Transient loss of consciousness

Peter O'Callaghan

Abstract

Transient loss of consciousness (T-LOC) is caused by cardiovascular (syncope), neurological (seizure) and psychological (non-epileptic attack disorder) conditions. Suspected cardiovascular causes should be further defined as either reflex/blood pressure regulatory or cardiac/arrhythmic disorders. Identifying select individuals at high risk of sudden death from amongst 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 taking a detailed history. Risk stratification into those at high and low risk of future cardiac arrest should be a standard part of the initial assessment of every T-LOC patient. Risk stratification is easily performed by considering the presence/absence of structural heart disease, family history of sudden unexplained death under 40 years of age and by systematic analysis of 12-lead ECG. Patients with high-risk features in whom 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; inherited cardiac conditions; syncope; sudden cardiac death; transient loss of consciousness

Introduction

Transient loss of consciousness (T-LOC) is defined as abrupt complete loss of consciousness, which is transient and self-limiting and not due to head trauma.[1] T-LOC is a subset of a much larger cohort of patients presenting acutely with collapse of unknown cause. When defined in this strict manner the causes of T-LOC are limited to cardiovascular causes (syncope), primary neurological causes (seizure/epilepsy) and psychogenic causes (non-epileptic attack disorder). Syncope is T-LOC due to cerebral hypoperfusion. It is characterized by both loss of consciousness and loss of postural tone. T-LOC is very prevalent, affecting up to 50% of the general population at some stage in their life.[2] 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 are due to out-of-hospital cardiac arrest.[3] A significant minority of these patients will have had experienced syncope before cardiac arrest. A challenge for clinicians is to identify the high-risk individual from amongst a large number of patients with benign causes of T-LOC, and to intervene effectively to prevent future sudden death. This chapter will summarize the clinical features associated with different types of syncope and will emphasize that risk stratification should become an integral part of the clinical assessment.

Peter O'Callaghan *MB FRCP is a Consultant Cardiologist in the Department of Cardiology, University Hospital of Wales, Cardiff, UK.*
Competing interests: none declared.

Clinical assessment

The first task the clinician faces 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 one may be asked to assess in an emergency department or rapid-access T-LOC clinic. The key to assessing a patient with T-LOC lies in 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 in appearance and either motionless or exhibiting coarse asymmetrical jerking movements (myoclonic jerks secondary to cerebral hypoxia). The patient is oriented soon after regaining consciousness. Neurological events are characterized either by no prodrome or a stereotypical aura culminating in loss of consciousness, during which the patient has tonic–clonic movements of all four limbs. The patient remains 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/arrhythmic disorders. Reflex forms of syncope frequently have an identifiable trigger (e.g. prolonged standing) and autonomic-mediated prodromal features (nausea due to vagal activation and sweating due to sympathetic activation), and result in post-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/arrhythmic causes of syncope have no identifiable trigger and little evidence of autonomic activation, and recover quickly with no post-event confusion or fatigue.

Risk stratification

Establishing a clinical diagnosis in some patients with T-LOC can be an extremely difficult and challenging conundrum. In contrast, stratifying risk in all patients with T-LOC into those at low risk and those potentially at 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 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

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

A detailed history focussing on prodromal features, an eye-witness account of the event itself and features during recovery can be employed to differentiate cardiovascular from primary neurological causes of T-LOC. Cardiovascular causes can be divided into blood pressure regulatory problems (reflex syncope and postural hypotension) and cardiac causes (bradycardia, tachycardia and mechanical obstruction). 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 systolic blood pressure drop between lying and standing or by recording a systolic BP <90 mmHg

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

Table 1

forms of significant structural heart disease can usually be suspected by eliciting abnormal physical signs, such as a displaced apex beat (dilated cardiomyopathy), a thrusting apex beat (hypertrophy) or a systolic murmur due to left ventricular outflow tract obstruction (aortic stenosis and hypertrophic cardiomyopathy). In contrast, patients with excellent exercise capacity are unlikely to have significant structural heart disease.

The commonest cause of structural heart disease in Western societies is atherosclerotic coronary artery disease. Over a period of many years this results in coronary artery occlusion and myocardial infarction (MI). Infarction permanently scars the ventricle, and the risk of a cardiac arrest and sudden death due to ventricular tachycardia and ventricular fibrillation is directly proportional to the amount of ventricular scarring. This relationship is now so well established that consideration is given to the implantation of defibrillators (ICDs) in all post-MI survivors with an ejection fraction lower than 30%.[4] Sudden cardiac death due to pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF) accounts for 12% of all natural deaths and 50% of all cardiovascular deaths.[3] Some patients are fortunate that scar-related VT self-terminates, 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.[5] National guidelines in the UK recommend that patients with unexplained syncope and significant structural heart disease should be considered to have a life-threatening event until proved otherwise.[6]

Family history of sudden death under 40 years old

Inherited cardiac conditions that predispose to sudden arrhythmic death include cardiomyopathies (hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and familial dilated cardiomyopathy) and channelopathies (congenital long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia and short QT syndrome). These conditions are all 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, the majority of whom will 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 then ask about any tragedies such as drownings or single-vehicle road traffic accidents in the more extended family. Patients with unexplained T-LOC and a family history of sudden death under 40 years old should be assessed as a matter of urgency by a cardiologist who specializes in heart rhythm disorders.[6]

Absence of a family history does not exclude an inherited arrhythmic condition. In fact, up to 25% of cases can be due to new sporadic mutations.[7] Neurologists should constantly be alert to the possibility that patients with conditions such as congenital long QT syndrome may be referred to their clinics and be prepared to pick up relatively easily the more obvious cases with ECG abnormalities.[7]

Relevant 12-lead ECG abnormalities

The importance of ECG abnormalities in the assessment of patients with T-LOC is stressed in the National Institute for Health and Clinical Excellence T-LOC guidance 2010 (red flags)[2,8] or prevented in 99.9% of patients by ICD implantation.[9] An approach that seeks pro-actively to diagnose or rule out specific conditions will usually result in a greater diagnostic yield rather than the simple inspection of the ECG (Figures 1 and 2). In patients aged 40 years or over, the emphasis should be on excluding acquired cardiac conditions, such as ventricular scarring (e.g. pathological

