This is a well-established worldwide phenomenon where primary care practitioners, despite awareness of hypertension targets for BP, do not institute changes to treatment (including referral) despite BP levels being persistently above target. The most common reason cited for this is that the patient’s BP is at an acceptable level.

Another very important obstacle is the lack of specialist facilities for referral of hypertensive patients in the public sector. In the Western Cape the waiting time to see a specialist physician as an outpatient is often >6 months and there is only one specialist hypertension clinic in the entire region where only 4 - 5 new patients can be seen per week.

**Conclusion**

Essential hypertension should primarily be managed by primary care practitioners. However, in certain circumstances referral to a specialist for evaluation and treatment is necessary.

**Further reading**


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**Falls, dizziness and syncope in the elderly - when to refer**

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The prevalence of falls increases with age as postural reflexes become slower and degenerative diseases affecting balance and mobility manifest. Every fall that needs medical attention requires an explanation and all elderly patients should be asked if they have fallen. Falls not only have significant morbidity in themselves but are frequently the presenting symptom of a new or progressive underlying pathology (10% of falls occur during and acute illness, especially if there is delirium). Two unexplained falls within 1 year is also an indication for full assessment. The causes of falls are always a combination of extrinsic (environmental hazards), intrinsic (patient pathology) and situational (risk-taking behaviour) factors.

**Establishing the cause of the fall**

History and examination should first be directed at identifying the intrinsic factors in the patient that led to the fall so that intervention can be targeted to reduce the risk of further falls and then, because frailty is generally not reversible, identifying environmental risks so that these can be reduced. In order to identify the pathologies leading to falls one must understand how balance is maintained. As people age the postural reflexes slow, demonstrated clinically as postural sway, which leads to an inability to correct quickly for changes in posture (impaired static and dynamic balance). This, combined with deconditioning, sensory impairments and degenerative joint problems, all common in the elderly, predisposes to falls.

**Maintaining balance** *(Fig. 1)*

A comprehensive falls assessment requires a multidisciplinary evaluation of the extrinsic (environmental) and intrinsic factors that have contributed to the fall so that effective interventions can be instituted to prevent further falls. The intrinsic causes of falls can be divided into those that are constantly present and lead to
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impaired of gait and balance, which can be screened for in a functional assessment, or cognitive impairment and those which are intermittent, which are either dizziness, syncope and seizures or associated factors such as drugs or alcohol. The history includes the circumstances of the fall to identify environmental factors, as well as known medical conditions and medication and any symptoms that could indicate unidentified intrinsic pathology, particularly dizziness or syncope (Fig. 2). The history should also include the current functional status of the patient including both instrumental activities of daily living (IADLs) such as shopping, gardening, cooking, management of financial affairs and ability to use transport and activities of daily living (ADLs) such as basic self-care – bathing, dressing, grooming as well as continence and mobility. Impairment or decline in ADLs and IADLs are useful indicators of new or progressive pathology and increasing frailty.

Physical assessment

Physical assessment includes screening for sensory impairment (vision and Romberg’s sign, which identifies functionally significant sensory neuropathy or vestibular pathology) as well as cognitive assessment (date, delayed recall of 3 objects and clock drawing as sensitive as MMSE), a gait and balance assessment to identify significant neurological and musculoskeletal pathology and a CVS assessment which includes lying and standing BP as well as an ECG (Fig. 3).

Performance evaluation (functional assessment of gait and balance)

• Get-up-and-go – stand up from chair (knees 90°) without using arms, walk 3 m, turn and walk back and sit down. Should take < 14 seconds, watch for proximal weakness on rising, postural instability on standing up, abnormal gait, staggering on turning, which suggests more subtle pathology.
• Romberg’s sign identifies clinically significant peripheral neuropathy and postural sway.
• Sternal nudge identifies slow postural reflexes predisposing to falls.
• Tandem walk – there are no significant balance problems if 10 steps are successfully completed.
• One-legged-stance < 3 seconds = increased risk of falls.

At the end of this assessment one may have identified one or more pathologies that could have led to the fall. Common causes of falls are given in Fig. 4. Each of these needs to be assessed and managed. One needs to identify those patients who need referral.

Who needs referral for physiotherapy?

Even when the fall has resulted in no injury, a third of elderly people who have experienced a fall will develop a fear of falling, which will lead to their curtailing activities and thus a decline in function. Physiotherapy specifically to address this will prevent a fear of falling from becoming disabling. Most fallers benefit
from a short course of physiotherapy
When one has identified deconditioning and poor postural reflexes as the cause of falls, physiotherapy focused on balance training will improve postural control. Patients who have irreversible abnormal gait (e.g. previous stroke) can be assessed by a physiotherapist for the correct walking aid and can be trained to use it.

