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# Primary Presentations of Syncope in the Older Adult Population

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*Syncope is a common presenting complaint in the older adult population. Unfortunately, cognitive issues and the fact that most falls are not witnessed in older adults can make the separation of falling and syncope quite difficult. In fact, about one third of older adults will have amnesia for faints, even if they are cognitively normal. A systemic approach can help separate cardiac from neurocardiovascular causes and avoid future mortality and morbidity.*

***Key words:*** *syncope, aging, neurocardiovascular instability, Holter monitoring, tilt table testing*

*Kenneth M. Madden, MSc, MD, FRCP(C), Assistant Professor, Division of Geriatric Medicine, Department of Medicine, University of British Columbia, Vancouver, BC.*

## Syncope—The Scope of the Problem

Syncope is a common presenting complaint, accounting for approximately 3% of all visits to both the emergency department and the primary care setting. The rates of syncope increase with age,[1] making the older adult population especially vulnerable to injury from this syndrome. Fainting-related falls, which are responsible for increased hospital admissions, hip fractures, and institutionalization have a direct cost to the Canadian health care system of 60 million dollars per year.[2,3]

## Is this Fall Due to Syncope?

Syncope is defined as "a transient loss of consciousness."[4] It is commonly taught in residency and medical school that this syndrome is primarily diagnosed by presyncopal symptoms (lightheadedness, giddiness, nausea, tunnel vision, and spots in the vision) that tend to precede the loss of consciousness. Unfortunately, in the older population one cannot always separate syncope-related falls and non-syncope-related falls. Cognitively normal older patients who faint under controlled laboratory conditions will not recall the event 30% of the time.[5] Cognitively impaired patients will be unable to recall presyncopal complaints, and this is compounded by the fact that most falls in older adults are unwitnessed.[6] Since syncope cannot always be reliably distinguished from falling in the older adult population, any unexplained repeated falling should have syncope considered as a contributing factor.

## Different Etiologies for Syncope

It is impossible to determine the cause of syncope in all cases. A prospective study of a standardized clinical evaluation (which included Holter monitoring, ambulatory loop monitoring, and tilt-table testing) failed to find an etiology 24% of the time.[7] Etiologies of syncope can be divided into cardiac causes and neurocardiovascular instability (Table 1). Regardless of cause, all patients with syncope syndromes should be tested and treated for osteoporosis and prescribed hip protectors.

