Transient loss of consciousness

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Abstract

Transient loss of consciousness (T-LOC) is usually caused by cardiovascular (syncope), neurological (seizure) and psychological (non-epileptic attack disorder) conditions. Suspected cardiovascular causes should be further defined as reflex/blood pressure regulatory or cardiac/heart rhythm disorders. Identifying select individuals at high risk of sudden death from a large cohort of patients with more benign causes of T-LOC is a major challenge. The key to assessing a patient with T-LOC lies in a detailed history. Risk stratification into patients at high and low risk of future cardiac arrest should be an integral part of the initial assessment of every T-LOC patient. Risk stratification is easily performed by considering the presence or absence of structural heart disease and family history of sudden unexplained death below 40 years of age, and by systematic analysis of a 12-lead electrocardiograph. Patients with high-risk features whose T-LOC is thought to be cardiovascular in origin should be referred to a heart rhythm specialist for urgent assessment. In these cases, T-LOC is an opportunity to intervene with highly effective therapies before a cardiac arrest occurs.

Keywords Arrhythmia; cardiac arrest; cardiomyopathy; channelopathy; electrocardiogram; implantable cardioverter defibrillator; inherited cardiac conditions; sudden cardiac death; syncope; transient loss of consciousness

Key points

- Transient loss of consciousness (T-LOC) can be caused by cardiovascular (syncope), neurological (seizure) or psychological (non-epileptic attack disorder) causes
- Risk stratification into those at high risk and low risk of future cardiac arrest is easy based on history and 12-lead ECG analysis, and should be a standard component of the initial assessment of every patient with T-LOC
- The challenge is to identify a minority of high-risk individuals from the larger number of patients with benign causes of T-LOC
- T-LOC in high-risk patients should be considered an opportunity to intervene and, if appropriate, offer effective treatments to prevent future death from cardiac arrest
- Patients with T-LOC at low risk of sudden death in whom a diagnosis has not been established can at least be reassured that their condition is benign and not life-threatening

Introduction

Transient loss of consciousness (T-LOC) is defined as abrupt complete loss of consciousness that is transient, self-limiting and not caused by head trauma. T-LOC is a subset of a much larger cohort of patients presenting acutely with collapse of unknown cause. Defined in this manner, the causes of T-LOC are limited to cardiovascular (syncope), primary neurological (seizure/epilepsy) and psychogenic (non-epileptic attack disorder) causes. Syncope is T-LOC caused by cerebral hypoperfusion. It is characterized by both loss of consciousness and loss of postural tone. T-LOC is highly prevalent, affecting up to 50% of the general population at some stage. Its importance lies in the clinical challenge associated with diagnosis and the fact that a proportion of T-LOC patients are at high risk of sudden death, which is usually both predictable and preventable.

Approximately 12% of all natural deaths result from out-of-hospital cardiac arrest.[1] A significant minority of these patients will have experienced syncope before cardiac arrest. A challenge for clinicians is to identify the high-risk individual from the large number of patients with benign causes of T-LOC, and to intervene effectively to prevent future sudden death. This article summarizes the clinical features associated with different types of syncope and emphasizes that risk stratification should become an integral part of clinical assessment.

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Clinical assessment

The clinician's first task is to confirm that the patient has had an episode of T-LOC and to exclude other conditions, such as metabolic disorders, intoxications, falls and coma, which can present to an emergency department or rapid-access T-LOC clinic. The key to assessing the patient is taking a detailed, methodical history (Table 1).

Features in the history are used to differentiate events that are primarily cardiovascular (syncope) from those that are primarily neurological (seizure). Cardiovascular events are generally preceded by prodromal symptoms (dizziness, lightheadedness, tunnel vision) culminating in loss of consciousness, during which eyewitnesses notice the patient to be pale and either motionless or exhibiting coarse asymmetrical jerking movements (myoclonic jerks secondary to cerebral hypoxia). The patient becomes oriented soon after regaining consciousness. Neurological events are characterized by either a lack of prodrome or a stereotypical aura culminating in loss of consciousness, during which the patient has tonic—clonic movements of all four limbs. Patients remain confused for a longer period after regaining consciousness than is the case after a cardiovascular event.

