Epidemiology, Pathophysiology and Treatment of Different Types of Syncope: A Review

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Abstract: Syncope is a sudden transient loss of consciousness and motor control accompanied by loss of postural tone and followed by spontaneous recovery. A wide variety of cardiovascular disorders can cause an abrupt fall in cerebral perfusion that may manifest as recurrent or isolated episodes of syncope and presyncope, which is a feeling of lightheadedness or dizziness and near collapse not associated with loss of consciousness or motor control. The major causes of syncope are reflex syncope (60-65% of cases), psychogenic syncope (15-20% of cases), cardiac syncope (15-20% of cases), vascular syncope (2-3% of cases) and metabolic syncope (2-3% of cases). Syncope is a common medical problem and the prevalence of syncope increases with age from 0.7% in men aged 35-44 to 5.6% in men over the age of 75. This article focuses on the general overview of different types of syncope which includes common causes, epidemiology, pathophysiology, symptoms, signs and treatment of different types of syncope.

Key words: Syncope · Epidemiology · Loss of consciousness · Pathophysiology

INTRODUCTION

Syncope is a transient loss of consciousness due to inadequate blood flow to the brain. The mechanism of the syncope in susceptible individuals includes reflex cardiac standstill (always reversible) commonly following a surprising bump to the head. Syncope may be due to deficient blood flow resulting from peripheral circulatory failure, cerebral vascular accident (stroke), cardiac arrhythmia, transient cardiac standstill in Stokes-Adams syndrome (a condition in which blood flow to the brain is temporarily reduced because of an irregular heartbeat), altered blood chemistry as in hyperventilation or hypoglycemia. The various predisposing factors of syncope include fatigue, prolonged standing, nausea, pain, emotional disturbances, anemia, dehydration and poor ventilation [1].

Common Causes of Syncope: The major causes of syncope are reflex syncope which includes vasodepressor, vasovagal, orthostatic and vagotonic causes. The psychogenic or neurogenic syncope includes seizures and hysteric causes. The cardiac syncope includes dysrhythmias, obstructive and ischemic causes. The vascular syncope includes cerebrovascular accidents, transient ischemic attacks, migraines and subclavian steal syndrome. The metabolic syncope includes hypoglycemia, hypoxia and hyperventilation [2].

Diagnosis of the Cause of Loss of Consciousness: When loss of consciousness is temporary and recovers spontaneously it is referred to syncope. Temporary loss of consciousness is a result of a temporary reduction in the blood flow to the brain. This can lead to lightheadedness or a "black out" episode of loss of consciousness. There are many conditions which can temporarily impair the brain blood supply. Temporary loss of consciousness can be caused by heart conditions and conditions that do not directly involve the heart.

Temporary loss of consciousness is caused by conditions that do not directly involve the heart include:

- A shift in body position from lying or sitting to a more vertical position (postural hypotension)
- Dehydration
- Blood pressure medications

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Diseases of the nerves to the legs of the elderly

Diabetes

Parkinson's disease.

A decreased total blood volume or poor tone of the nerves of the legs from these conditions causes a disproportionate distribution of the blood in the legs, instead of up to the brain, when standing. Other common non-heart causes of temporary loss of consciousness include fainting after blood is drawn or after certain situational events (situational syncope) such as after urination, defecating and coughing. This occurs because of a reflex of the involuntary nervous system (vasovagal reaction) that leads to slowing of the heart rate and dilation of the blood vessels in the legs thus lowering the blood pressure. The result is that less blood (therefore less oxygen) reaches the brain as it is directed to the legs.

Epidemiology and Pathophysiology: Syncope is a common medical problem and accounts for approximately 3% of emergency room visits and 1-6% of hospital admissions. The prevalence of syncope increases with age from 0.7% in men aged 35-44 to 5.6% in men over the age of 75. In long-term care institutions, the annual incidence is approximately 6%. The elderly represent the population at greater risk for most causes of syncope. The young and middle-aged populations can have syncope from any of the mentioned causes, but reflex syncope is the most common syncope in the old age group. Underlying cardiac, seizure, vascular or metabolic problems are potential risk factors of syncope. The interruption of blood flow to the brain for longer than 8-15 seconds can lead to loss of consciousness [3].

