Reflex Syncope & Orthostatic Intolerance Syncope

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Reflex syncope is the most frequent cause of syncope. The pathophysiology of reflex syncope is a transient loss of consciousness due to a neural-mediated reflex with systemic vasodilatation and/or bradycardia, leading to systemic hypotension and cerebral hypoperfusion. Major types of reflex syncope include vasovagal syncope, situational syncope and carotid sinus syncope.

Orthostatic hypotension is a postural decrease in systolic blood pressure of at least 20 mmHg, or in diastolic blood pressure of at least 10 mmHg, leading to dizziness or syncope. The pathophysiology of orthostatic hypotension consists of primary or secondary autonomic dysfunction and volume depletion.

The purposes of clinical assessment in patients presenting with syncope include identification of the underlying cause and risk stratification. Besides history, physical examination, electrocardiogram and echocardiography, tilt table test with sublingual nitroglycerin or intravenous infusion of isoproterenol may be considered in patients with clinically suspected reflex syncope or orthostatic hypotension.

To avoid profound syncope, patients who develop syncope or other symptoms associated with vasovagal reflex or orthostatic hypotension should assume the supine position with legs raised at the onset of symptoms. However, for now, no single therapy has been proven effective for recurrent vasovagal syncope. Explanation of risk and provision of reassurance are indicated in all patients with vasovagal syncope. In patients with recurrent vasovagal syncope, physical isometric counterpressure manoeuvres (PCM) may be considered, and cardiac pacing should also be considered in patients with dominant cardioinhibitory reflex syncope. Adequate hydration and salt intake is the most effective therapy in patients with orthostatic hypotension. However, midodrine, fludrocortisones and PCM should also be considered if hydration and salt intake are not adequate.