hydrocephalus. However clinical assessment identifies most of the common causes of dizziness, and enables the provision of an explanation for the patient's symptoms. Reassurance often seems to result in an improvement in symptoms especially when anxiety is a contributing factor.

The next challenge is treatment. This will obviously depend on the cause, and it is important to remember that there are likely to be several. It is worth ensuring that vision is corrected as far as possible, and that anxiety is actively managed. This has been shown to improve symptoms in younger patients even when the underlying cause of dizziness cannot be treated. Postural hypotension should also be corrected as far as possible.

Treatment is most difficult in those with central vascular disease and cervical osteoarthrosis. Any risk factors such as atrial fibrillation, hypertension and smoking should be aggressively managed. Aspirin may reduce the risk of stroke in this group. In terms of improving the symptoms of unsteadiness and/or lightheadedness associated with both cerebrovascular disease and cervical osteoarthrosis, physiotherapy is very useful.[13] There's good evidence that

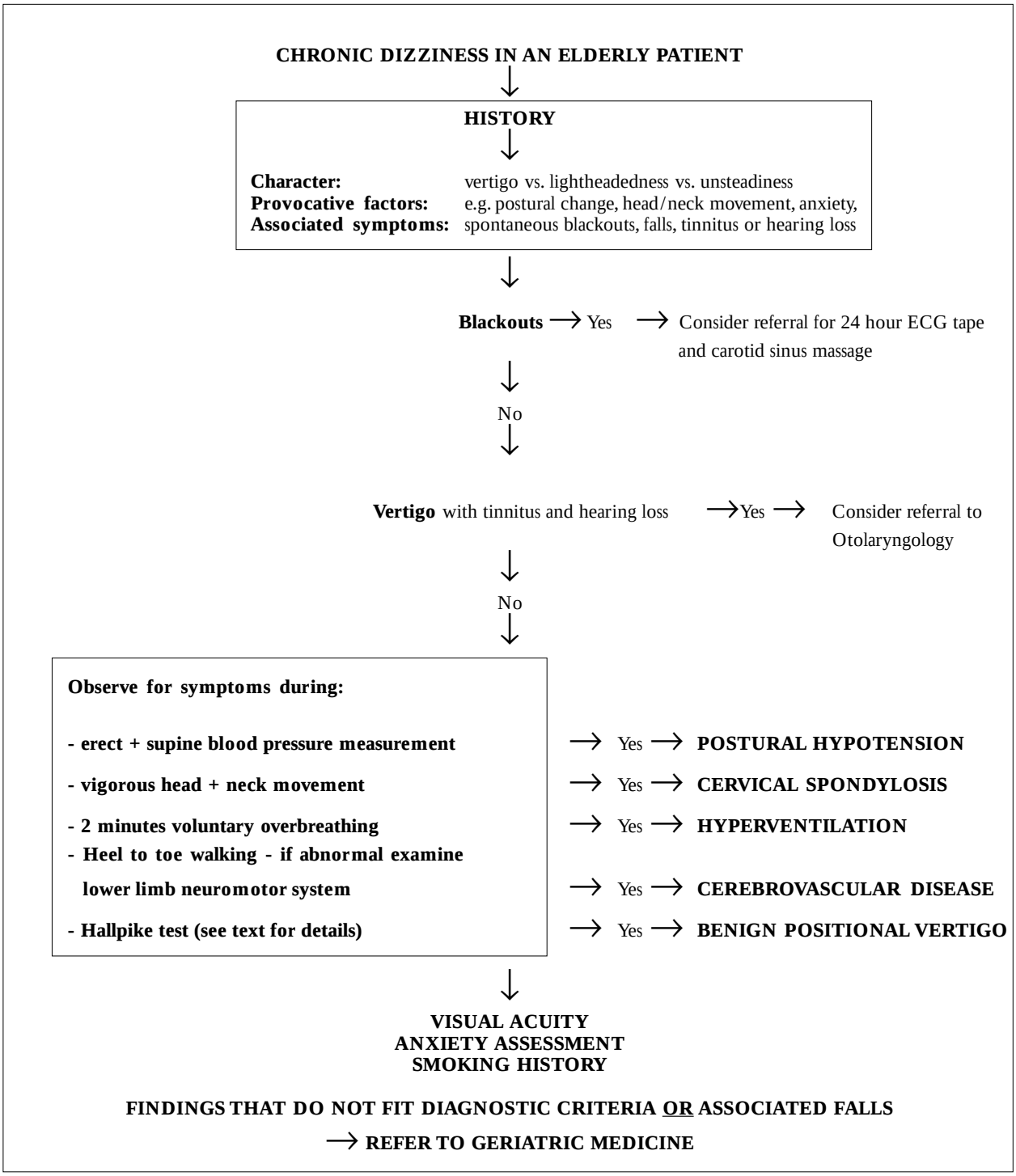

FIGURE 7
An algorithm for the assessment of older patients with chronic dizziness.

balance can be improved with specific training in old age[14] and also that it reduces the subsequent risk of falls.[15] Habituation to positions that provoke dizziness can help, possibly by encouraging adaption and reducing anxiety:[16] this is the basis of so-called vestibular rehabilitation.

CONCLUSION

This paper has concentrated on a practical approach to managing patients with dizziness due to the common causes. It cannot be denied that many exotic and fascinating pathologies that can underly dizziness have not been discussed, but these are rarely seen in general medical practice and patients are better served by those with a good working knowledge and simple management plan for the common disorders.

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