Types of Syncope: The different types of syncope are summarized below [4]:

Vasovagal syncope: Vasovagal syncope is the common faint associated with a stress response of the autonomic nervous system which can either suddenly lower the pulse rate, blood pressure or both together. Vasovagal syncope results from vagal stimulation that leads to bradycardia, reduced cardiac output and decreased cerebral perfusion. This type of syncope is usually triggered by a reduction in venous return which occurs due to prolonged standing, excessive heat or a large meal. Some variants of vasovagal syncope occur in the presence of identifiable triggers (e.g. cough syncope, micturition syncope) and are collectively known as situational syncope.

Orthostatic syncope: Orthostatic or postural syncope results from a decrease in blood pressure with change to the upright position, usually with compensatory increase in pulse rate. This drop may be due to drugs especially diuretics, vasodilators, antihypertensives, antidepressants, antipsychotics, sedative and hypnotics that lower intravascular volume and peripheral resistance.

Vagotonic syncope: Vagotonic syncope is seen in several situations most commonly posttussive, postmicturition and following a Valsalva’s maneuver.

Symptoms and Signs of Reflex Syncope: Reflex syncope is heralded by vague nausea, warmth, sweating, clamminess, blurred vision and lightheadedness. Associated with these symptoms may be history of recent bad news, emotional distress, heavy coughing and urination. The patient experiencing a vasovagal type of reflex faint is very likely to feel nauseated and sweaty before fainting and often appears pale and feels clammy. The various signs of reflex syncope include confused, pale and restless state of patient. The patient may be diaphoretic and the pulse, which is initially rapid, is usually slow or unobtainable. Pulpillary dilation, yawning and hyperventilation also may occur. Unconsciousness is usually brief (seconds to a few minutes) and once the patient reaches the supine position.

Diagnosis and Treatment of Reflex Syncope: No single laboratory test will diagnose a common faint in the case of reflex syncope. If the history suggests reflex syncope and the physical examination is normal, no diagnostic studies are needed. A careful drug history, a search for underlying diseases and a carefully taken set of vital signs in the recumbent, sitting and standing position will
confirm the orthostatic syncope. Head-up tilt testing or Tilt table testing may help demonstrate these blood pressure changes and help reproduce the syncopal symptoms [5]. This test involves lying the patient on a table that is then tilted to an angle of 60-80 degrees for up to 45 minutes while the ECG and blood pressure are monitored which can be used to confirm the diagnosis. A positive test is the occurrence of a syncope episode with associated bradycardia or hypotension or both. Addition of sublingual nitroglycerin (400µg) has been shown to increase the total positivity rate to 70% with a specificity of 94%. Once syncope has occurred; cerebral perfusion can usually be restored by placing the patient supine with the legs elevated. Reflex syncope can usually be prevented by removing or avoiding precipitants (e.g., a hot, close environment or the upright position during painful procedures), by lying down or by placing the head between the knees during the presyncopal period. The treatment of orthostatic syncope depends on the underlying cause. Drug induced syncope requires modification or withdrawal of the agent, volume depletion requires fluid replacement and metabolic neuropathies often respond to treatment of the underlying cause [6]. The nonpharmacological measures in the treatment of orthostatic syncope includes careful assumption of the upright position, pressure stockings, alcohol abstention, avoidance of prolonged bed rest, contracting legs muscles before rising or standing for prolonged periods and increasing salt intake are helpful. If the above measures fail, then pharmacological measures are used. Fludrocortisone (sodium-retaining steroid) which reduces the vasodepressor response and relieves the symptoms of vasodepressor carotid sinus syndrome. Therapy should begin with 0.1 mg orally daily and can be increase to 0.3-0.4 mg daily with several weeks, but caution should be taken that this therapy is contraindicated in patients with congestive heart failure. Indomethacin, 25-30 mg three times a day with meals may improve symptoms by opposing the vasodilator effect of endogenous prostaglandins. Some other agents such as: metoprolol (50 mg orally twice daily); transdermal scopolamine (one 0.5- mg disk applied to the skin every 3 days); disopyramide (100 mg two capsules orally twice daily); theophylline (200 mg orally twice daily); dextroamphetamine (one 5-mg capsule orally each morning) and fluoxetine (20-mg orally daily) [7].

Disopyramide works through a combination of negative inotropic, anticholinergic and direct peripheral vasoconstrictive actions. Fluoxetine may help in preventing recurrent syncope when other therapies have failed. In patients with vasovagal syncope associated with bradycardia asystole, drug therapy such as: metoprolol, theophylline, or disopyramide is often effective in preventing syncope.

**Neurogenic or Psychogenic Syncope:** Neurological syncope is due to obstruction of vertebrobasilar artery and is associated with neurological signs. The neurological or psychogenic syncope includes seizures and hysterical causes.

Epilepsy is a chronic central nervous system disorder in which brief episodes of seizures appear with or without loss of consciousness [8]. It is characterized by excessive electrical discharge of certain neurons in the brain leading to body movements [9, 10]. Hysteria is the conversion of mental or emotional illness into physical symptoms. It produces syncope-like symptoms without true changes in the cardiovascular, central or automatic nervous system.

**Symptoms and Signs of Neurogenic or Psychogenic Syncope:** A seizure is associated with an abrupt loss of consciousness and their may be associated warning symptoms involving certain smell, sounds or visual cues. The patient will often be confused and disoriented for up to 30 minutes after the seizure.

In seizure disorders, eyewitness accounts are important and usually consists of the classic tonic-clonic movement disorder, incontinence and postictal phase. The patient is often fatigue groggy or confused and may smell of urine. Patients with hysterical syncope often presents with signs of normal pulse, skin color and blood pressure.

**Treatment of Neurogenic or Psychogenic Syncope:** The epileptic seizures are nearly correlated with abnormal and excessive discharges in electroencephalogram (EEG). Electroencephalography and computerized tomography (CT) are useful studies in patients with new-onset seizures. If hysteria is suspected, counseling or psychiatric referral is recommended. Treatment of the underlying seizure disorder should control further seizures. Standard anticonvulsants may be used to control seizures.

**Cardiac Syncope:** Cardiac syncope is sudden loss of consciousness, with momentary premonitory symptoms or without warning which occur due to cerebral anemia.
caused by obstructions to cardiac output. The cardiac syncope includes dysrhythmias, obstructive and ischemic causes [11].

**Dysrhythmias:** include bradyarrhythmias, tachyarrhythmia, heart block and sick sinus syndrome cause syncope through a decrease in cerebral perfusion.

**Bradyarrhythmias:** Bradyarrhythmias are usually sudden or abrupt in onset with transient asystole or severe bradycardia and profound decreases in cerebral perfusion.

**Tachyarrhythmias:** The onset of syncope in cases of tachycardia depends on the rate (usually symptomatic at > 160 beats per minute) in addition to how well the cardiac output is maintained.

**Heart Block and Sick Sinus Syndrome:** Heart block and sick sinus syndrome can lead to syncope if there is 5 seconds or more of ineffective systoles.

**Obstructive Syncope:** The structural abnormalities of aortic stenosis, asymmetric septal hypertrophy, atrial myxoma and mitral valve prolapse interfere with effective cardiac output and are often worsened with exercise. In this syncope, a pulmonary embolus must be large enough to decrease effective pulmonary circulation and oxygenation.

**Cardiac Ischemic Events:** Syncope may occur with acute myocardial infarction (MI) or pump dysfunction. Coronary vasospasm or severe angina with resultant transient dysrhythmias or hypotension can also lead to syncope.

**Symptoms and Signs of Cardiac Syncope:** The dysrhythmias usually cause an abrupt loss of consciousness and warning symptoms seldom occur, unless associated with angina or myocardial infarction. Bradyarrhythmias usually produced syncope within 12-15 seconds when the patients is supine but within 5-6 seconds when the patient is upright. Patients who are experiencing tachyarrhythmias usually present with a sensation of palpitation, chest pain or pressure rather than abrupt loss of consciousness. The obstructive syncope is usually associated with exertion which may be abrupt or gradual. Syncope in the setting of ischemia usually has preceding symptoms of acute angina or myocardial infarction. The various sign of cardiac syncope are consistent with poor perfusion and rapid or slow pulse, depending upon underlying dysrhythmias.