If the history suggests a cardiovascular cause of loss of consciousness (syncope), further questioning is needed to differentiate reflex/blood pressure regulatory problems from cardiac/heart rhythm disorders. Reflex forms of syncope frequently have an identifiable trigger (e.g. prolonged standing) and autonomic-mediated prodromal features (nausea caused by vagal activation, sweating from sympathetic activation), and result in post-

History-taking when assessing a patient with transient loss of consciousness (T-LOC)

A detailed history focussing on prodromal features, an eyewitness account of the event itself and features during recovery can be used to differentiate cardiovascular from primary neurological causes of T-LOC. Cardiovascular causes can be divided into blood pressure regulatory problems (reflex syncope, postural hypotension) and cardiac/heart rhythm causes (mechanical obstruction, bradycardia, tachycardia). Postural hypotension, not covered in this table, occurs while standing, is frequently associated with symptoms of presyncope on assuming an upright posture and is best diagnosed at the time of presentation by recording a >20 mmHg drop in systolic blood pressure between lying and standing, or by recording a systolic blood pressure <90 mmHg

	Reflex	Cardiac	Seizure
Trigger	Common (e.g. standing)	Rare	Rare
Prodrome	Common	Uncommon or brief	Aura
Autonomic activation	Yes (nausea, sweating)	No	No or rare
Onset	Gradual	Sudden	Sudden
Colour	Pale	Pale	Normal, red, blue or pale
Convulsive jerks	None or brief	None or brief	Common/prolonged
Incontinence	Uncommon	Uncommon	Common
Tongue-biting	Uncommon	Uncommon	Common (lateral tongue)
Duration	Brief	Variable	Variable
Post-event confusion	Rare	Rare	Common
Post-event fatigue	Common	Rare	Common

Table 1

event fatigue that can last many hours. Indeed, in patients with a convincing history of syncope, nausea as a symptom gives the clinician great reassurance that the pathophysiological mechanism is benign rather than life-threatening. In contrast, cardiac/heart rhythm causes of syncope have no identifiable trigger, a brief or often absent prodrome and little evidence of autonomic activation, and individuals recover quickly with no post-event confusion or fatigue.

Risk stratification

Establishing a clinical diagnosis in some patients with T-LOC can be difficult. In contrast, stratifying all patients with T-LOC into those at low risk and high risk of sudden death is easy and should become a standard part of the initial assessment of every T-LOC patient. To stratify risk, assess whether the patient has a personal history of structural heart disease or a family history of an inherited cardiac condition, and consider relevant 12-lead electrocardiograph (ECG) abnormalities.

Structural heart disease

Patients with a history of myocardial infarction or congestive heart failure and unexplained syncope are at high risk of future cardiac arrest. A new diagnosis of heart failure can be considered in patients with exertional dyspnoea who have noticed a reduction in their exercise capacity in recent months. Less common forms of significant structural heart disease can usually be suspected by eliciting abnormal physical signs. These include a displaced apex beat (dilated cardiomyopathy), a thrusting apex beat (hypertrophy) or a systolic murmur caused by left ventricular outflow tract obstruction (aortic stenosis, hypertrophic cardiomyopathy). In contrast, patients with excellent exercise capacity are unlikely to have significant structural heart disease.

The most common cause of structural heart disease in developed societies is atherosclerotic coronary artery disease. Over many years, this results in coronary artery occlusion and myocardial infarction. Infarction permanently scars the ventricle, and the risk of a cardiac arrest and sudden death from ventricular tachycardia (VT) and ventricular fibrillation (VF) is directly proportional to the amount of ventricular scarring. This relationship is now so well established that consideration is given to use of an implantable cardioverterdefibrillator (ICD) in all patients with heart failure and an ejection fraction less than 35%. Sudden cardiac death due to pulseless VT or VF accounts for 12% of all natural deaths and 50% of all cardiovascular deaths.[1] Some patients are fortunate that scar-related VT is self-terminating, resulting in syncope rather than out-of-hospital cardiac arrest. This life-threatening event should be recognized by the medical community as an opportunity to intervene and prevent a future death from cardiac arrest. Untreated syncope secondary to VT in patients with significant structural heart disease is associated with 20–30% fatality within 2 years.[2] UK national guidelines recommend that patients with unexplained syncope and significant structural heart disease should be considered to have a life-threatening event until proved otherwise.

Family history of sudden death below 40 years of age

Inherited cardiac conditions that predispose to sudden arrhythmic death include cardiomyopathies (hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, familial dilated cardiomyopathy) and channelopathies (congenital long QT syndrome, Brugada syndrome, catecholaminergic polymorphic VT, short QT syndrome). These are inherited in an autosomal manner with variable phenotype penetrance.

A family history should be obtained tactfully in a way that does not cause anxiety in patients with undiagnosed T-LOC, most of whom have a benign aetiology. I usually enquire about the age and health of each parent and sibling, and the cause of any premature deaths. For completeness, I discreetly ask about any tragedies such as drownings or single-vehicle road traffic accidents in the more extended family. Patients with unexplained