

FLORIDA SOUTHERN COLLEGE

Syncope in Athletes

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## Abstract

Syncope is defined as the transient loss of consciousness with an inability to maintain postural tone followed by a spontaneous recovery. Syncope is a frequently encountered problem with etiologies ranging from benign to life threatening. It accounts for approximately 3% of emergency department visits and between 1 and 6% of acute hospital medical admissions. Syncope is an underlying symptom of a number of different medical conditions. The etiologies can be categorized as cardiac, metabolic, psychiatric, or neurological. The most frequent diagnosis category is cardiac. Cardiac causes of syncope can further be subdivided into the following categories: structural disease, cardiac arrhythmias, neurocardiogenic syncope, carotid sinus syncope, or orthostatic (postural) syncope.

When a patient has experienced syncope, a thorough assessment explores associated symptoms, physical examination, environment, drugs, and family history. The most commonly used tests for syncope include electrocardiogram (ECG), kidney function tests, table tilt test, carotid sinus massage, and exercise testing. Immediate treatment includes evaluating the individual's airway, breathing, and circulation (ABC) status and determining if emergency medical help is necessary. Secondary treatment of syncope includes education, maneuvers to avert episodes, drug therapy, and pacemakers. With regard to prognosis associated with syncope, two important elements should be considered: (1) risk of death and life-threatening events; and (2) risk of reoccurrence of syncope resulting in physical injury. The majority of patients who have sustained syncope can be comforted by the knowledge that the prognosis is excellent and the risk of death is minimal to non-existent.

Keywords: syncope, neurocardiogenic syncope, athlete

## Introduction

Syncope is a commonly encountered problem<sup>1</sup>. The etiology ranges from benign conditions, such as vasovagal reactions, to life-threatening conditions, such as hypertrophic cardiomyopathy<sup>2</sup>. It accounts for approximately 3% of emergency department visits and between 1 and 6% of acute hospital medical admissions affecting 6 per 1000 people per year<sup>1</sup>.

Syncope is defined as a transient loss of consciousness with an inability to maintain postural tone followed by a spontaneous recovery<sup>1</sup>. The onset of syncope is rapid, being no more than ten to twenty seconds after onset of premonitory symptoms (if any)<sup>4</sup>. Recovery is spontaneous, complete, and usually prompt, as opposed to other medical conditions that will not reverse themselves to normal without medical intervention. Examples of such conditions include coma (i.e. hypoglycemia), stroke, cardiac arrest, and intoxicated states (alcohol, narcotics)<sup>4</sup>. Syncope is a symptom, not a disease, and should be classified according to the underlying cause<sup>3</sup>.

## Etiology

Syncope is an underlying symptom of a number of different medical conditions. The causes of syncope can be broadly categorized as cardiac, metabolic, psychiatric, or neurologic.

### *Cardiac*

Cardiac syncope is due to a heart or blood vessel condition that interferes with blood flow to the brain<sup>14</sup>. Cardiac syncope can be subdivided into structural, cardiac arrhythmias, neurally mediated syncope, and orthostatic.

**Structural diseases** are relatively infrequent causes of fainting but are particularly important to recognize, as they are warnings of potentially life-threatening conditions<sup>4</sup>. Syncope is of great concern when associated with conditions in which there is fixed or dynamic obstruction to left ventricle outflow<sup>12</sup>. Patients with underlying cardiac disease are at greater risk for recurrent syncopal events than are other patients with syncope<sup>5</sup>.

**Cardiac arrhythmias** (abnormal electrical activity of the heart) cause syncope due to bradycardia or tachycardia<sup>4</sup>. Tachycardia (fast heart rate) doesn't allow enough time for the heart to fill with blood between each heartbeat, decreasing the amount of blood the heart can deliver to the body<sup>8</sup>. With bradycardia (slow heart rate), the heart's ability to pump blood may be compromised due to inadequate force production<sup>8</sup>. Both induce hemodynamic impairment, which can cause a critical decrease in cardiac output and cerebral blood flow<sup>12</sup>. In either case, syncope tends to occur at the onset of the dysrhythmia problem, before compensatory vasoconstriction has a chance to respond and support the central systemic pressure<sup>4</sup>.

The most common type of cardiac syncope is neurocardiogenic (vasovagal). **Neurocardiogenic syncope**, with a mean prevalence of 22% in the general population, is defined as a syndrome in which "triggering of a neural reflex results in a usually self-limited episode of systemic hypotension characterized by both bradycardia and peripheral vasodilation"<sup>3</sup>. The balance between the chemicals adrenaline and acetylcholine is disrupted<sup>8</sup>. Adrenaline stimulates the body, while acetylcholine does the opposite<sup>8</sup>. When the vagus nerve is stimulated, excess acetylcholine is released resulting in the heart rate slowing and blood vessels dilating, making it harder for blood to defeat gravity and pump

to the brain<sup>8</sup>. This temporary decrease in blood flow may cause a syncopal episode<sup>8</sup>.

