More about...When to refer

When to refer a patient with hypertension

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Hypertension is a common chronic disorder affecting 20 - 30% of adults in South Africa. Individuals who are normotensive at 55 years have a 95% lifetime risk of developing hypertension. It is therefore essential that the majority of patients should be treated by primary health care practitioners. Comprehensive hypertension guidelines have been developed for the management of hypertension in South Africa and the majority of patients should be able to receive appropriate treatment in the primary care setting. However, in certain circumstances it may be necessary for the hypertensive patient to be referred to a specialist for further evaluation and management. This article addresses the most important reasons for referral by a primary care practitioner. These are outlined in Table I.

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 at the beginning of the visit and may persist for 10 - 15 minutes or longer and is associated with an increased heart rate. On average the rise in BP may be as high as 27/14 mmHg in the first 2 - 4 minutes, but varies considerably among patients. Office 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 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

(albumin/creatinine ratio >30 mg/mmol) and a creatinine >133 µmol/l in males and ≥124 µmol/l in females. It is important to note that any hypertensive patient is automatically at high risk regardless of BP levels – the target BP should be <130/80 mmHg. Referral to a specialist for specific therapy to address these underlying conditions is often warranted. For example, a patient with chronic kidney disease may require both angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers for renal protection. Similarly, patients with stroke, ischaemic heart disease or heart failure may need further investigation and treatment of their neurological or cardiac disease by a specialist.

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.

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

Joint National Hypertension Guideline Working Group 2006. South African Hypertension Guideline. *S Afr Med J* 2006; 96: 337-362.

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

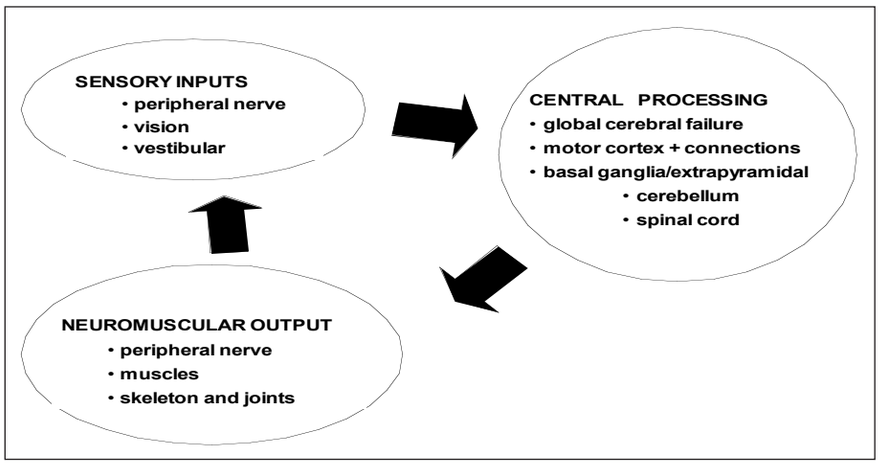

Fig. 1. Factors maintaining balance.

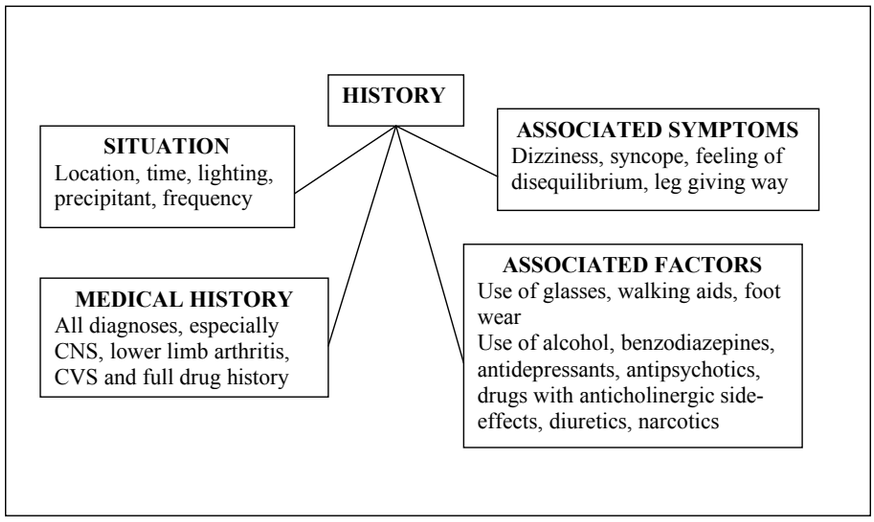

Fig. 2. Comprehensive assessment of falls.

