Symposium: Clinical Practice

Syncope-related falls in the elderly

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Abstract Age-related physiological impairments of heart rate, blood pressure and cerebral blood flow, in combination with comorbid conditions and concurrent medications, account for an increased susceptibility to syncope in older adults. Common causes of syncope are orthostatic hypotension, neurally-mediated syncope (including carotid sinus syndrome) and cardiac arrhythmias. A high proportion of older patients with cardiovascular syncope present with falls and deny loss of consciousness. Patients who are cognitively normal and have unexplained falls should have a detailed cardiovascular assessment. (J Geriatr Cardiol 2005; 2 (2): 74-83).

Key Words syncope; falls; elderly

Introduction

Syncope is a symptom defined as a transient, self-limited loss of consciousness, usually leading to a fall. The onset of syncope is usually rapid, and the subsequent recovery is spontaneous, complete and prompt.1 Individuals who describe syncope will often experience more frequent episodes of presyncope. Presyncope or near-syncope refers to a condition when the patient feels as though syncope is imminent. Symptoms may be non-specific (e.g. dizziness) and tend to overlap with those of the premonitory phase of true syncope.

Epidemiology of syncope

The epidemiology of syncope in old age has not been well studied. The greatest difficulty in assessing the magnitude of the problem is that it is only recently acknowledged that there is considerable overlap between syncope and falls. This is likely to have led to a significant under-estimate of the frequency of syncope in the elderly. From available data, the incidence of syncope is at least 6% per year, with a 10% prevalence and a 30% two-year recurrence rate.2

Pathophysiology of syncope in the elderly

The temporary cessation of cerebral function that causes syncope is due to transient and sudden reduction of cerebral blood flow to areas of the brain responsible for consciousness (reticular activating system). Cerebral perfusion pressure is largely dependent on systemic arterial pressure. Any factor that either decreases cardiac output or total peripheral vascular resistance diminishes systemic arterial pressure and cerebral perfusion.3

Age-related physiological impairments of heart rate, blood pressure (BP) and cerebral blood flow, in combination with co-morbid conditions and concurrent medications, account for an increased susceptibility to syncope in older adults. Baroreflex sensitivity is blunted by aging, manifesting as a reduction in heart rate response to hypotensive stimuli.4 The elderly are prone to reduced blood volume due to excessive salt wasting by the kidneys as a result of diminished renin-aldosterone activity,5 a rise in atrial natriuretic peptide6 and concurrent diuretic therapy. Low blood volume together with age-related diastolic dysfunction can lead to a low cardiac output, which increases susceptibility toorthostatic hypotension (OH) and neurally-mediated syncope. Cerebral autoregulation, which maintains a constant cerebral perfusion pressure over a wide range of systemic BP changes, is altered in the presence of hypertension and possibly by aging.5,7 As a result, sudden mild to moderate declines in BP which would not cause any embarrassment of cerebral perfusion in a younger individual can markedly effect perfusion pressures in the elderly adult and leave them very vulnerable to presyncope or syncope.

Etiology of syncope

Table 1 provides a pathophysiological classification of the commoner causes of transient loss of consciousness. The commonest causes of syncope in the elderly are OH, neurally-mediated syncope and cardiac arrhythmias.6,8,9 OH is an attributable cause of syncope, including carotid sinus syndrome, in 20-30% of events.5,10 Carotid sinus hypersensitivity is rare before the age of 40. The prevalence increases with age and with cardiovascular, cerebrovascular and neurodegenerative co-morbidity.11-13 Cardioinhibitory carotid sinus syndrome has been recognised as an attributable cause of syncope in elderly in up to 20-30% of cases.8,14 Vasodepressor carotid sinus syndrome is likely to be equally prevalent.5,10 Up to 15% of syncope in the elderly is neurally-mediated while 20% is due to cardiac arrhythmias.8,9
Neurally-mediated reflex syncopal syndromes*  
Neurocardiogenic / vasovagal syncope (common faint)  
Carotid sinus syndrome  
Situation syncope  
gastrointestinal stimulation (swallow, defecation, visceral pain) micturition (post-micturition)  
cough  
glossopharyngeal and trigeminal neuralgia  
Orthostatic  
Autonomic failure  
primary autonomic failure syndromes  
secondary autonomic failure syndromes  
drugs and alcohol  
Volume depletion  
Cardiac arrhythmia as primary cause  
Sinus node dysfunction (including tachycardia/bradycardia syndrome)  
Atrioventricular conduction system disease  
Paroxysmal supraventricular and ventricular tachycardias  
Inherited syndromes (eg long QT syndrome, Brugada syndrome)  
Implanted device (pacemaker) dysfunction  
Structural cardiac or cardiopulmonary disease  
Cardiac valvular disease  
Acute myocardial infarction/ischæmia  
Obstructive cardiomyopathy  
Atrial myxoma/Acute aortic dissection  
Pericardial disease or tamponade  
Cerebrovascular  
Vascular steal syndromes  