Vasovagal syncope has three distinct phases: a prodrome, loss of consciousness, and a postsyncopal phase<sup>5</sup>. A precipitating event or situation (e.g., emotional stress, trauma, pain, sight of blood, prolonged standing) usually is identifiable<sup>5</sup>. The prodrome, characterized by diaphoresis, epigastric discomfort, extreme fatigue, weakness, yawning, nausea, dizziness, and/or vertigo, results from increased parasympathetic tone and may last seconds to several minutes<sup>5</sup>. Lying down or removing the stimulus may abort the syncopal episode. The postsyncopal phase may last hours or, rarely, days and may include protracted confusion, disorientation, nausea, dizziness, and a general sense of poor health<sup>5</sup>.

**Carotid sinus syncope** deserves special mention. Carotid sinus hypersensitivity is a well-recognized cause of syncope and falls in older persons, responsible for up to a third of symptoms in patients referred to tertiary care<sup>26</sup>. Carotid sinus syncope is suggested by a history of syncope after head turning, shaving, or wearing a tight collar, particularly in older patients<sup>5</sup>. Carotid sinus hypersensitivity is an age related phenomenon, rarely diagnosed in patients with syncope younger than 50 years of age<sup>26</sup>. The carotid sinus reflex plays a central role in blood pressure homeostasis<sup>21</sup>. Changes in stretch and pressure are detected by baroreceptors in the heart, carotid sinus, aortic arch, and other large vessels<sup>21</sup>. Afferent impulses are transmitted by the carotid sinus, glossopharyngeal, and vagus nerves to the brain stem<sup>21</sup>. Efferent limbs are carried through sympathetic and vagus nerves to the heart and blood vessels, controlling heart rate and vasomotor tone<sup>21</sup>. In carotid sinus hypersensitivity, mechanical deformation of the carotid sinus (located at the bifurcation of the common carotid artery) leads to an

exaggerated response with bradycardia or vasodilatation, resulting in hypotension, presyncope, or syncope<sup>21</sup>.

**Orthostatic (postural) syncope** is also common, and most often is associated with movement from lying or sitting to a standing position<sup>4</sup>. This is common in older patients that have underlying medical problems (i.e. diabetes, certain nervous system diseases), or persons who are dehydrated from hot environments or inadequate fluid intake<sup>4</sup>. Orthostatic hypotension may be due to volume depletion medications that alter vascular tone and heart rate, secondary autonomic dysfunctions (i.e. chronic disease such as diabetes mellitus and from toxins), or primary forms of autonomic failure<sup>6</sup>. Certain commonly prescribed medications such as diuretics, beta-adrenergic blockers, antihypertensives, or vasodilators (i.e. nitroglycerin) predispose postural syncope<sup>4</sup>.

### **Other categories**

The other three categories (metabolic, psychiatric, and neurological) that cause syncope are often overlooked. Metabolic causes of syncope are rare, accounting for less than 5% of syncopal episodes<sup>20</sup>. The most common metabolic cases are hypoglycemia, hypoxia, and hyperventilation<sup>20</sup>. Syncope in a diabetic is more often due to autonomic insufficiency rather than hypoglycemia<sup>20</sup>. Dysfunction leading to syncope, while multifactorial and complex, is most commonly thought to be metabolic in nature or secondary to insufficient cerebral cellular perfusion, or both<sup>28</sup>. Psychiatric illness must be considered as a cause of syncope, especially in young patients and those with multiple syncopal episodes who also have other nonspecific complaints<sup>17</sup>. The disorders that may cause syncope include generalized anxiety and panic disorders, major depression, somatization disorder, and alcohol and substance abuse<sup>17</sup>.

Pain can stimulate the vagus nerve and is a common cause of vasovagal syncope<sup>8</sup>. Other noxious stimuli can do the same thing, including a situational stressor<sup>8</sup>. Syncope can be situational related to micturition, defecation, coughing, or gastrointestinal stimulation<sup>5</sup>. The mechanism involves a similar vagal stimulation in addition to the decreased venous return associated with the Valsalva maneuver<sup>5</sup>. The Valsalva maneuver occurs when a person tries to exhale forcibly with a closed glottis (the windpipe) so that no air exits through the mouth or nose as, for example, in strenuous coughing, straining during a bowel movement, or lifting a heavy weight<sup>27</sup>. The Valsalva maneuver impedes the return of venous blood to the heart<sup>27</sup>.