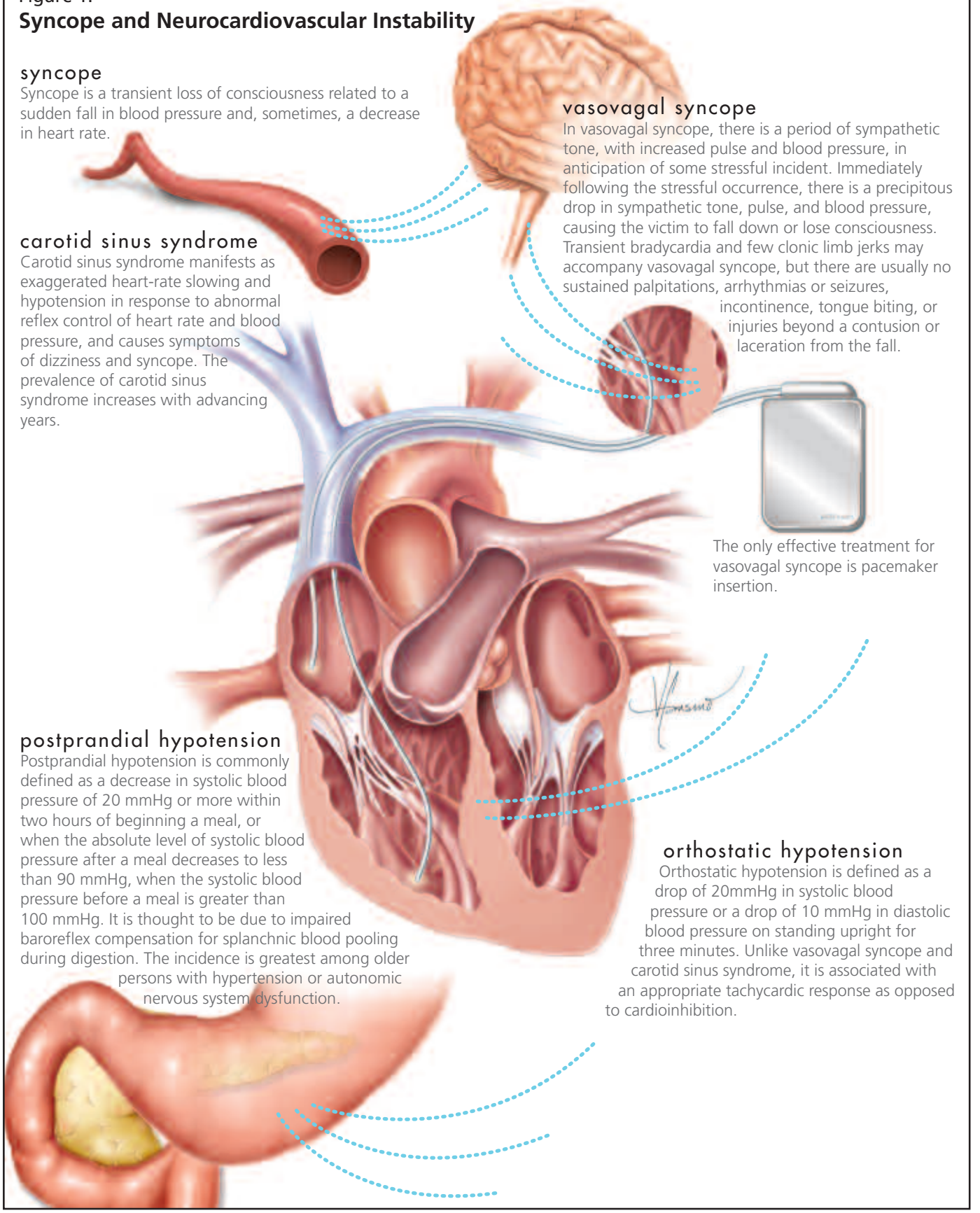
## Cardiac Causes of Syncope

Although cardiac causes of syncope are less common than neurocardiovascular causes, they are associated with a significantly higher one-year mortality than in other syncope patients.[8,9] Syncope due to cardiac causes (Table 1) is either due to organic heart disease (usually aortic stenosis in older adults) or arrhythmias (usually sinus node disease or heart blocks), which can have a significant cardiac mortality as well as mortality due to injury.[8,9] Organic heart disease is diagnosed on cardiac auscultation, assisted by the targeted use of echocardiography. Arrhythmias are diagnosed through electrocardiography and the targeted use of Holter monitoring and implantable loop recorders (see Diagnostic Approach below). Almost all cardiac causes of syncope require referral to a cardiologist for appropriate medical, surgical, and pacemaker therapy.[10]

## Neurocardiovascular Instability

Neurocardiovascular instability is defined as age-related changes in blood pressure and heart rate behaviour,

Figure 1:
**Syncope and Neurocardiovascular Instability**



### syncope
Syncope is a transient loss of consciousness related to a sudden fall in blood pressure and, sometimes, a decrease in heart rate.

### carotid sinus syndrome
Carotid sinus syndrome manifests as exaggerated heart-rate slowing and hypotension in response to abnormal reflex control of heart rate and blood pressure, and causes symptoms of dizziness and syncope. The prevalence of carotid sinus syndrome increases with advancing years.

### vasovagal syncope
In vasovagal syncope, there is a period of sympathetic tone, with increased pulse and blood pressure, in anticipation of some stressful incident. Immediately following the stressful occurrence, there is a precipitous drop in sympathetic tone, pulse, and blood pressure, causing the victim to fall down or lose consciousness. Transient bradycardia and few clonic limb jerks may accompany vasovagal syncope, but there are usually no sustained palpitations, arrhythmias or seizures, incontinence, tongue biting, or injuries beyond a contusion or laceration from the fall.