* Neurally-mediated reflex syncope syndrome refers to the Bezold-Jarisch reflex that when triggered gives rise to inappropriate vasodilatation and bradycardia  
1 Orthostatic syncope occurs when the autonomic nervous system is incapacitated, resulting in failure of vasoconstrictor mechanisms thereby resulting in hypotension  
2 Cardiac arrhythmias resulting in reduced cardiac output  
3 Structural heart disease can cause syncope when circulatory demands outstrip the hearts ability to increase its output  
4 Steal syndromes rarely can cause syncope when a blood vessel has to supply both part of the brain and an arm  

Overlap of falls and syncope  
The traditional view of syncope and falls is to regard them as separate conditions with different etiologies. It has been suggested that an accurate history of the event from the patient and an available witness will allow the differentiation between syncope and falls and will point to the diagnosis in over 40% of cases. This approach is problematic in elderly for a variety of reasons. Up to one-third of cognitively normal elderly have amnesia for witnessed loss of consciousness. Similarly over one-third of community-dwelling elderly did not recall having fallen three months after a documented fall event. In addition, witness accounts of syncopal events are only available in 40-60% of older people attending a syncope clinic with recurrent symptoms. In patients with cognitive impairment, the differentiation between falls and syncope is likely to be even more difficult. Experience from dedicated syncope and falls facilities would reinforce the evidence of an overlap between these syndromes. A review of three studies, which included a total of 109 patients for whom cardioinhibitory carotid sinus syndrome was an attributable cause of falls, 38% of patients presented with falls alone or falls and dizziness but denied syncope. Of the fallers, 51% demonstrated amnesia for loss of consciousness during diagnostic testing. Two-thirds of patients with OH as an attributable cause of symptoms presented with falls only, or falls and dizziness. In a further case series of 169 patients who attended a dedicated syncope and falls clinic, over one-third presented with a history of unexplained falls, two-thirds of these had an attributable cardiovascular diagnosis, of which the majority were carotid sinus syndrome, OH, cardiac arrhythmias and neurally-mediated syncope.

More recently there is emerging evidence from fall intervention studies that treatment of identified attributable cardiovascular causes of syncope and falls resulted in reduced subsequent frequency of falling. Similarly, preliminary data from SAFE-COG, a study of multifactorial intervention strategies in cognitively impaired fallers) suggests that treatment of cardiovascular abnormalities may reduce fall frequency in patients with dementia living in the community. Another possible explanation for the overlap between syncope and falls is that moderate haemodynamic changes insufficient to cause true syncope may result in falls in individuals already compromised by gait and balance instability and slow protective reflexes. It seems that syncope in elderly and falls are often indistinguishable and in fact are manifestations of similar pathophysiological processes.

Carotid sinus syndrome  
Carotid sinus syndrome (CSS) is diagnosed when carotid sinus hypersensitivity is documented in a patient with otherwise unexplained dizziness, falls, presyncope or syncope, in whom carotid sinus massage reproduces presenting symptoms. The cardioinhibitory subtype is diagnosed if carotid sinus massage produces asystole exceeding 3 seconds, the vasodepressor subtype if there is a fall in systolic blood pressure (SBP) exceeding 50 mmHg in the absence of significant bradycardia and a mixed subtype if both are present. Carotid sinus massage is a crude and unquantifiable technique prone to both intra and inter-operator variation. Treatment strategies depend on the clinical subtype:

1. Cardioinhibitory carotid sinus syndrome - Treatment of choice is a permanent pacemaker. Recurrent syncope caused by CSS, producing greater than 3 seconds of asystole in the absence of medication that depresses the sinoatrial or the atrioventricular conduction, is a class I indication for pacemaker implantation. Recurrent syncope without clear provocative events and with a hypersensitive cardioinhibitory response is a class IIa indication according to ACC/AHA guidelines. Dual-chamber pacing significantly reduces syncope, falls, injury and hospital admissions.