Neurological causes of syncope include migraines, seizures, and transient ischemic attacks<sup>20</sup>. They are surprisingly uncommon causes of syncope, accounting for less than 10% of all cases of syncope<sup>20</sup>. Most patients with neurological cause of syncope have seizures rather than true syncope<sup>20</sup>.

### **Athletes and syncope**

It is important to consider the relationship between athletes and syncope. Exercise-related syncope occurs either during or immediately after a period of exercise<sup>28</sup>. Reflex syncope during exercise is caused by marked hypotension without bradycardia<sup>29</sup>. Post-exertional syncope is almost invariably due to autonomic failure or to a neurally-mediated mechanism and is characterized by hypotension which can be associated with marked bradycardia or asystole; it typically occurs in subjects without heart disease<sup>29</sup>. The sports medicine literature also recognizes the term exercise-associated collapse (EAC) to describe athletes who are unable to stand or walk unaided as a result of light-headedness, faintness, dizziness or syncope<sup>28</sup>. EAC specifically excludes orthopedic

injuries (e.g., sprained ankle, leg cramps) that would preclude completing a sports event<sup>28</sup>.

While the literature on exercise-related syncope is limited, several themes are consistent. First, exertion is associated with a minority of syncopal events, representing only 3 to 20 percent of cases<sup>28</sup>. Second, although the great majority of cases of syncope are benign and have a favorable outcome, young and otherwise healthy adults who present with exertional (rather than nonexertional) syncope have a greater probability of organic etiology (such as hypertrophic cardiomyopathy or arrhythmogenic right ventricular dysplasia)<sup>28</sup>. When athletes experience syncope repeatedly, it is more likely that cardiac complications or disease are involved, but proper diagnosis and medications may improve the possibility that the athlete can return to sport<sup>30</sup>. Conversely, syncope during effort was not associated with a cardiac cause of syncope in patients without structural heart abnormalities<sup>29</sup>. In these cases the syncope mechanism may be a manifestation of exaggerated reflex vasodilation<sup>29</sup>.

Vasovagal syncope is probably the most common type of fainting seen among athletes<sup>30</sup>. During exercise, increases in heart rate and stroke volume result in a dramatic rise in cardiac output<sup>28</sup>. Muscular activity is crucial to maintaining this cardiac output because muscle contractions are the driving pressure that sustains venous return<sup>28</sup>. After exercise, without the muscular activity to increase venous return, cardiac filling may decrease dramatically because of the reduction in left ventricular end-diastolic volume and stroke volume<sup>28</sup>. Vigorous contractions of the ventricle in response to prolonged standing, dehydration, exercise, and increased blood levels of the hormone epinephrine are believed to initiate a vasovagal reaction by activating cardiovascular receptors to



initiate a reflex in the brain that produces vasovagal syncope<sup>30</sup>. The enhanced tone of the vagus nerve in endurance athletes probably explains why they are more susceptible to exercise-induced vasovagal syncope<sup>30</sup>.

In the absence of structural heart disease, syncope occurring during or immediately after exercise is invariably a benign condition and a strong predictor of a good outcome, either in athletes or in the sedentary population<sup>29</sup>. Orthostatic hypotension that occurs after exercise, usually in association with sudden cessation of activity, is much less ominous than the sudden loss of consciousness that occurs during exercise (which suggests an arrhythmic or cardiac etiology)<sup>28</sup>. There is no reason to consider athletes different from sedentary subjects<sup>29</sup>. A summary of the causes of syncope is listed in Box 1.

### **Signs and Symptoms**

The signs and symptoms of syncope include nausea, dizziness, blurred vision, and diaphoresis<sup>2</sup>. Some individuals may experience presyncope symptoms. The presence of presyncopal symptoms such as nausea, diaphoresis (sweating), dizziness, and a feeling of warmth may suggest vasovagal syncope<sup>1</sup>. Precipitant factors (micturition and coughing) may suggest situational syncope, and a positional aspect (syncope precipitated by rising from a sitting position) may suggest orthostatic syncope<sup>1</sup>. It is important to identify features in the history that may point to seizure activity, the most important of which is the presence of a post-ictal phase<sup>1</sup>. The post-ictal phase is the transition from seizure back to the individual's normal state signifying the recovery period for the brain<sup>7</sup>. While confusion may be present immediately after syncope, this should not last for more than a minute<sup>1</sup>. Other important symptoms prior to the syncopal event include chest pain,

sudden onset of headache, palpitations, back pain, or focal neurological deficits<sup>1</sup>. The presence of any of these may suggest an alternative serious cause<sup>1</sup>.