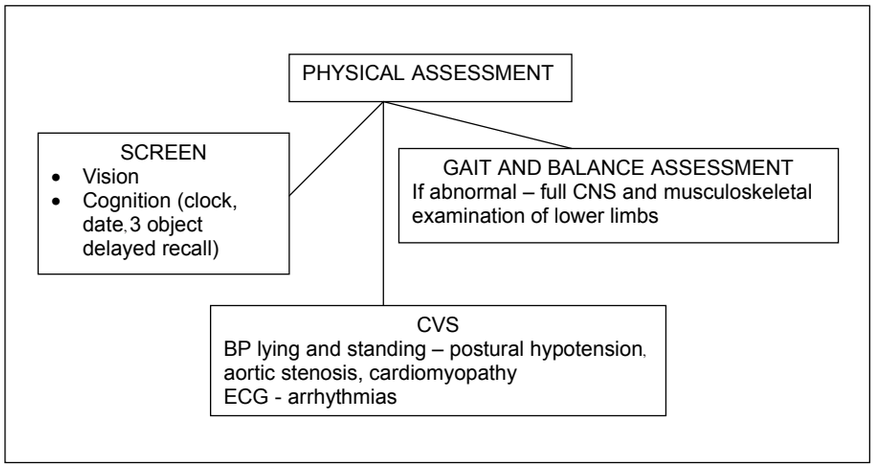

Fig. 3. Physical examination of patients presenting with a fall.

impairment 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).

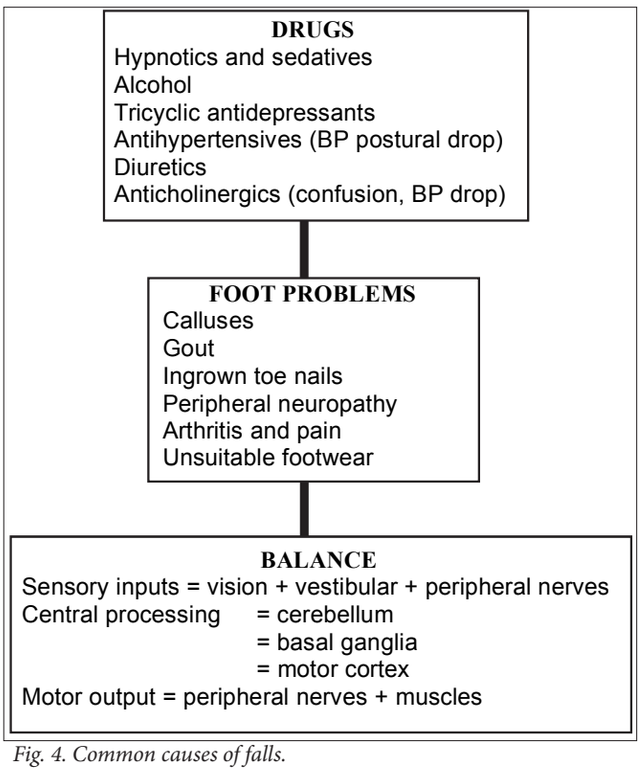

Fig. 4. Common causes of falls.

The history should also include the current functional status of the patient including both instrumental activities of daily living (IADLs) such as shopping, housekeeping, 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