The only effective treatment for vasovagal syncope is pacemaker insertion.

### postprandial hypotension
Postprandial hypotension is commonly defined as a decrease in systolic blood pressure of 20 mmHg or more within two hours of beginning a meal, or when the absolute level of systolic blood pressure after a meal decreases to less than 90 mmHg, when the systolic blood pressure before a meal is greater than 100 mmHg. It is thought to be due to impaired baroreflex compensation for splanchnic blood pooling during digestion. The incidence is greatest among older persons with hypertension or autonomic nervous system dysfunction.

### orthostatic hypotension
Orthostatic hypotension is defined as a drop of 20mmHg in systolic blood pressure or a drop of 10 mmHg in diastolic blood pressure on standing upright for three minutes. Unlike vasovagal syncope and carotid sinus syndrome, it is associated with an appropriate tachycardic response as opposed to cardioinhibition.

predominately resulting in hypotension and bradyarrhythmia.[11] Orthostasis has been shown to cause pooling of up to 800 mL of blood volume in the lower limbs.[12] The normal physiologic response to orthostasis involves an increase in heart rate (to maintain cardiac output) and peripheral vasoconstriction. A deranged neurocardiovascular response results in a failure to maintain cerebral blood flow and can be divided into four types: vasovagal syncope (VVS), carotid sinus syndrome (CSS), orthostatic hypotension (OH), and postprandial hypotension (PPH) as illustrated in Figure 1.[11]

Vasovagal syncope (VVS) is the cause of approximately 30% of syncopal events in patients aged 60 years and older. This age-related increase in VVS susceptibility is exacerbated by dehydration, either through poor intake or through medications such as diuretics. The pathophysiology of vasovagal syncope involves the inappropriate stimulation of intraventricular mechanoreceptors as the heart attempts to maintain cardiac output in the face of orthostasis. Intraventricular mechanoreceptor stimulation triggers the Bezold-Jarish reflex, leading to vasodilatation and bradycardia.[13] VVS classically presents as a syncopal spell that occurs after a long period of standing, usually in the setting of dehydration (due to gastrointestinal illness or diuretics). Some have advocated the use of beta-blockers to reduce mechanoreceptor stimulation[14] but randomized controlled trials have not supported their use.[15] The only effective treatment for VVS is pacemaker insertion,[16] but this is usually reserved for troublesome, repeated cases that do not resolve with conservative care. Usually VVS is a self-limited condition, but unfortunately even a single fall can cause a debilitating fracture in an older adult.

Carotid sinus syndrome is characterized by an overshoot of the arterial baroreceptor response that results in transient asystole and vasodilatation.[17,18] It is present in 25% of older

**Table 1:** Causes of Syncope

| |
|---|
| **Cardiac** |
| *Arrhythmias* |
| Tachyarrhythmias (ventricular tachycardia, Torsade de pointes, superventricular tachycardia) |
| Bradyarrhythmias (sick sinus syndrome, heart blocks) |
| *Organic Heart Disease* |
| Aortic stenosis |
| Pulmonary embolism |
| Myocardial infarction |
| **Neurocardiovascular Instability** |
| Vasovagal syncope |
| Carotid Sinus syndrome |
| Orthostatic hypotension |
| Postprandial hypotension |
| **Syncope-like Syndromes** |
| Seizures |
| Psychiatric |
| Transient ischemic attacks |

**Table 2:** Causes of Orthostatic Hypotension

| |
|---|
| **Drugs** |
| Alpha-blockers |
| Beta-blockers |
| Parkinson's drugs (bromocriptine, levodopa, etc.) |
| Diuretics |
| Calcium channel blockers |
| Angiotensin-converting enzyme inhibitors |
| Angiotensin receptor blockers |
| Narcotics/opioids |
| Benzodiazepines |
| Tricyclic antidepressants |
| Nitrates |
| **Dysautonomia** |
| *Peripheral* |
| Autonomic neuropathy (diabetes, vitamin B12 deficiency, etc.) |
| Alcoholic polyneuropathy |
| Guillain-Barré syndrome |
| Paraneoplastic syndrome |
| *Central* |
| Multiple system atrophy (central dysautonomia) |
| **Hypovolemia** |
| **Low Cardiac Output** |
| Aortic stenosis |
| Myocardial infarction |
| Cardiomyopathy |
| **Venous Pooling** |
| Alcohol |
| Pyrexia |
| Exercise |
| Hot bath/hot environment |
| Sepsis |

adults who present to the emergency department with a fall[19] and has been shown to be an independent risk factor for hip fractures.[20] Spells can present with a preceding history of neck stimulation (such as turning the head or tight collars) but usually there is no recognizable trigger.[17] It is diagnosed by carotid sinus massage in the upright position[21] while on a cardiac monitor. There is no pharmacological therapy for CSS, but patients who present with a predominant asystolic response as opposed to vasodilation have been shown in randomized controlled trials to respond to pacemaker insertion.[10,18,22,23] In fact, pacemaker insertion in older adults with cardioinhibitory CSS has been shown to reduce falls by two-thirds and reduce injurious events by 70%.[18]

Orthostatic hypotension (OH) is defined as a drop of 20 mm of Hg in systolic blood pressure or a drop of 10 mm of Hg in diastolic blood pressure on standing upright for three minutes.[24] Significant orthostatic hypotension that occurs immediately on standing but recovers within three minutes is usually not a concern, but a failure to recover has been linked to an increased risk of falling even if asymptomatic.[25,26] Unlike VSS and CSS, it is associated with an appropriate (but ultimately ineffective) tachycardic response as opposed to cardioinhibition. It is usually caused by medications (especially vasodilators) or dehydration, but there is a variety of other causes (Table 2). Once the underlying cause has been addressed, mild orthostatic hypotension can be treated by nonpharmacological measures. These include slowly assuming the upright position, increasing sodium intake, and elevating the head of the patients bed 30 degrees at night (to prevent the loss of intravascular volume during nighttime supine diuresis). Physical exercise, hot temperatures, and alcohol consumption all cause vasodilatation and can worsen this syndrome. Isometric exercise maneuvers (such as squeezing crossed legs together) can raise blood pressure by approximately 20 mg of Hg and can prevent a syncopal spell. Refractory OH should be treated initially with fludrocortisone (a mineralocorticoid agonist that expands the intravascular volume) unless there are contraindications (congestive heart failure or other reasons for volume overload).[12] Midodrine (an alpha-1 agonist that causes vasoconstriction that has been validated in a randomized controlled trial[27]) should be added if fludrocortisone is ineffective or contraindicated.

**Table 3:** Syncope Workup in the Primary Care Setting

| |
|---|
| **All older adults presenting with syncope need the following:** |
| Bone density to screen for osteoporosis |
| Hip protectors |
| Electrocardiogram |
| Orthostatic vitals |
| **Is there a high pretest probability of a cardiac cause?** |
| Exercise-induced syncope, syncope in supine or sitting position |
| Evidence of structural heart disease (on exam or echocardiogram) |
| Evidence of low systolic function (on exam or echocardiogram) |
| Poor systolic function |
| Abnormal electrocardiogram |
| If there is, get a 24-h Holter (followed by a 48-h Holter if negative).<br>Consider implantable loop recorder if frequent and troublesome.<br>If unsure if there is structural heart disease on physical exam, get an echocardiogram. |
| **Is there orthostatic hypotension on physical exam (drop >20/10 in three minutes)?** |
| If there is, fix underlying cause.<br>Consider fludrocortisone and midodrine if refractory to more conservative measures. |
| **Are most of these spells postprandial (within 60 minutes after eating)?** |
| If there is, consider a trial of dietary modifications (more frequent smaller meals with lower carbohydrate content). |
| **Is this patient eligible for or agreeable to a pacemaker? Are the spells repeated and troublesome?** |
| If this is the case, consider referral to geriatric medicine specialist/internist/cardiologist for further workup for other causes of neurovascular instability (specifically carotid sinus hypersensitivity and vasovagal syndrome). |
| **Is this actually syncope?** |
| In all cases of syncope that you are unable to diagnose, consider the fact that this may not be syncope at all. Consider other etiologies such as seizures, gait disorders, or psychiatric diagnoses. If unsure if this is syncope or not, consider referral to a geriatric medicine specialist or a general internist. |