### **Evaluation, Referral and Diagnostic Tests**

Clinical assessment of syncope is challenging, owing to the heterogeneous nature of underlying causes, ranging from benign neurocardiogenic syncope to potentially fatal dysrhythmias and pulmonary embolism<sup>1</sup>. A thorough assessment of the associated symptoms, physical examination, environment, drugs, and family history can provide important information about the cause. During the initial evaluation of a syncope patient, it is important to obtain a thorough medical history. The history should also include a thorough review of systems<sup>6</sup>. A detailed physical examination should be performed including the following: orthostatic vital signs, complete blood count, blood glucose, and electrolytes<sup>2</sup>. Other essential laboratory tests include an electrocardiogram (ECG), kidney function tests, table tilt test, carotid sinus massage, and exercise testing<sup>8</sup>. Head computed tomography (CT), magnetic resonance imaging (MRI), electroencephalography (EEG), and carotid ultrasonography should be ordered only when history or physical examination suggests a neurologic cause or after testing for cardiac or neurally mediated causes of syncope has been completed<sup>5</sup>. In the evaluation of syncope, the presence of a structural heart disease has emerged as the most important factor for predicting the risk of death, as well as the likelihood for arrhythmias<sup>6</sup>.

#### *Electrocardiogram*

A standard 12 lead ECG is warranted in all cases of syncope unless the history and physical examination reveal an obvious non-cardiac cause<sup>1</sup>. There are several ECG ambulatory-monitoring systems available: conventional Holter monitoring, in-hospital,

and external or implantable loop recorders<sup>12</sup>. An electrocardiogram is a noninvasive test that is used to reflect underlying heart conditions by measuring the electrical activity of the heart<sup>8</sup>. Abnormal ECG findings occur in about 90 percent of patients with cardiac-induced syncope but in only 6% of patients with neurally mediated syncope<sup>5</sup>. A further study showed that an abnormal ECG, defined as rhythm or conduction abnormality, atrioventricular block, signs of an old myocardial infarction, left or right ventricle hypertrophy or frequent premature ventricular contractions, was a predictor for dysrhythmic syncope<sup>1</sup>.

Most ECG monitoring in syncope is undertaken with external twenty-four hour Holter recorders (see figure 1) connected to the patient via external wiring and adhesive ECG patches<sup>9</sup>. The relevant findings on electrocardiographic monitoring are symptoms that occur in conjunction with arrhythmias and symptoms that occur without concomitant arrhythmias<sup>6</sup>. Symptoms that occur without accompanied arrhythmias can potentially exclude arrhythmias as a cause of the symptoms<sup>16</sup>. Holter monitoring is useful in unexplained syncope or when/if an arrhythmic etiology is suggested by history in a patient at relatively high risk for arrhythmia<sup>15</sup>. The major limitation is that patients may not experience symptoms or cardiac arrhythmias during the recording period<sup>15</sup>. The physical size of the device may impair the ability of patients to sleep comfortably or engage in activities that precipitate or reproduce symptoms<sup>15</sup>. Therefore, it is not surprising that Holter monitoring has a low diagnostic yield<sup>15</sup>. In-hospital monitoring is warranted only when a patient is at high risk for life-threatening arrhythmias<sup>12</sup>. An external loop recorder has a loop memory that continuously records and deletes ECG<sup>12</sup>. When activated by the patient, typically after the symptoms have occurred, the ECG is

stored and can be retrieved for analysis<sup>12</sup>. A recent study found that external loop recorders had an increased diagnostic yield, when compared with Holter monitoring because patients can wear it for a month<sup>12</sup>.

### *Echocardiogram*

Echocardiography is unlikely to be helpful in the absence of known cardiac disease, a history suggestive of cardiac disease, or an abnormal ECG<sup>5</sup>. However, in patients with syncope who have a history of heart disease or an abnormal ECG, echocardiography is useful<sup>5</sup>. This test provides information about the type and severity of an underlying heart disease, which may be useful for risk stratification<sup>12</sup>. If moderate to severe structural heart disease is found, evaluation is directed toward a cardiac cause of syncope<sup>12</sup>. On the other hand, if minor structural abnormalities are discovered, the probability of a cardiac cause in syncope may not be high, and the evaluation may proceed as in patients without structural heart disease<sup>12</sup>. Recently, implantable loop recorders have become available to study patients with unexplained recurrent syncopal events. This device has a solid-state loop memory capable of storing electrocardiographic events up to 40 minutes before and 2 minutes after the activation. It can be easily placed subcutaneously under local anesthesia and has a battery life of 36 months<sup>25</sup>.