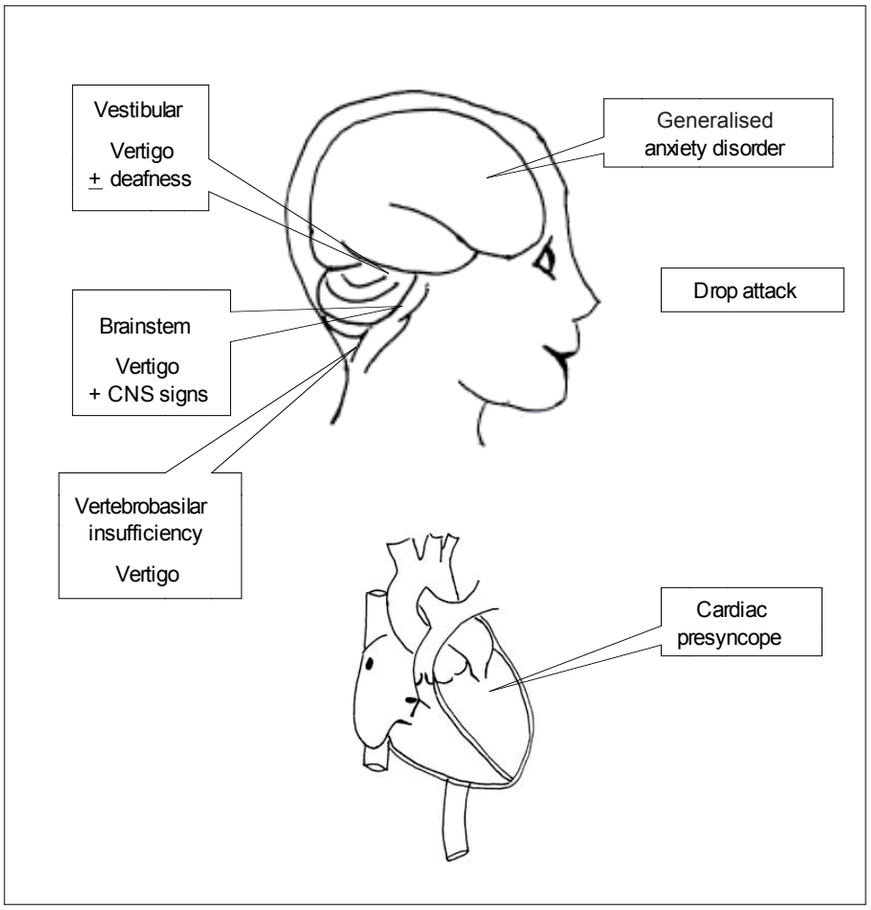

Fig. 5. Falls associated with dizziness.

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.

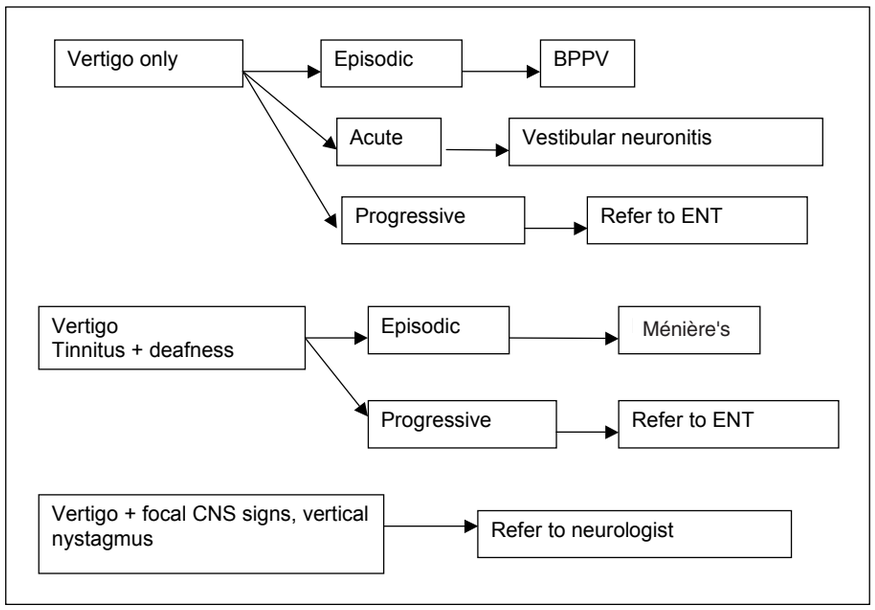

Fig. 6. Causes of vertigo.

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 dilation of the endolymphatic system. The management is salt restriction and diuretics but vestibular sedatives may give symptomatic relief. In severe cases the

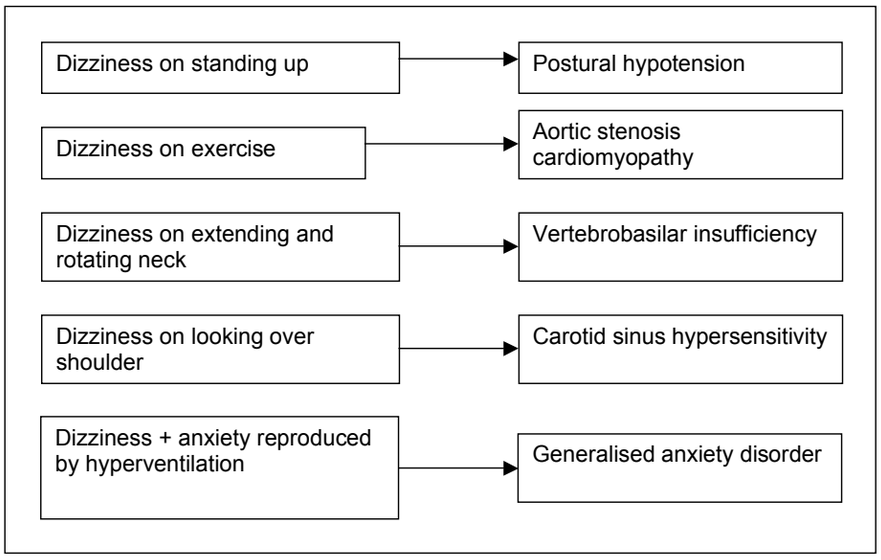

Fig. 7. Causes of dizziness.

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

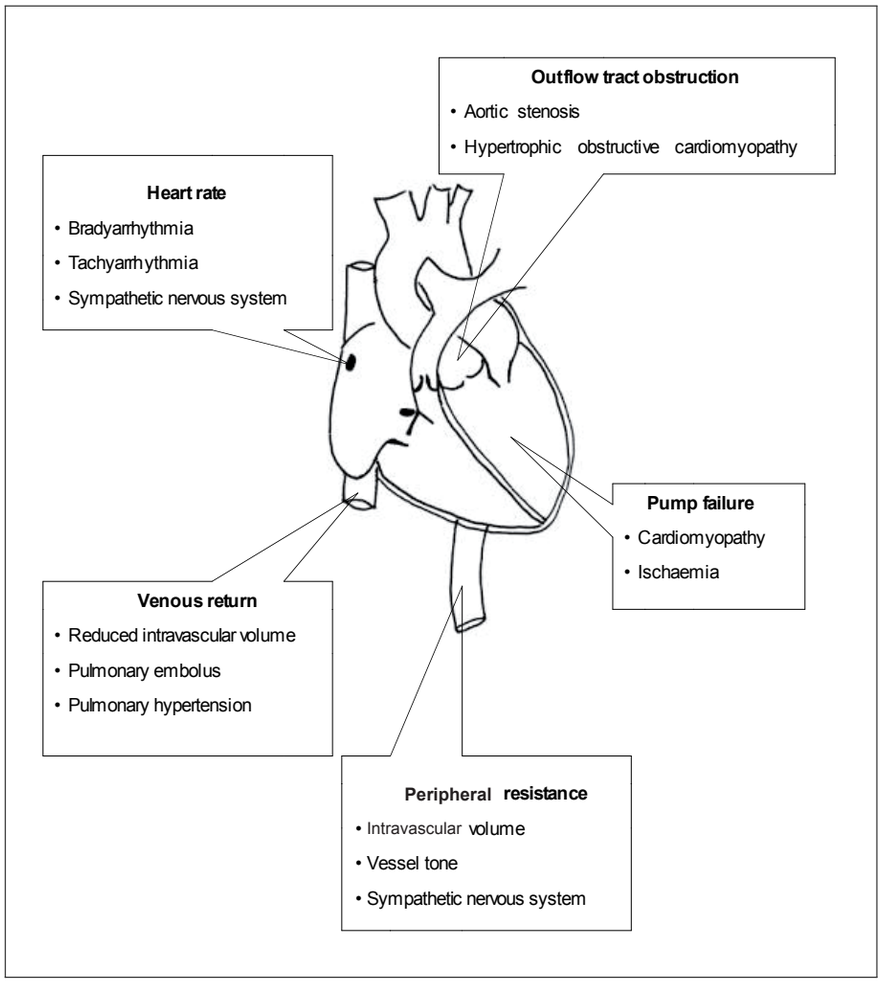

Fig. 8. Causes of cardiac syncope.