2. Vasodepressor carotid sinus syndrome - Management of vasodepressor CSS is much more difficult. Dihydroergotamine and fludrocortisone have been used with limited effect. Side effects of these medications in the elderly
Orthostatic hypotension

Orthostatic hypotension is a reduction in SBP by at least 20mmHg or diastolic BP of at least 10mmHg within 3 minutes of standing.

The prevalence of OH varies between 4-33% among community-dwelling elderly. The prevalence and magnitude of falls in SBP increase with age and are associated with general frailty and excessive mortality.

Several pathological conditions are associated with OH. Stout and Kenny describe a consecutive series of 70 patients with OH. The results are shown in Table 2. Drug-induced OH is common. Table 3 outlines likely culprit medication.

Table 2. Etiology of orthostatic hypotension in older adults referred to a regional syncope service

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Prevalence (%)</th>
</tr>
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<tbody>
<tr>
<td>Drug induced</td>
<td>28</td>
</tr>
<tr>
<td>Autonomic failure</td>
<td>27</td>
</tr>
<tr>
<td>Age-related</td>
<td>20</td>
</tr>
<tr>
<td>Multiple system atrophy</td>
<td>13</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>5</td>
</tr>
<tr>
<td>Unclassified</td>
<td>2</td>
</tr>
</tbody>
</table>


A number of non-neurogenic conditions are also associated with, or exacerbate, pre-existing OH. They include haemorrhage, diarrhoea, vomiting, burns, haemodialysis, diabetes insipidus, adrenal insufficiency, fever and extensive varicose veins.

Diagnosis of OH involves demonstration of a postural fall in BP after standing. Reproducibility of OH depends on the time of measurement and on autonomic function. The measurement should be carried out as early in the morning as is practical after maintaining a supine posture for at least 10 minutes. Phasic beat-to-beat BP measurements are more sensitive than sphygmomanometer measurements.

Treatments to deduce the effects of venous pooling are particularly useful in OH. These include physical maneuvers (compression stockings and calf exercises) and vasoconstriction (adrenergic agonist midodrine). Increasing circulating blood volume, by increased salt and fluid intake and by the use of fludrocortisone, reduces the detrimental effects of venous pooling. Desmopressin has been used in resistant cases of OH for its anti-diuretic and mild pressor effects. As drugs are commonly indicated in the causation of OH, it is paramount that all efforts be made to ensure rational drug prescribing, minimising all non-essential therapy.

Hypertension and orthostatic hypotension

Aging is associated with an increased risk of hypertension. Hypertension is known to impair baroreflex-sensitivity and restricts ventricular filling. Paradoxically hypertension increases the risk of episodic hypotension. A strong relationship between supine hypertension and OH has been reported among unmedicated, institutionalised elderly subjects. Hypertension also alters the thresholds at which cerebral autoregulation occurs. Older subjects with

Table 3. Commonly used drugs causing orthostatic hypotension

<table>
<thead>
<tr>
<th>Class of drug</th>
<th>Mechanism of orthostatic hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antihypertensives</strong></td>
<td></td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>Inhibition of angiotensin II production</td>
</tr>
<tr>
<td>Adrenergic nerve blocking agents</td>
<td>α-Adrenergic blockade</td>
</tr>
<tr>
<td>α-Adrenoceptor antagonists</td>
<td>α-Adrenergic blockade</td>
</tr>
<tr>
<td><strong>Coronary vasodilators</strong></td>
<td></td>
</tr>
<tr>
<td>Nitrates</td>
<td>Vasodilatation</td>
</tr>
<tr>
<td><strong>Diuretics</strong></td>
<td></td>
</tr>
<tr>
<td>Thiazide or loop</td>
<td>Volume depletion</td>
</tr>
<tr>
<td><strong>Psychoactive drugs</strong></td>
<td></td>
</tr>
<tr>
<td>Phenothiazines/Tricyclic antidepressants</td>
<td>α-Adrenergic blockade/α-Adrenergic blockade</td>
</tr>
<tr>
<td><strong>Anti-Parkinsonian drugs</strong></td>
<td></td>
</tr>
<tr>
<td>Levodopa/Bromocriptine</td>
<td>Not clearly understood Activate vascular dopaminergic receptors</td>
</tr>
<tr>
<td><strong>CNS depressants</strong></td>
<td></td>
</tr>
<tr>
<td>Ethanol</td>
<td>Vasodilatation</td>
</tr>
</tbody>
</table>
hypothesis are thus more likely to experience intermittent hypotension and be less able to compensate for its effects, with the result that they are exposed to cerebral ischaemia. In addition, with increasingly aggressive BP control targets, patients are likely to be taking two or three antihypertensive medications which all impair cardiovascular reflexes and further increase the risk of OH.