Who needs referral to an occupational therapist?
When there are irreversible physical or cognitive impairments and the risk of falling is increased one can minimise the risk by assessing and altering the patient's environment. These patients would benefit from a home visit by an occupational therapist to assess home hazards and recommend measures to address them.

Falls associated with dizziness
Dizziness can be caused by vestibular, brainstem or posterior cerebral circulation pathology and presyncope (Fig. 5). In differentiating these it is important to determine whether there is true vertigo (sense of rotatory motion). Vertigo can either be peripheral (originating from the vestibular apparatus with or without involvement of the 8th cranial nerve, causing deafness/tinnitus) or central, due to brainstem pathology where the nystagmus is vertical and nausea, vomiting and ataxia or other CNS signs should alert the clinician.

When to refer for vertigo (Fig. 6)
Benign paroxysmal positional vertigo (BPPV)
This condition is due to otoliths in the semicircular canal. The typical history is vertigo occurring on turning the head, typically on turning to the affected side in bed; the attack normally lasts a minute. This can be diagnosed using the Hallpike manoeuvre and treated with the Epley’s manoeuvre (85 - 95% success). In resistant cases an ENT opinion is needed.

Vestibular neuronitis
This is a self-limiting condition associated with a viral upper respiratory tract infection which results in vertigo and nystagmus of a few days’ duration. It can be treated symptomatically. Prednisone for 10 days may shorten the course of the illness. This should not be confused with suppurative labyrinthitis associated with otitis media, which requires intravenous antibiotics, or Ramsay Hunt syndrome, caused by varicella zoster, involving the 7th and 8th cranial nerves with deafness, tinnitus and facial palsy where acyclovir and prednisone are beneficial if started early.

Ménière’s disease (endolymphatic hydrops)
This is a clinical diagnosis based on a classic triad of fluctuating sensorineural hearing loss, tinnitus and attacks of vertigo which may last up to a few hours. The disease starts unilaterally but half of patients progress to bilateral disease. The cause is excess endolymph production causing dilatation of the endolymphatic system. The management is salt restriction and diuretics but vestibular sedatives may give symptomatic relief. In severe cases the

Fig. 5. Falls associated with dizziness.
Fig. 6. Causes of vertigo.
patient can be referred to an ENT specialist for shunting procedures or vestibular nerve ablation.

**Dizziness without vertigo**

When there is no vertigo the problem can be either episodic, where impaired cerebral perfusion is the problem (presyncope or vertebrobasilar disease), a general feeling of disequilibrium, common in the elderly and often associated with cervical spine degenerative disease, associated with malaise where there is underlying disease, or psychogenic (generalised anxiety disorder) (Fig. 7). The difficulty is distinguishing those who have true dizziness rather than a ‘fuzzy feeling in the head’. True dizziness is usually episodic. These patients all need a thorough cardiac examination including lying and standing blood pressure and ECG. Postural hypotension is defined as a fall in the systolic blood pressure of 20 mmHg within 3 minutes of standing but one can only ascribe the dizziness to this if the fall in blood pressure is accompanied by symptoms of dizziness.

**When to refer**

**Postural hypotension**

Most postural hypotension in the elderly is due to a combination of central CNS autonomic degeneration leading to slowed reflex vasoconstriction on standing combined with drugs which either interfere with autonomic reflexes or cause volume depletion. The majority can be managed by stopping or reducing the offending drugs and advising the patient to sit before standing when vasodilated (getting out of bed or bath, after big meal) and avoiding alcohol. Peripheral autonomic neuropathy is also common in diabetes mellitus. If the postural hypotension does not resolve with these simple measures one can consider either volume depletion due to cortisol deficiency (Addison’s disease) which can be screened for by measuring 9 am serum cortisol or primary central autonomic failure (multiple system atrophy (MSA)). In this condition and related syneucleinopathies, the postural hypotension may be accompanied by parkinsonism or cerebellar signs. Cortisol deficiency requires referral to an endocrinologist and MSA to a neurologist.

**Dizziness on exertion**

Dizziness on exertion is usually related to poor cardiac output. In severe cardiomyopathy, if the medical therapy has been optimised, there is little further to offer the patient. In the case of unexplained exertional dizziness one should consider silent aortic stenosis or myocardial ischaemia, in which case a cardiology assessment is indicated. In the case of known or suspected aortic stenosis the presence of this symptom is an indication for referral for valve replacement.

**Vertebrobasilar insufficiency**

The typical history is one of transient dizziness when the head is extended and rotated, e.g. drawing curtains, reaching for the top shelf. True vertebrobasilar transient ischaemic attacks (TIAs) are usually accompanied by other neurological symptoms and last at least several minutes. If symptoms are severe or TIAs recurrent one can consider referral to a neurosurgeon with expertise in posterior cerebral artery disease.

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**Fig. 7. Causes of dizziness.**

- **Dizziness on standing up**
  - Postural hypotension
  - Aortic stenosis cardiomyopathy
  - Vertebrobasilar insufficiency
  - Carotid sinus hypersensitivity
  - Generalised anxiety disorder

**Fig. 8. Causes of cardiac syncope.**

- **Outflow tract obstruction**
  - Aortic stenosis
  - Hypertrophic obstructive cardiomyopathy

- **Pump failure**
  - Cardiomyopathy
  - Ischaemia

- **Peripheral resistance**
  - Intravascular volume
  - Vessel tone
  - Sympathetic nervous system
Fig. 9. Investigation of an episode of loss of consciousness.

**More about...**

**LOSS OF CONSCIOUSNESS**

<table>
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<td>CVS assessment identifies cause of syncope</td>
<td>Investigate and manage for generalised seizures</td>
<td>Manage or refer as appropriate</td>
</tr>
<tr>
<td>If ≥ 3 episodes in 6 months refer for full syncope work-up</td>
<td>If no cause found consider implantable loop ECG recorder or seizures</td>
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**Carotid sinus hypersensitivity**

Because the diagnosis of carotid sinus hypersensitivity requires a carotid sinus massage (needs to be performed with intravenous access and full resuscitation equipment ready) and the treatment is a permanent pacemaker, the suspicion of this diagnosis should result in referral to a cardiologist.

**Generalised anxiety disorder**

This is a diagnosis of exclusion and can only be made where cardiac, ENT and CNS pathology have been excluded. The diagnosis demands that other symptoms related to anxiety such as easy fatigue, restlessness, poor sleep, irritability, poor concentration and muscle tension have been present for a minimum of 6 months. The dizziness is described as a funny feeling in the head and can be reproduced by hyperventilation. This problem seldom leads to falls.

**Falls not associated with any symptoms**

Where there are recurrent unexplained falls (normal gait and balance assessment, CVS and ECG) ‘drop attacks’ can be considered. This is an ill-defined condition, usually in the very old and frail with multiple pathologies, where there is a sudden loss of postural tone with no impairment in consciousness. The person falls to the ground with a feeling that all limbs have suddenly gone weak but recovers completely within a minute. The problem with this diagnosis is that in 40% of unexplained falls there has been transient loss of consciousness (syncope) that has not been noticed, often the presenting feature in carotid sinus hypersensitivity. All patients who have this problem require a full work-up for syncope, which requires referral to a cardiologist or geriatrician.

**Conditions leading to cardiac syncope**

A definitive diagnosis of cardiac syncope can be made in 50% of patients after the initial assessment (Fig. 8).

- Postural hypotension – manage as previously. If syncope persists despite improvement in erect BP refer for syncope investigation.
- Arrhythmias – if there is atrial fibrillation with slow ventricular response rate adjust medication and refer for permanent pacemaker (PPM) if no improvement. If the patient has sick sinus syndrome, 2nd and 3rd degree heart block, refer to a cardiologist.
- Structural cardiac lesion – if severe (aortic stenosis, hypertrophic obstructive cardiomyopathy (HOCM), ischaemic heart disease (IHD), pulmonary hypertension or severe cardiomyopathy (CMO)) refer to a cardiologist for appropriate investigation and intervention.
- Typical situational syncope with Valsalva manoeuvre (defaecation, micturition and cough syncope) can usually be managed with explanation and management of the underlying problem, but needs referral for syncope investigation if it persists.

**Unexplained syncope**

When syncope is unexplained despite a thorough cardiac examination and ECG (Fig. 9) the possibilities are neurocardiogenic syncope, carotid sinus hypersensitivity, silent aortic stenosis or HOCM or tachyarrhythmias, such as ventricular tachycardia. The investigation of unexplained syncope may be unrewarding if the occurrence is infrequent. The indication for a full syncope work-up is 3 episodes in 6 months. These patients require a tilt test (neurocardiogenic syncope), carotid sinus massage (carotid sinus hypersensitivity), echocardiogram (structural heart disease) and 24-hour ECG monitoring (arrhythmias). These investigations are best done by a syncope clinic or cardiologist specialising in syncope.

**TB patients not improving on therapy**

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Chemotherapy, which was initially introduced in the early 1940s, and subsequently improved with the intro-duction of rifampicin and pyrazinamide-based short-course therapy, results in a major impact on the outcome of TB. With no chemotherapy one-quarter of patients will die within 2 years and 50% within 5 years. Another 25% will cure themselves but a further 25% will remain persistently sputum positive.1 The response to TB therapy is usually rapid, with a decline in the number of acid-fast bacilli on smear within 2 weeks. Approximately 50% of patients will have negative smears and cultures after 2 months, 75% after 4 months and 98% after 6 months. Although...