**Treatment of Cardiac Syncope:** Patients believed to have cardiac syncope should be hospitalized and undergo cardiac monitoring, especially elderly individuals with a prior history of cardiac disease (i.e., coronary artery disease, congestive heart failure, valvular heart disease, obstructive cardiomyopathy) or those persons who experience sudden syncope without warning. All of these patients should receive an ECG. Patients with a normal initial ECG are unlikely to have an underlying dysrhythmia or experience sudden death. Ambulatory cardiac monitoring with continuous-loop ECG recorders and echocardiography may be utilized if infrequent dysrhythmias or obstructive lesions are suspected. The continuous-loop ECG recorder is useful for evaluating recurrent syncope. An exercise stress test may help identify dysrhythmia which was not seen on ambulatory cardiac monitoring. Cardiac angiography may be needed to help clarify ischemic or anatomic abnormalities [12]. Drug therapy or withdrawal may suffice for many bradyarrhythmias and tachyarrhythmias and surgery may be indicated for structural or ischemic conditions. A demand pacemaker is indicated only when heart block or severe bradycardia has been proven responsible for the syncope [13, 14].

**Vascular Syncope:** Vascular syncope includes cerebrovascular accidents, transient ischemic attacks, migraines and hyperventilation. Cerebrovascular disease is rarely the cause of a faint. Perhaps, subclavian steal is the best example in this class, but it is extremely uncommon. Loss of consciousness may occur during a vertebrobasilar transient ischemic attack (TIA), but not without accompanying evidence of brain stem dysfunction such as cranial nerve deficits (e.g., diploplia, swallowing difficulties, dysarthria), paresis or ataxia. Their absence during a spell of unconsciousness virtually rules out TIA as a cause.

Vascular syncope is not a common finding in transient cerebral ischemia or cerebral vascular accidents unless severe bilateral disease or coexistent brain stem ischemia occurs. Arm exercise decreases the muscle bed’s peripheral resistance and shunts blood from the vertebral artery, leading to cerebral ischemia and eventual syncope.

**Symptoms and Signs of Vascular Syncope:** It is a rare for a cerebral vascular accidents or transient ischemic attacks to cause syncope, unless there is significant reticular activating system involved. Subclavian steal syndrome, on the other hand, can lead to significant posterior brain stem ischemia with symptoms consisting of diploplia, dysarthria, dysphagia, dysesthesia and dizziness during
peak arm exercise activities. Focal neurological deficits may or may not be present following cerebral vascular accidents, transient ischemic attacks or migraine.

**Treatment of Vascular Syncope:** Head computerized tomography and carotid and vertebral artery studies can be helpful in patients suspected of having a vascular cause for their syncope. Subclavian vascular studies are warranted if subclavian steal is suspected. If test results indicate a stenotic lesion of 75% or greater in the carotid system, surgery should be contemplated. If the posterior circulation is involved, aspirin (300-1300 mg per day) or anticoagulants may be necessary. Surgery is indicated for symptomatic proximal subclavian artery occlusion [15].

**Metabolic Syncope:** Metabolic syncope includes hypoglycemia, hypoxia and hyperventilation. Metabolic entities are more prone to alter consciousness than to cause true syncope, but they must be kept in mind, as they are treatable.

**Symptoms and Signs of Metabolic Syncope:** Restlessness, confusion and anxiety are common signs and symptoms of metabolic syncope as in hypoxia or hypoglycemia. Syncope can occur in any position. Pupils may be dilated in hypoxia and carpopedal spasm may accompany in hyperventilation. In hyperventilation, the patient often relates a sensation of smothering or shortness of breath in conjunction with circumoral, facial and extremity paresthesias.

**Treatment of Metabolic Syncope:** Arterial blood gas and blood sugar can be obtained if the physician suspects hypoxia or hypoglycemia as the cause of the patient’s syncope. Hypoglycemia should be treated immediately with one or two ampules of 50% dextrose in water intravenously; the underline caused must then be determined and treated reasons for underlying hypoxia and hyperventilation should be investigated as appropriated and treatment should be instituted [16].

**CONCLUSION**

The review is aimed at different types of syncope, their epidemiology, pathophysiology and treatment. The major causes of syncope are reflex syncope, neurologic syncope, cardiac syncope, vascular syncope and metabolic syncope. The complaint of syncope is common and presents a challenging diagnostic exercise since the causes range from the trivial to the serious. Attempt is made in above review article to enumerate different general measures, prevention and treatment (both non pharmacological and pharmacological) and other diagnostic criteria for management of different types of syncope.

**REFERENCES**