### *Table Tilt Test*

Once cardiac arrhythmias, structural heart disease, and non-cardiac causes of syncope have been ruled out, head up tilt testing is usually the next line of testing. Head-up tilt table testing has become a widely accepted tool in the clinical evaluation of patients' presenting with syncopal symptoms<sup>10</sup>. Tilt testing is an orthostatic stress test, used when neurocardiogenic syncope is suspected<sup>3</sup>. The patient is normally instructed to

fast overnight or several hours before the testing if scheduled later in the day<sup>10</sup>. To diminish the possibility of a “false positive” test it is reasonable to consider the provision of parenteral fluid replacement before initiating the procedure<sup>10</sup>. The patient is in resting supine position for twenty to forty-five minutes<sup>11</sup>. The procedure should be explained to the patient, who should be securely strapped to the tilt table prior to the assumption of the upright position to prevent collapse and injury if syncope occurs during syncope (see figure 2)<sup>11</sup>. The patient should also be instructed to avoid lower extremity movement to maximize venous pooling<sup>11</sup>. A minimum of three electrocardiographic leads should be recorded simultaneously and continuously throughout the study<sup>10</sup>. Blood pressure recordings should also be done continuously throughout the study<sup>10</sup>. The angle and duration of the test are the most powerful determinants of its diagnostic utility. The evidence available to date suggests that a tilt angle between 60° and 80° are optimal in creating sufficient orthostatic stress without increasing the number of false positives<sup>11</sup>. In general, a tilt test response will be deemed to be positive for vasovagal syncope if the syncopal symptoms are reproduced by the provocation of neurally mediated hypotension or bradycardia, or both, as of result of the procedure<sup>10</sup>. Hypotension is blood pressure that is lower than 90/60 mmHg<sup>18</sup>. Bradycardia is defined as a resting heart rate of 60 beats/minute or less<sup>2</sup>. The optimal duration for the tilt table test has yet to be determined. However, in the absence of pharmacological provocation, durations of thirty to forty-five minutes at 60° to 80° have become widely accepted in laboratory evaluation of older adolescents and adult patients<sup>10</sup>. The indications and contraindications for this test are summarized in Box 3.

*Carotid Sinus Hypersensitivity*

The patient should be supine with the neck slightly extended. The carotid pulsation should be palpated in the upper portion of the neck and pressure should be applied at the point in the direction of the vertebral column for 5 seconds (see figure 3)<sup>16</sup>. Both sides should be tested, but never simultaneously. For safety, the carotid artery should first be auscultated for bruits, and the presence of significant cerebrovascular disease should be excluded by the history and examination<sup>16</sup>. It has been observed that pressure at the site where the common carotid artery bifurcates produces a slowing heart rate and fall in blood pressure<sup>12</sup>. In some individuals, this reflex initiated by the carotid sinus massage results in an abnormal response<sup>12</sup>. A ventricular pause lasting more than 3 seconds and/or a fall in systolic blood pressure of more than 50mmHg defines carotid sinus hypersensitivity<sup>12</sup>. Carotid sinus hypersensitivity is a common finding in older male individuals<sup>12</sup>. Box 4 lists the indications and contraindications for the carotid sinus massage testing.

*Exercise Testing*

Exercise-induced syncope is a rare finding, occurring in up to 5% of patients with unexplained syncope<sup>9</sup>. The exercise stress test should be performed after the echocardiogram<sup>29</sup>. The test should be designed to reproduce the conditions that provoked the specific syncopal event and challenge the individual athlete<sup>29</sup>. Exercise testing should be performed in patients who experience episodes of syncope during or shortly after exertion<sup>9</sup>. Exercise testing can diagnose ischemia and exercise-induced tachyarrhythmias or reproduce exercise-associated and exertional syncope<sup>5</sup>. A modified treadmill test with abrupt termination has a high diagnostic value<sup>9</sup>. Syncope during exercise may be cardiac

in origin with marked hypotension without bradycardia<sup>9</sup>. By contrast, postexertional syncope, as distinct from exertional syncope, results from autonomic failure and reflex-mediated mechanisms<sup>5</sup>. It has been supposed that adrenaline plays an important role in the mechanisms leading to neurally mediated syncope associated with exercise<sup>9</sup>.

A witness history should be sought and a drug history taken to identify the use of antihypertensive or other cardiac medication, and drugs that cause bradycardia, hypotension, or prolong the QT interval (erythromycin, quinine and major tranquilizers)<sup>1</sup>. A menstrual history should also be taken in women of childbearing age, as syncope is a not uncommon presentation of ectopic pregnancy<sup>1</sup>. In addition, neurocardiogenic syncope is relatively common in early pregnancy<sup>1</sup>. Some patients presenting with syncope may be under the influence of alcohol or recreational drugs. While these substances may lead to collapse, syncope is unlikely to occur as a direct consequence of either alcohol or recreational drugs<sup>1</sup>. Finally, a family history of cardiac disease or sudden unexplained death or history of syncope is important to ascertain.