Neurally-mediated syncope

Neurally-mediated syncope or vasovagal syncope is the simplest faint. It was previously believed that it was a rare phenomenon in elderly but more recently it has been estimated to be the cause of 20% of recurrent syncope. In elderly referred to a syncope clinic over a 6 month period, 11% had neurally-mediated syncope which commonly co-existed with CSS and OH. In unselected elderly patients presenting to an Accident and Emergency department with unexplained or recurrent falls, neurally-mediated syncope was present in 16-18%. In those with recurrent neurally-mediated syncope, treatment should be aimed initially at withdrawal of any culprit medications. In elderly patients with serious co-morbidity this may not be possible. An explanation of the cause of the syncope, along with clear guidelines on avoidance techniques may be all that is required. Various medications have been tried in the treatment of resistant neurally-mediated syncope, each directed at various points in the abnormal reflex. Midodrine may reduce symptom frequency by preventing peripheral vasoconstriction and decreased baroreceptor sensitivity. Fludrocortisone will increase plasma volume but is poorly tolerated in the elderly. Trials of the use of beta adrenergic antagonists have shown some benefit in uncontrolled trials but these effects have not been demonstrated in long-term follow-up studies and are not currently recommended for neurally-mediated syncope.

Postprandial hypotension

In healthy elderly, SBP falls by 11-16mmHg and heart rate rises by 5-7 beats per minute of eating. In the majority of fit, as well as frail elderly, most hypotensive episodes go unnoticed. In patients with hypertension, OH, and autonomic failure, the postprandial BP fall is much greater and occurs without a corresponding heart rate rise.

Dietary adjustments may be sufficient to manage postprandial hypotension. Simple carbohydrates should be replaced by complex carbohydrate and smaller, more frequent, meals should be substituted. Additional options include treatment with a non-steroidal anti-inflammatory agent such as indomethacin, the somatostatin analogue octreotide or caffeine. Caffeine, taken along with food, prevents hypotensive symptoms in fit as well as frail elderly.

Cardiac disease-related syncope

Table 1 lists cardiac diseases which commonly present with syncope.

Arhythmias causing syncope

Bradyarrhythmias and tachyarrhythmias may cause a sudden decrease in cardiac output and cause sudden syncope. Initially bradyarrhythmias may be partially compensated for by prolonged ventricular filling, resulting in increased stroke volume and maintained cardiac output. As the heart rate slows further, this compensatory mechanism is overwhelmed and cardiac output falls and presyncope or syncope occurs. Similarly, mild to moderate tachycardias increase cardiac output while faster heart rates result in decreased ventricular diastolic filling, reduced cardiac output, hypotension and syncope. It has also been recognised that supraventricular and paroxysmal atrial fibrillation may activate cardiac mechanoreceptors and induce neurally-mediated syncope - rapid, vigorous, ventricular contraction in the setting of a relatively empty ventricle.

Physiological impairments associated with aging, the effects of multiple medications and co-morbidity may predispose elderly to syncope even in the setting of brief arrhythmias, while similar arrhythmias may not produce any symptoms in a younger individual.

Arrhythmias cause approximately 20% of syncopeal episodes in elderly. Sick sinus syndrome and ventricular tachycardias are the most common arrhythmic causes identified. Syncope is a central manifestation of sick sinus syndrome, being reported in 25-70% of patients. Electrocardiographic features include sinus bradycardia, atrial pauses, arrest or exit block. When bradyarrhythmias are associated with episodic supraventricular tachycardias, tachycardia/bradycardia syndrome is said to be present. Ventricular tachycardias generally occur in the setting of known organic heart disease, especially in those with poor left ventricular systolic function. The severity of associated symptoms is related to the rate, the duration and myocardial pump function. Torsades de Pointe and syncope in elderly occurs in those with the acquired Long QT syndrome. Acquired Long QT syndromes are often secondary to electrolyte disturbance or anti-arrhythmic drugs such as amiodarone, procainamide, disopyramide and flecainide.

Structural cardiac and cardiopulmonary disease causing syncope

Syncope with exertion is commonly a manifestation of organic heart disease in which cardiac output is fixed. Syncope is reported in up to 42% of patients with severe aortic stenosis, commonly with exercise. A number of possible mechanisms of syncope have been proposed: 1) neurally-mediated via cardiac mechanoreceptor stimulation, and 2) inability to increase cardiac output in response to exercise because of severe left ventricular outflow tract obstruction, coupled with
Table 4. Clinical features suggestive of specific causes of real or apparent loss of consciousness

<table>
<thead>
<tr>
<th>Symptom or finding</th>
<th>Possible cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>After sudden unexpected, unpleasant sight, sound or smell</td>
<td>Neurally-mediated</td>
</tr>
<tr>
<td>Prolonged standing or crowded, warm place</td>
<td>Neurally-mediated or autonomic failure</td>
</tr>
<tr>
<td>Nausea or vomiting associated with syncope</td>
<td>Neurally-mediated</td>
</tr>
<tr>
<td>After meals</td>
<td>Postprandial</td>
</tr>
<tr>
<td>After exertion</td>
<td>Neurally-mediated or autonomic failure</td>
</tr>
<tr>
<td>With head rotation, pressure on carotid sinus</td>
<td>Carotid sinus syndrome</td>
</tr>
<tr>
<td>Within seconds or minutes of standing</td>
<td>Orthostatic hypotension</td>
</tr>
<tr>
<td>Temporal association with start or change of dose of medication</td>
<td>Drug-induced</td>
</tr>
<tr>
<td>During exertion or when supine</td>
<td>Cardiac</td>
</tr>
<tr>
<td>Preceded by palpitation</td>
<td>Tachyarrhythmia</td>
</tr>
<tr>
<td>Family history of sudden death</td>
<td>Long QT syndrome, Brugada syndrome, HCM</td>
</tr>
<tr>
<td>Associated with vertigo, dysarthria, diplopia</td>
<td>Brainstem ischaemia</td>
</tr>
<tr>
<td>With arm exercise</td>
<td>Subclavian steal</td>
</tr>
<tr>
<td>Confusion after attack lasting more than 5 minutes</td>
<td>Seizure</td>
</tr>
<tr>
<td>Tonic-clonic movements, automatism, blue face</td>
<td>Seizure</td>
</tr>
<tr>
<td>Frequent attacks associated with somatic symptoms and no organic heart disease</td>
<td>Psychiatric illness</td>
</tr>
</tbody>
</table>

Table 5. Important historical feature of possible syncope

Circumstances just prior to attack
- Position (supine, sitting, standing)
- Activity (rest, change in posture, exercise associated, micturition, cough, swallowing, defecating)
- Predisposing factors (crowds, warm places, prolonged standing, fear, pain, neck movement)

Onset of attack
- Nausea, vomiting, feeling cold, sweating, pain in neck or shoulders, blurred vision

Attack (witness)
- Way of falling (slumping over)
- Colour (pallor, cyanosis)
- Duration of loss of consciousness
- Movements and duration (tonic, clonic, tonic-clonic, myoclonic, automatisms)

End of attack
- Nausea, vomiting, sweating, confusion, muscle aches, skin colour, injury, chest pain, incontinence

Background
- Family history of sudden death
- Previous cardiac disease
- Neurological history (Parkinson’s disease, epilepsy, narcolepsy)
- Metabolic disorders
- Medication (vasoactive drugs, anti-arrhythmics, diuretics, QT prolonging agents)
- Frequency of episodes
- Driving history
Peripheral vasodilatation associated with exercise resulting in hypotension and syncope.

Syncope is an important prognostic indicator in aortic stenosis. In the absence of valve replacement the average survival is 2-3 years. Exertional syncope is also commonly associated with hypertrophic cardiomyopathy. The mechanism of syncope is similar to that of aortic stenosis. In addition, approximately 25% of subjects with hypertrophic cardiomyopathy have ventricular tachycardias, which may induce syncope.46

Elderly with acute myocardial infarction may present atypically. Five to 12% may present with syncope. Various mechanisms may cause this syncope such as rhythm disturbance or acute pump failure leading to hypotension (e.g. extensive infarction, papillary muscle rupture and interventricular septal rupture). Rare abnormalities such as atrial myxoma, pericardial disease and tamponade or aortic dissection may present with syncope. Pulmonary embolism should also be considered as a cause of syncope.

Diagnostic evaluation of syncope in elderly

A major issue in the use of diagnostic tests is that syncope is a transient symptom. Typically, patients are asymptomatic at the time of assessment and the opportunity to capture a spontaneous event during diagnostic testing is rare. Multiple illness are common in elderly; subjects over the age of 65 years have an average of 3.5 illnesses.47 It is important to carefully attribute a diagnosis rather than assume that the presence of an abnormality known to produce syncope or hypotensive symptoms is the causative factor. In order to attribute a diagnosis with certainty, patients must have symptom reproduction during investigation and preferably alleviation of symptoms, presyncope and syncope.

The most important elements in the evaluation of syncope in elderly are to establish whether the patient has actually experienced syncope and to select the appropriate cardiovascular investigations to define the cause. Table 4 shows conditions which may be confused with syncope.

History and physical examination

A detailed history and physical examination can result in a diagnosis in up to 40% of cases.8 An accurate witness account should always be sought but this is likely to be available in only 40% of elderly patients.8 Important elements of the history are shown in Table 5. These should be key features in the diagnostic work-up. Clinical features suggestive of specific causes of syncope are shown in Table 5.

Dizziness is a frequent accompanying symptom in subjects who have unexplained falls, presyncope and syncope. Clinical features of dizziness can further help to identify an underlying cause of symptoms. Four types of dizzy symptom have been recognised - vertigo, disequilibrium, lightheadedness and non-specific.49 Lightheadedness is often associated with an underlying cardiovascular cause of symptoms, vertigo with peripheral vestibular or central lesions and unsteadiness or disequilibrium with central degenerative disease.50 In addition, dizziness is most likely to be attributed to a cardiovascular cause if there is associated pallor, syncope, history of prolonged standing or a feeling of the need to sit or lie down when symptoms occur.50 Physical examination is used to diagnose specific entities and exclude others. Orthostatic BP recording, cardiovascular findings and neurological examination are crucial in this regard. Assessment of gait, mobility, muscle strength and the use of walking aids are important in patients complaining of unexplained falls and possible syncope. Assessment of vision is also important.

Orthostatic blood pressure measurement

Supine BP measurements should be taken after a minimum of 10 minutes at rest. BP should be recorded for up to 3 minutes while standing unaided. Measurements may be continued longer if BP is still falling after 3 minutes. A decrease in SBP of greater than 20mmHg, a 10mmHg fall in diastolic BP, or a fall in the SBP to 90mmHg or less is considered diagnostic of OH, regardless of whether or not symptoms occur. In patients with unexplained syncope or falls an attributable diagnosis of OH depends on symptom reproduction.

Baseline electrocardiogram

An abnormal ECG may be found in up to 50% of patients presenting with syncope.51 When abnormal, the ECG may disclose an arrhythmia associated with syncope (2-11% of patients), or more commonly an abnormality that may predispose to arrhythmia development and syncope.51 An abnormality of the baseline ECG is an independent predictor of cardiac syncope and is associated with increased mortality. Equally important, a normal ECG is associated with a low risk of cardiac syncope.

Prolonged electrocardiographic monitoring and electrophysiological testing

An arrhythmic cause of syncope is strongly suspected, the main tools of investigation are prolonged ambulatory electrocardiograms, patient activated recorders, implantable loop recorders and electrophysiological studies. Diagnostic yield from 24 hour Holter monitoring is low, only 4% of patients have correlation of symptoms and arrhythmia. Extending monitoring to 72 hours does not significantly improve diagnostic yield. Patient activated external loop recorders have a higher diagnostic yield compared with prolonged ambulatory monitoring but do not have good rhythm-symptom correlation in more than a third of subjects.52 Older patients must have sufficient manual dexterity and cognitive function to use these systems. Prospective recording is useful in those with pre-syncope, but of no benefit in patients with sudden collapse who do not have warning to enable them to activate the recorder. Continuous external loop recorders are more useful in this situation. These recorders will save a variable period prior to activation, as well as a short period of post event recording. Implantable loop recorders offer
extended ECG monitoring over a 18 month period. Using an activation device the patient, a family member or carer 'freezes' the loop during or after a typical syncopal episode thus storing the preceding segment, which can be retrieved later using a standard pacemaker programmer. Initial experience using an implantable loop recorder in subjects over age 60 years with suspected arrhythmic causes of syncope suggest that it may prove a valuable diagnostic tool.\textsuperscript{53}

**Ambulatory blood pressure monitoring**

Ambulatory BP monitoring is predominantly used in the management of hypertension, it can however play a role in the diagnosis and management of hypotensive disorders. Information such as the pattern of diurnal BP behaviour, postprandial dips in BP, and BP changes after medication are useful in patients suffering from syncope and dizziness. A reversal of the diurnal BP pattern is frequently observed in subjects with symptomatic OH.\textsuperscript{54}

**Carotid sinus massage**

The subject should lie supine on a footplate-type tilt table for a minimum of 5 minutes with continuous surface ECG and BP monitoring (where possible phasic beat-to-beat monitoring is preferred as the BP nadir in response to carotid sinus massage occurs around 18 seconds and returns to baseline at 30 seconds). Firm, longitudinal massage should be performed for 5 seconds at the site of maximal pulsation at the right carotid sinus, located between the superior border of the thyroid cartilage and the angle of the mandible.\textsuperscript{55} Massage is repeated over the left carotid sinus supine and then repeated right and left erect (70° head-up tilt). Only performing carotid sinus massage in the supine position will fail to demonstrate an abnormal response in 20-40% of those with CSS.\textsuperscript{56} Table 6 shows contra-indications to carotid sinus massage.

