hypertension is generally not associated with an increased cardiovascular risk. It should be suspected in a patient with an elevated BP level in the office or clinic without evidence of target organ damage (TOD), and normal or lower BP recorded at home. It may also occur in a hypertensive patient and be an important cause of pseudo-resistant hypertension. Referral to a specialist for evaluation and 24-hour BP monitoring may be appropriate, but BP monitoring at home, with a properly validated electronic device, is an alternative approach. It must also be remembered that the norms of home BP monitoring are different to those in the office. A BP of 140/90 mmHg in the office is equivalent to 135/85 mmHg in the home setting.

**Resistant hypertension**

Resistant hypertension is defined as a BP >140/90 mmHg or >130/80 mmHg in patients with diabetes or renal disease despite adherence to treatment with full doses of at least 3 antihypertensive drugs, including a diuretic. Before referral the primary care practitioner should ensure that office hypertension is excluded by home or 24-hour BP monitoring (if available) and that the patient fully adheres to therapy and is not taking interfering substances such as non-steroidal anti-inflammatory drugs (NSAIDs), amphetamines or other sympathomimetics, herbal supplements or liquorice, or steroids. In addition, if the patient is obese, he/she should first be started on a diet and an exercise programme.

**Hypertensive emergency or urgency**

Despite advances in chronic hypertension management, hypertensive emergencies and urgencies remain serious complications. These are not common, but likely to be encountered by all clinicians because of the high prevalence of chronic hypertension.

A hypertensive urgency is defined as a BP >180/110 mmHg in a patient with severe headache with or without shortness of breath or oedema, but with no immediate life-threatening neurological, renal, ocular or cardiac complications such as are seen in hypertensive emergencies. Ideally, all patients with hypertensive urgency should be referred for in-hospital treatment, but in the public sector this not always possible. A hypertensive emergency exists when acute elevation of BP is associated with acute and ongoing organ damage to the kidneys, brain, heart, eyes (grade 3 or 4 retinopathy) or vascular system. These patients need rapid (within minutes to a few hours) lowering of BP to safe levels and referral for immediate hospitalisation, preferably in the intensive care setting, is mandatory.

**Suspected secondary cause**

At the first evaluation of a hypertensive patient the primary practitioner should consider the possibility of a secondary cause (Table II) established by history, examination, and a few basic investigations. For instance, unexplained hypokalaemia may suggest a diagnosis of primary aldosteronism. Sweats, palpitations, and weight loss, on the other hand, may suggest a phaeochromocytoma, or a renal artery bruit a renal artery stenosis. Secondary causes should be strongly considered in any young hypertensive patient, particularly in the absence of a family history of hypertension or obesity, and in patients with unexplained resistant hypertension (as discussed above).

**Table I. Reasons for referring a hypertensive patient to a specialist**

- Suspected white coat hypertension
- Resistant hypertension
- Hypertensive urgency or emergency
- Suspected secondary cause
- Associated clinical conditions
- Masked hypertension
- Intolerance of multiple antihypertensive drugs

**White coat or office hypertension**

White coat or office hypertension is defined as an elevated blood pressure (BP) in the office or clinic and a normal BP in familiar surroundings. The elevated BP is evident in the first 2 - 4 minutes, but varies considerably among patients. Office hypertension is generally not associated with an increased cardiovascular risk.

**Table II. Common identifiable causes of hypertension**

- Sleep apnoea
- Drug-induced
- Chronic kidney disease
- Primary aldosteronism
- Renovascular
- Cushing's syndrome
- Phaeochromocytoma
- Coarctation

**Associated clinical conditions**

The following are considered associated clinical conditions by the recent South African Hypertension Guideline: ischaemic heart disease, cardiac failure, stroke, transient ischaemic attack, grade 3 or 4 retinopathy (haemorrhages, exudates or papilloedema), peripheral vascular disease and chronic kidney disease. Chronic kidney disease is defined as overt albuminuria.
One of the most important obstacles to referral of hypertensive patients is that most primary care practitioners 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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**Masked or nocturnal hypertension**

Masked hypertension is defined as a normal BP in the office but an elevated BP on either home or ambulatory BP monitoring. This condition has only recently been recognised with the greater use of automated BP monitoring. In essence it is the reverse of white coat or office hypertension. Despite the normal office BP this condition is not benign and the ambulatory or home monitoring more accurately reflects the cardiovascular risk.

Nocturnal hypertension refers to the loss of night-time dipping of BP (non-dipper). Although the office or daytime BP is normal or relatively well controlled, the overall BP burden is increased. This also has been increasingly recognised with the use of 24-hour BP monitoring, and carries an increased cardiovascular risk.

Masked or nocturnal hypertension should be suspected in a patient with normal or controlled BP in the office and in whom there is TOD or progressive signs of TOD despite antihypertensive treatment. Specialist evaluation is strongly recommended.

**Intolerance to multiple antihypertensive drugs**

Occasionally patients develop side-effects to multiple antihypertensive drug classes, e.g. a cough caused by ACE inhibitors, oedema due to calcium-channel blockers, erectile dysfunction due to diuretics, or wheezing due to beta blockers. In these instances referral to a specialist is important to motivate for less commonly used and restricted drugs at the primary care level.

**Obstacles to referral of hypertensive patients**

One of the most important obstacles to appropriate referral is physician inertia. 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.

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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...
improvement 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.

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

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**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.
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.

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.

When to refer for vertigo (Fig. 6)

Benign paroxysmal positional vertigo (BPPV)

This condition is due to ooliths 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 dilation of the endolymphatic system. The management is salt restriction and diuretics but vestibular sedatives may give symptomatic relief. In severe cases the
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 dizzi-ness 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

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

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

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More about...
circulation vascular intervention. If this is not available the treatment is the control of vascular risk factors and antiplatelet therapy.

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.

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
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 smear 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

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.

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Drug resistance
Prevalence of isoniazid resistance in South Africa is about 10%. Multidrug resistance, which is defined as resistance to isoniazid and rifampicin with or without additional drugs, has a combined prevalence of between 2.5% and 4.5%. Although drug resistance should be suspected when a patient fails to respond, it is essentially a laboratory diagnosis. Currently the major problem in confirming the diagnosis of drug resistance is the fact that confirmation usually takes between 6 and 8 weeks. During this time the patient may deteriorate further and even die, as well as spread the disease further. The organism has to first be grown in liquid media in the MBGIT system and identified as Mycobacterium tuberculosis. Then drug susceptibility must be performed after sub-culturing the organism onto media impregnated with the antibiotic and the colony growth must be examined. Rapid tests are available to detect rifampicin resistance which is a marker for MDR TB. They include the PCR-based Hain test and the phage-based fast plaque test. Unfortunately at the time of writing these are not available as routine tests in the public health system, although they are available at private laboratories. With these systems MDR TB can be identified within 24 - 72 hours. This is a major advance since MDR TB is rapidly identified if suspected and appropriate treatment started, so preventing the patient from deteriorating and maybe even dying, as well as preventing further spread of the disease.

If TB has been proven and the patient is clearly deteriorating despite good compliance, then a strong case can be made for starting the patient on standardised MDR TB treatment while awaiting laboratory confirmation of drug resistance.

Inadequate drug levels
If sputum results show that the organism is fully susceptible, inadequate drug levels should be suspected. This may be due to malabsorption, accelerated metabolism or both. Tests to determine serum levels of rifampicin are available, although it is possible to establish levels of any drug. When there is complete susceptibility and deterioration (although ideally rifampicin levels taken 1 and 2 hours after ingestion can be requested), a case can be made for starting the patient on standardised MDR TB treatment in view of the fact that there may be 'clinical resistance'.

Vomiting
Failure to improve could be due to intolerance of the medication, particularly vomiting, which must be recognised and managed with anti-emetics. If all else fails, the offending drug should be discontinued and possibly substituted.

Malabsorption
While malabsorption of TB drugs is uncommon and usually seen in HIV-positive patients, low drug levels should be considered as a cause of non-improvement.

Coexistent disease
Advanced HIV/AIDS and diabetes can cause TB therapy to fail.

Poor-quality medication
While this is an unlikely factor, it has been known to occur in the past when medication is obtained from dubious sources.

Non-improvement on TB therapy can present a challenge. Consideration of the above causes should help to sort out the problem. However, there will be times where referral to a pulmonologist or infectious diseases specialist is warranted for further work-up and management, particularly if the diagnosis of TB may be wrong. A pulmonologist will review the case in relation to performing fibreoptic bronchoscopy to clarify the diagnosis. This may also help in establishing conditions coexistent with TB.

Drug-resistant TB, especially M(\x)DR TB, will also need specialist management from time to time despite the adoption of DOTS Plus therapy with the use of standardised therapy.

References