## **Treatment**

Immediate treatment for a patient who has sustained syncope is to evaluate airway, breathing, and circulation (ABC) status<sup>2</sup>. It is important to protect the individual from injury. If the person does not regain consciousness within a minute or two, or there is an absence of pulse and breathing, it is important to contact emergency medical help immediately<sup>22</sup>. If the patient is pale, sweating, and has good airway, breathing, and circulation, the next step is to raise the feet, thereby assisting the venous return to the vital organs, particularly the heart and brain<sup>2</sup>. It may be helpful to loosen belts, collars, or tight clothing<sup>22</sup>. An individual who has just fainted will probably regain consciousness

quickly, but may continue to feel weak for a brief time<sup>22</sup>. To keep from fainting again, the person should stay lying quietly for a few minutes<sup>22</sup>. Even when people recover promptly, they should contact their doctors about a first fainting attack, about repeated fainting spells, or about other possible symptoms including irregular heartbeat, chest pain, shortness of breath, blurred vision, confusion, or trouble talking<sup>22</sup>.

Secondary treatment of syncope includes education, maneuvers to avert episodes, drug therapy, and pacemakers<sup>3</sup>. Education of the mainstay treatments includes recognition and avoidance of predisposing situations, and maneuvers to abort the episodes such as lying down<sup>9</sup>. Therapy is determined by the underlying cause.

Treatment of patients with orthostatic hypotension should include volume replacement when the patient has intravascular volume depletion and the discontinuation or reduction of the dose of drugs that may be responsible for orthostatic syncope<sup>6</sup>. It is reasonable for all patients to receive advice and education on factors that influence systemic blood pressure, such as avoiding sudden head-up postural changes, standing still for a prolonged period of time, prolonged recumbence during daytime, straining during micturition and defecation, hyperventilation, high environmental temperatures, severe exertion, large meals and alcohol<sup>23</sup>. Patients should also be instructed to rise slowly from the bed or chair and to move legs prior to rising to facilitate venous return from the extremities<sup>19</sup>. An additional treatment of orthostatic syncope is chronic expansion of intravascular volume by encouraging a higher than normal salt and fluid intake<sup>23</sup>. In highly motivated young patients with recurrent syncope triggered by orthostatic stress, the prescription of progressively prolonged periods of enforced upright posture (tilt training) may reduce syncope recurrence<sup>12</sup>. However, this treatment is hindered by the



low compliance of patients to continue the training program for a long period<sup>20</sup>. Other useful treatments may include elevation of the head, elastic stockings, antigravity or g-suits, salt loading, and pharmacologic agents such as sympathomimetic amines, monoamine oxidase inhibitors, and beta-blockers<sup>19</sup>.

Many drugs have been tested in the treatment of syncope. In general, while the results have been satisfactory in uncontrolled trials or short term controlled trials, long-term placebo-controlled prospective trials have been unable to show a benefit of the active drug over placebo<sup>23</sup>. Beta-blockers are preferred as initial treatment, as they are believed to reduce the degree of mechanoreceptor activation and block the effects of circulating catecholamines<sup>3</sup>.  $\beta$ -Adrenergic antagonists are the most widely used agents<sup>19</sup>. However, beta-adrenergic blocking drugs have failed to be effective in several long-term follow up studies<sup>23</sup>.  $\alpha$  agonists work by increasing peripheral vascular resistance and reducing vascular capacitance (to cause increased venous return)<sup>3</sup>. Serotonin reuptake inhibitors and bupropion SR are also effective<sup>19</sup>. They selectively block serotonin, which has been shown to induce vagally mediated bradycardia and blood pressure lowering<sup>3</sup>. Fludrocortisone, a mineralocorticoid that promotes renal reabsorption of sodium to cause an increase blood volume has been used in the treatment of vasodepressor syncope in both children and adults<sup>3</sup>. Vascular volume and preload are maintained through the resultant sodium and water retention by fludrocortisone, thereby preventing activation of the cardiac mechanoreceptors<sup>3</sup>. The mineralocorticoid hydrofludrocortisone may be helpful for the refractory patients with recurrent vasovagal syncope but side effects, including increase resting blood pressure, limit their usefulness<sup>19</sup>.