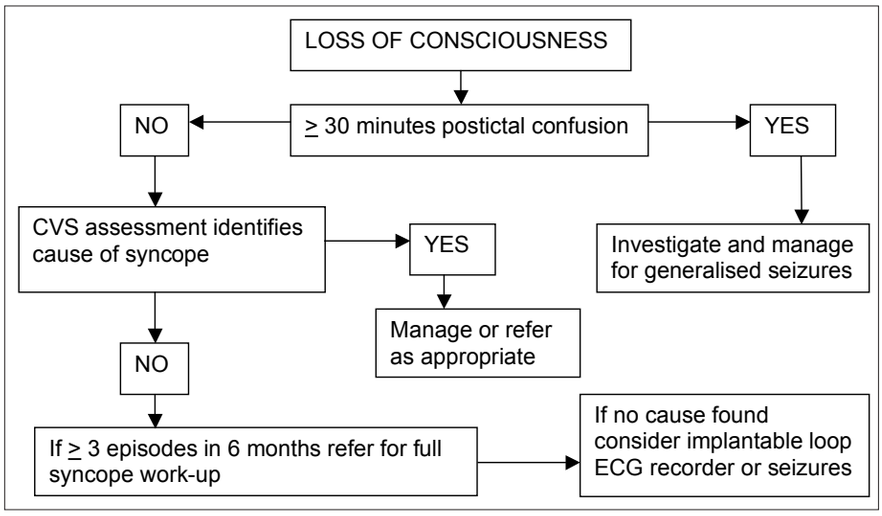

Fig. 9. Investigation of an episode of loss of consciousness.

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.

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.

Falls associated with loss of consciousness

When the history is one of true loss of consciousness the differential diagnosis is syncope or generalised seizure. The presence of jerking movements, injuries and incontinence is not specific for seizure disorders. The most useful characteristic in distinguishing a seizure from cardiac syncope is that full consciousness is restored in less than 5 minutes with syncope, but generalised seizures are followed by at least 30 minutes of postictal confusion and drowsiness.

Cardiac syncope is due to a temporary reduction in cerebral perfusion. The problem can be either a reduction in cardiac output (stroke volume or heart rate) or a reduction in peripheral vascular resistance. The CVS examination always includes a supine and erect blood pressure and an ECG.

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

cure rates of 98% occur under trial conditions, chemotherapy given under programmatic conditions results in cure rates of only about 70%. This is far short of the WHO's criteria for a successful TB control programme of 85%. The major reason for this is interruption of therapy, with rates as high as 33%.

Failure to improve on TB therapy is problematic. There are a number of reasons for this, and they include the following:

- wrong diagnosis
- coexistent conditions with TB
- poor compliance with therapy
- drug resistance
- vomiting
- malabsorption
- inadequate drug levels
- poor quality TB medication.

Wrong diagnosis

Patients are often commenced on TB treatment without a positive direct smear or with just a single scanty positive smear. However, many conditions mimic active TB, including fungal infections such as cryptococcosis, subacute and chronic pneumonia including necrotising pneumonia and pneumocystis pneumonia. Various non-infective conditions also mimic TB, including malignancies such as carcinoma of the lung, lymphoma and Kaposi's sarcoma as well as sarcoidosis. In addition, post-destructive tuberculous lung disease with cavitation and mycetoma formation may be mistaken for active TB.

When any patient is commenced on empiric therapy without bacteriological proof, close follow-up is important and if the disease does not respond appropriately then it should be strongly suspected that the condition is not TB. If smears were initially positive and there is failure to improve appropriately then there could be one of the above coexistent conditions.

It's important to realise that occasionally the initial diagnosis may be spurious. False-positive smears, but more commonly false-positive cultures, can occur and are due to faulty sputum collection or laboratory contamination. The latter occurs in at least 3% of cases.[2]

Poor compliance

When the TB has initially been confirmed and the diagnosis is not in doubt and there is failure to improve, it is essential as a first step to check compliance by questioning the patient and attending relatives, examining the clinic card the patient carries and if necessary making enquiries at the TB clinic. If compliance is found to be an issue appropriate steps must be taken, having established the reason for the poor compliance.

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.

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