## Key Points

- Syncope is a common problem in older adults.
- Many older adults will not recall presyncopal symptoms. This may be due to cognitive issues but will also occur in one-third of older adults with normal memory.
- The key approach to syncope is to separate cardiac from neurocardiovascular causes. This is done by history, physical exam, and targeted use of Holter monitoring and echocardiography.
- There will be no cause found in one-quarter of syncope cases.

Postprandial hypotension is an uncommon condition in older adults where the increase in splanchnic blood flow after meals results in hypotension.[28,29] If a patient complains of frequent postprandial presyncopal or presyncopal spells after eating, a trial of dietary modification should be attempted (specifically, more frequent smaller meals and a reduction in the simple carbohydrate content of food).[11]

## Diagnostic Approach to Syncope

An organized approach to syncope of unknown origin hinges on the characterizing of cardiac versus neurocardiovascular causes of syncope. An intensive workup for cardiac causes of syncope should not be performed in all syncopal patients. The pre-test likelihood (therefore increasing your diagnostic yield) of a cardiac cause for syncope is highest in patients with abnormalities on ECG, with structural heart disease (as determined by physical exam and targeted echocardiography), and with classic arrhythmic syncope symptoms (exertional syncope or syncope in the seated or supine position). For neurocardiovascular causes, orthostatic hypotension is unusual in that it is diagnosed at the bedside and requires no further diagnostic testing. Intensive testing for other causes of neurocardiovascular instability (VVS, CSS) should only be performed in patients where the syncope is a recurrent problem, since these are usually treated by pacemaker insertion. A step-by-step approach to managing syncope in the primary care setting is outlined in Table 3. Individual diagnostic modalities are discussed separately below.

## Holter Monitoring

The overall diagnostic yield of 24-hour Holter monitoring is quite low in the workup of syncope (7%), but this yield was doubled in the presence of electrocardiographic abnormalities.[7] Holter monitoring should never be used as a screening tool for all older fallers—a comparison between older adults who fell frequently and non-falling older adults revealed no difference in rates of abnormalities on Holter monitoring.[30] Holter monitoring should be targeted to patients with ECG abnormalities, heart disease (including congestive heart disease), and those who have symptoms that are highly suspicious for arrhythmia (syncopal spells while seated or the lying position or exertional syncope). Targeted Holter monitoring should consist of an initial 24-hour recording followed by a 48-hour recording if the first one was negative.

## Echocardiography

The role of echocardiography in the workup on syncope remains uncertain since the yield has prospectively found to be quite low in patients that present with syncope to the emergency department.[7] Therefore, echocardiography should only be used to detect structural heart disease or to check for low systolic function (if not already determined by your physical exam).

## Implantable Loop Recorders (ILRs)

ILRs are devices that are surgically implanted in the upper chest and can provide cardiac monitoring for months as opposed to merely one or two days. They have been shown to increase the rate of diagnosis and reduce hospitalizations in syncope patients.[31] However, insertion of these devices require referral to a specialized cardiology centre; the devices should therefore only be used if symptoms are intolerable and repeated Holter monitoring has been unhelpful.

## Tilt Table Testing

Tilt table tests attempt to diagnose VVS by reproducing a vasovagal episode under laboratory conditions. Although still occasionally helpful in making the diagnosis of VVS, the lack of a gold standard response for comparison[32] and their only moderate reproducibility[32] make them not particularly helpful for diagnosing VVS in the primary care setting.[33]

## Conclusion

Diagnosing syncope can be a challenge in any population, but cognitive issues and the overlap between syncopal and nonsyncopal falls in older adults can make diagnosis especially difficult. An organized approach that carefully separates cardiac from neurocardiovascular causes (with a recognition that a large portion of syncope presentations remain undiagnosed) combined with careful acquisition of collateral history from the patients' family and caregivers can help speed diagnosis and avoid future morbidity from falls.

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