The rationale for the use of a pacemaker in vasovagal syncope is that bradycardia often occurs at the time of syncope<sup>17</sup>. Cardiac pacing appears to be beneficial in cases of carotid sinus syncope<sup>12</sup>. The prevention of bradycardia is the main physiological mechanism by which a pacemaker can prevent attacks of syncope<sup>17</sup>. However, patients with vasovagal syncope often experience reductions in blood pressure at the beginning of a syncopal episode and heart rate changes later<sup>17</sup>. If profound hypotension has already occurred, pacing therapy will not help patients even if bradycardia or asystole has been demonstrated at the time of syncope<sup>17</sup>. Pacing may be life saving in patients with bundle branch block and syncope in whom the mechanism of the faint is suspected to be intermittent atrioventricular block<sup>23</sup>. A summary of the treatment protocols is listed in box 5.

### **Prognosis, Return to Participation, and Prevention**

With regard to prognosis associated with syncope, two important elements should be considered: (1) risk of death and life-threatening events; and (2) risk of reoccurrence of syncope resulting in physical injury<sup>12</sup>. Structural heart disease and primary electrical disease are major risk factors for sudden cardiac death and overall mortality in patients with syncope<sup>12</sup>. It is essential to define the underlying structural heart disease and treat the cardiac disease in order to have an impact on mortality and sudden death rates<sup>16</sup>. Most deaths that occur seem to be related to the severity of the underlying disease rather than to syncope<sup>12</sup>. Patients with vasovagal syncope can be comforted by the knowledge that their prognosis is excellent and their risk of death is minimal to non-existent<sup>13</sup>. The number of episodes of syncope during life is the strongest predictor of recurrences<sup>12</sup>. In patients presenting with syncope, recurrence rate is 34% over 3 years follow-up<sup>12</sup>.

Return to participation is dictated by the etiology of the syncope and how well the underlying problem can be controlled or eliminated<sup>2</sup>. Vasovagal syncope is not a cause for any athletic restrictions<sup>2</sup>. Management of neurocardiogenic syncope in competitive athletes is controversial<sup>28</sup>. A consultant who is familiar with this population optimally manages the condition<sup>28</sup>. While pharmacologic therapy may be warranted in carefully selected cases, nonpharmacologic therapy that is focused on training techniques and behavior modification should be attempted first<sup>28</sup>.

Prevention is influenced by the cause of the syncope. There may be opportunities to prevent episodes of syncope. Patients who have had a vasovagal episode may be aware of the warning signs and be able to sit or lie down before fainting to avert the syncope episode<sup>8</sup>. For older patients with orthostatic hypotension, waiting for a brief period after changing positions may help<sup>8</sup>. If dehydration was the cause of syncope, it is important to have adequate fluid intake<sup>8</sup>.

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**Box 1: Causes of syncope<sup>3</sup>*****Cardiac causes***

- Structural cardiac or cardiopulmonary disease (Aortic stenosis, mitral stenosis, pulmonary stenosis, left atrial myxoma, aortic dissection, acute myocardial infarction, cardiac tamponade, pulmonary embolism, obstructive cardiomyopathy)
- Cardiac arrhythmias (tachyarrhythmias, bradyarrhythmias)
- Neurally mediated syncopal syndrome (includes neurocardiogenic or vasovagal syncope, carotid sinus syncope, and situational syncope)
- Orthostatic (or postural) hypotension

***Metabolic causes***

- Hypoxia
- Anemia
- Hypoglycemia
- Hyper ventilation

***Psychiatric causes***

- Somatization disorders
- Hysteria
- Panic
- Fright

***Neurological causes***

- Seizure disorders
- Transient ischemic attacks

Box 2: Relevant Historical Features<sup>3</sup>

- Precipitating Factors (pain, anxiety)
- Postural or Exertional Symptoms
- The Situation (i.e. after urination)
- Associated Neurological Symptoms
- History of Cardiac Disease
- History of Psychiatric Illness
- Current Medications
- Family History of Sudden Death

**Box 3: Indications & Contradictions for Tilt Table Testing<sup>3</sup>****Indications**

- Recurrent syncope or single syncopal episode accompanied by physical injury or motor vehicle crash or occurring in a high risk setting (for example, pilot, surgeon, commercial vehicle driver) and no evidence of structural cardiovascular disease; or presence of structural cardiovascular disease but other causes of syncope ruled out by diagnostic testing
- Syncope induced by or associated with exercise
- Further evaluation of patients in whom an apparent specific cause of syncope has been established (for example, asystole, high atrioventricular block) but susceptibility to neurocardiogenic syncope may affect treatment plan.