<table>
<thead>
<tr>
<th>Table 6. Contraindications to carotid sinus massage</th>
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<tbody>
<tr>
<td><strong>Absolute</strong></td>
</tr>
<tr>
<td>Myocardial infarction within 3 months</td>
</tr>
<tr>
<td>Transient ischemic accident within 3 months</td>
</tr>
<tr>
<td>Stroke within 3 months</td>
</tr>
<tr>
<td><strong>Relative</strong></td>
</tr>
<tr>
<td>Previous ventricular fibrillation</td>
</tr>
<tr>
<td>Previous ventricular tachycardia</td>
</tr>
<tr>
<td>Presence of carotid bruit*</td>
</tr>
</tbody>
</table>

* Known to be a poor predictor of presence or severity of carotid artery stenosis; carotid Doppler studies may help to stratify risk of procedure

Adherence to these exclusion criteria result in a very low complication rates: two persistent neurological defects and nine transient ischaemic events per 16,000 episodes of carotid sinus massage.\textsuperscript{57}

**Head-up tilt table testing**

Head-up tilt table testing has been used to investigate the pathophysiology of orthostatic stress for over 50 years but it was not until the 1980’s that its clinical utility in the diagnosis of unexplained syncope was recognised.\textsuperscript{58} Kenny found that 67% of patients with otherwise unexplained syncope demonstrated a neurally-mediated reaction during head-up tilting, compared with only 10% of healthy controls. Head-up tilt testing has since evolved into the diagnostic test of choice in assessing neurally-mediated and related disorders. Many authors have proposed different protocols for diagnostic, investigative and therapeutic purposes. Tilt angles between 60-80° are optimal in creating sufficient orthostatic stress without increasing the number of false positives or negative tests.\textsuperscript{33,59,60} The duration of the test also varies between centres, but it is suggested that prolonged tilting for 30-45 minutes is optimal.\textsuperscript{60} Longer periods of tilting produce unacceptably high proportions of false positive results. Our own practice is to use a 70° tilt angle for 40 minutes.

**Conditions where tilt table testing is warranted**

- Evaluating elderly with recurrent unexplained falls
- The evaluation of recurrent syncope or a single syncopal event in a high risk patient (syncope resulted in injury or determination of cause has significant occupational consequences) whether or not the history is suggestive of neurally-mediated syncope and
- No evidence of structural cardiovascular disease, or
- Structural cardiovascular disease is present, but other causes of syncope have been excluded by appropriate testing.
- Further evaluation of patients in whom an apparent cause has been established (e.g. asystole, atrioventricular block) but in whom demonstration of susceptibility to neurally-mediated syncope could alter treatment choice.
- Part of evaluation of exercise associated syncope

Relative contraindications to tilt testing include proximal coronary stenosis, critical mitral stenosis, clinically severe left ventricular outflow obstruction and severe known cerebrovascular disease. The traditional classifications of vasovagal response are detailed in Table 7.

**Equipment, monitoring and environment**

To minimize stimuli affecting autonomic function, the test should be carried out in a quiet, dimly lit room at a comfortable temperature and should be as non-threatening as possible. Patients should fast for two hours prior to the procedure (in order to minimize the confounding effects of postprandial hypotension) and then rest supine for 20 minutes. Longer periods may be necessary if intravenous instrumentation is used because of the risk of false positive tests.\textsuperscript{61} Drugs affecting cardiovascular or autonomic function should be discontinued 5 half-lives pre-test, unless they are etiologically implicated. During the test the patient is instructed to avoid movements of the lower extremities to maximize venous pooling. The tilt table should be of the footplate support variety and allow rapid achievement of the upright position and allow calibrated angles of between 60 and 80°. A minimum of 3 ECG leads should be recorded simultaneously and continuously throughout the study. BP recording should ideally be the non-invasive continuous beat-to-beat method.
Advanced resuscitation equipment should be immediately available and to the standard required in exercise testing facilities. The test should be continuously supervised by a physician experienced in the management of the test and its possible complications.

The decision when to terminate the test influences the type of response. It is suggested that the test should continue until the precise occurrence of loss of consciousness with simultaneous loss of postural tone. Premature interruption of testing underestimates, and delayed interruption overestimates cardioinhibitory responses, and exposes the patients to the risks of prolonged hypotension and loss of consciousness. A consensus of when to stop the test does not exist and many physicians consider a steadily falling BP, accompanied by usual symptoms sufficient to stop the test.

Figure 1 is an algorithm of our current preferred clinical assessment and management of patients referred to our syncope and falls clinic, and demonstrates the complexity of managing these challenging patients. It is apparent that a physician who undertakes assessment of older subjects with falls/syncope requires a broad range of skills, with particular expertise in cardiovascular investigation in elderly, neurological training and knowledge of multi-professional functional assessment. Our 10 years of experience running a dedicated falls and syncope service suggests that appropriate investigation and management of these patients has key benefits, namely higher diagnostic rates and shorter hospital admissions with more efficient use of health service resources.

Table 7. Classification of neurocardiogenic syncope

<table>
<thead>
<tr>
<th>Response</th>
<th>Classification</th>
<th>Heart rate</th>
<th>Blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>Type 1</td>
<td>&gt;40 bpm or falls to &lt;40 bpm for &lt;10s</td>
<td>Falls before HR drops</td>
</tr>
<tr>
<td>Cardioinhibitory</td>
<td>Type 2A</td>
<td>&lt;40 bpm for &lt;10s</td>
<td>Falls before HR drops</td>
</tr>
<tr>
<td>Cardioinhibitory</td>
<td>Type 2B</td>
<td>&lt;40 bpm for &gt;10s, 3s asystole</td>
<td>Falls after HR drops</td>
</tr>
<tr>
<td>Vasodepressor</td>
<td>Type 3</td>
<td>Falls by &lt;10% of peak HR</td>
<td>Falls and produces syncope</td>
</tr>
</tbody>
</table>

Source: ACC/AHA guidelines for implantation of cardiac pacemakers and anti-arrhythmic devices. A report of the American College of Cardiology and the American Heart Association. Taskforce on practical guidelines (Committee on Pacemaker Implantation).25,26

Syncope

Initial evaluation

<table>
<thead>
<tr>
<th>Diagnostic</th>
<th>Suggestive</th>
<th>Inconclusive</th>
</tr>
</thead>
<tbody>
<tr>
<td>History, physical examination, ECG, SBP supine and upright, carotid sinus massage, blood chemistry and haematology</td>
<td>Cardiac</td>
<td>Cerebrovascular or psychiatric</td>
</tr>
<tr>
<td>2° step&lt;br&gt;Echocardiogram, Head CT, DEXA, DEXA, LUMBAR scans&lt;br&gt;Neurally-mediated&lt;br&gt;Cardiac</td>
<td>Neurally-mediated</td>
<td>Neurally-mediated&lt;br&gt;Cerebrovascular or psychiatric</td>
</tr>
<tr>
<td>3° step&lt;br&gt;EP study</td>
<td>Neurologist</td>
<td>CSM - Tilt test - ATP test&lt;br&gt;Psychiatric</td>
</tr>
<tr>
<td>4° step&lt;br&gt;CSM - Tilt test - ATP test</td>
<td>Neurologist</td>
<td>Cerebrovascular or psychiatric</td>
</tr>
<tr>
<td>5° step&lt;br&gt;Loop ECG</td>
<td>Frequent</td>
<td>Cerebrovascular or psychiatric</td>
</tr>
</tbody>
</table>

Fig. 1. Algorithm of assessment and management of patients referred to a dedicated syncope, falls and dizzy clinic
Summary

Accurate diagnosis of syncope in an older patient requires a high degree of clinical suspicion, thorough physical examination and carefully directed and interpreted investigations including repeated measurement of orthostatic BP, carotid sinus massage and head-up tilt testing. The importance of identifying a cause of syncope is vital if there is to be appropriate treatment to reduce associated falls, fractures and hospital admissions.

References

32. Lakatta EG. Do hypertension and aging have a similar effect on the myocardium? Circulation 1987;75(1 Pt 2):169-77.
35. Richardson DA, Bexton RS, Shaw FE, Kenny RA. Prevalence of cardioinhibitory carotid sinus hypersensitivity in patients 50 years or over presenting to the accident and emergency department with "unexplained" or "recurrent" falls. PACE: Pacing and Clinical Electrophysiology 1997;20(3 Pt 2):820-823.