**Contraindications**

- Syncope with severe left ventricular outflow obstruction (for example, aortic stenosis)
- Syncope in presence of severe mitral stenosis
- Syncope in setting of known critical proximal coronary artery disease
- Syncope in setting of known critical cerebrovascular disease



**Box 4: Indications & Contraindications for Carotid Sinus Massage<sup>5</sup>**

**Indications**

- History of syncope after head turning, shaving, or while wearing a tight collar
- Older patients with unexplained presyncope or falls.
- Negative cardiovascular and neurological investigations

**Contraindications**

- Present (past 3 months) myocardial infarction, transient ischemic attack, or stroke
- Presence of ventricular fibrillation, ventricular tachycardia, or carotid bruits.

**Box 5: Treatment protocol<sup>3</sup>****Education**

- Avoidance of triggering events
- Recognition of presyncopal symptoms and subsequent use of self help manoeuvres to avert syncope
- Class I recommendation; level of evidence C

**Volume expanders**

- Increased intake of salt and fluids through salt tablets or “sports” beverages
  - Class II recommendation; level of evidence B
- Moderate exercise training
- Class II recommendation; level of evidence B

**Tilt training**

- Progressively prolonged periods of enforced upright posture
- Class II recommendation; level of evidence B

**Drug therapy**

- Overall class II-III recommendation; level of evidence A-B
- $\beta$  blockers (class III; level A)
- Etilephrine ( $\alpha$ 1 agonist) (class III; level B)
- Modification or discontinuation of hypotensive drugs for comorbidities (class I; level C)
- Other agents—no recommendations due to lack of evidence of benefit of various drugs over placebo in several long term placebo-controlled, prospective trials

**Pacemaker treatment**

- Overall class I-II recommendation; level of evidence B
- Cardiac pacing in patients with cardioinhibitory or mixed carotid sinus syndrome (class I; level B)
- Cardiac pacing in patients with cardioinhibitory

Figure 1:

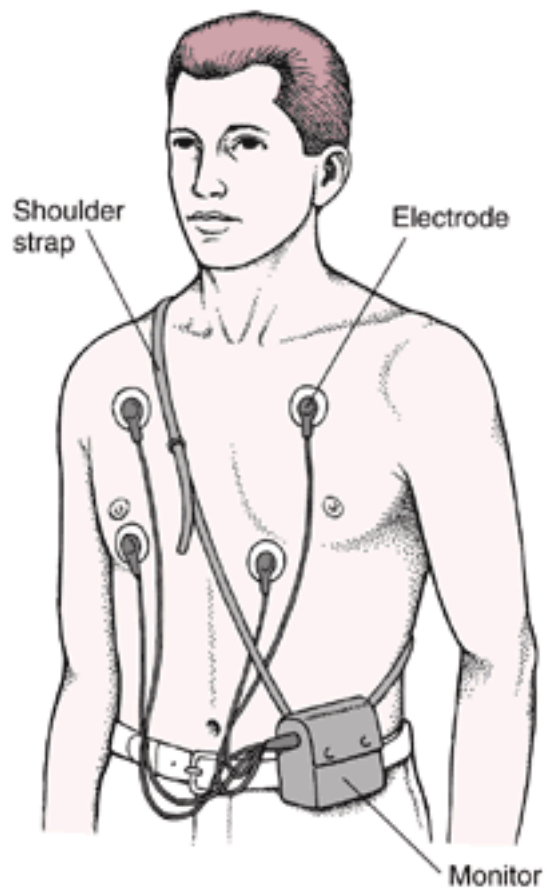


Figure 2:

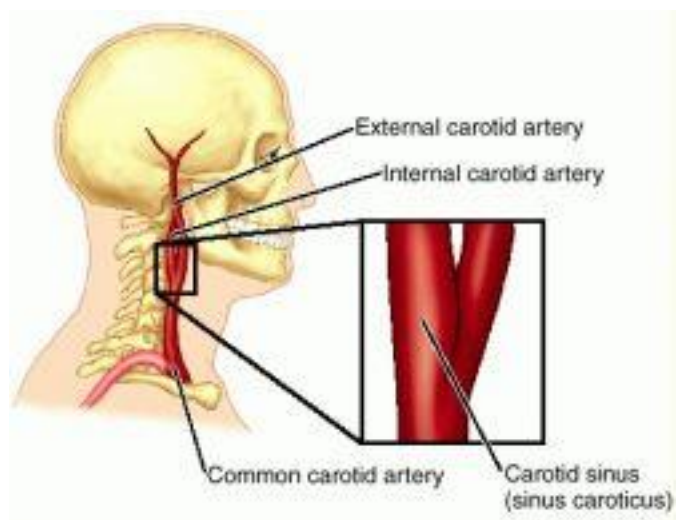


Figure 3:

