Convulsive syncope

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Introduction

This article includes discussion of convulsive syncope, anoxic seizure, hypoxic convulsion, and reflex anoxic seizure. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

In this article, the author provides an overview of convulsive syncope. Syncope is commonly accompanied by tonic or myoclonic muscle activity, eye deviations, automatisms, vocalizations, and hallucinations, which may all complicate the distinction from epileptic seizures. Differential diagnosis is based on the specific features and not the mere presence or absence of these phenomena. Recognition of syncope depends also on accurate information about premonitory symptoms and postictal events. Investigations such as tilt testing or creatine kinase levels may be helpful but are never diagnostic in isolation. EEG should not be ordered routinely. An increasingly recognized and treatable cause of syncope is ictal asystole or bradycardia during temporal lobe seizures.

Key points

- Syncope is often accompanied by tonic or myoclonic muscle activity.
- Myoclonus is usually brief, arrhythmic, and multifocal.
- Specific provocation and rapid reorientation helps to distinguish syncope from generalized tonic clonic seizures.

Historical note and terminology

Syncope is defined as a brief loss of consciousness and upright posture due to global cerebral hypoxia. The term “convulsive syncope” specifies a common variant of syncope that is accompanied by tonic or myoclonic activity.

Animal experiments on convulsive syncope date back to the middle of the 19th century, when Kussmaul and Tenner showed that anoxic convulsions occur after ablation of the telencephalon but not after destruction of the brainstem (Dell et al 1961). A century later, Dell and colleagues demonstrated that motor activation in the early stages of cerebral hypoxia resulted from the combined effects of direct hypoxic activation of the brainstem reticular formation and cortical suppression with subsequent reticular disinhibition (Dell et al 1961). Gastaut and colleagues were the first to conduct studies on the clinical and electroencephalographic features of experimentally induced syncope in human subjects (Gastaut and Fischer-Williams 1957). They observed tonic and myoclonic phenomena during syncope in the absence of epileptic EEG activity and coined the term “convulsive syncope."

Clinical manifestations

Presentation and course

Convulsions are an integral component of the brain's response to hypoxia. In experimental series, they represent the rule rather than the exception. Whether or not syncope manifests with convulsions depends on the degree of cerebral hypoxia (Passman et al 2003). Experimental syncope has been induced by various means such as the Valsalva maneuver, a combination of hyperventilation and Valsalva, exposure to acceleration on a centrifuge, venipuncture and blood loss, ocular compression, and ventricular arrhythmia; but the clinical phenomenology has proven to be consistent, irrespective of the procedure. Reported frequencies of syncopal convulsions vary from 12% (Lin et al 1982) to 100% (Rossen et al 1943); when film or video recordings were employed, they were observed in the order of 70% to 90% (Duvoisin 1962; Aminoff et al 1988; Whinnery and Whinnery 1990; Lempert et al 1994a). However, syncopal convulsions may be overlooked because of their fleeting nature and variable intensity. In clinical case series, convulsions have been witnessed in 5% to 12% of patients with vasovagal (neurally mediated) syncope and in 15% of patients with various causes of syncope (Alboni et al 2001; Graham and Kenny 2001; Sheldon et al 2002). Syncope induced by head-up tilt was found to be complicated by convulsions in 7% of adult patients in a retrospective study (Passman et al 2003) and in 12% of prospectively studied pediatric patients (Fernandez Sanmartin et al 2003). In a video-based series, 53% of patients had involuntary movements during tilt-induced syncope (LaRoche et al 2011).

Another video study in adolescents undergoing tilt testing documented myoclonus in 59%, facial grimacing in 40%, and vocalizations in 39% (Heyer et al 2016). Carotid sinus syncope during carotid angioplasty was convulsive in 79% (Martinez-Fernandez et al 2007). Convulsive syncope may be more common with an underlying cardiac arrhythmia rather than a vasovagal or orthostatic origin (Del Rosso et al 2005; Kanjwal et al 2009; MacCormick et al 2011).

Syncopal myoclonus may manifest itself as anything from a single twitch of the mouth to a storm of violent jerks affecting the entire body. It is often multifocal with asynchronous muscle jerks in different parts of the body.{embed="pagecomponents/media_embed" entry_id="8164"}{embed="pagecomponents/media_embed" entry_id="8165"} Alternatively, the syncope may be generalized with a few jerks of bilateral synchronous muscle activation.{embed="pagecomponents/media_embed" entry_id="8166"}Both forms of myoclonus may occur during an attack. Proximal and distal muscles are equally affected, and facial involvement is common. In contrast to epileptic muscle activity, syncopal myoclonus is not rhythmic and is only rarely sustained for more than half a minute (Lempert et al 1994a).

Tonic muscle activity during syncope typically consists of head and body extension with either flexion or extension of the arms and sometimes clenching of the fists (Gastaut and Fischer-Williams 1957; Duvoisin 1962; Lin et al 1982; van Dijk et al 2014; Heyer et al 2016). The intensity of the tonic component appears to be most pronounced in asystolic syncope with total cessation of cerebral blood flow. Brief but intense opisthotonic stiffening is also a common accompaniment of breath-holding attacks and other forms of childhood syncope (Stephenson 1991; Breningstall 1996; Fernandez Sanmartin et al 2003). When tonic body extension starts early in the course of syncope, a stiff fall may ensue rather than the usual flaccid sinking to the ground.{embed="pagecomponents/media_embed" entry_id="8167"}

In addition to these elementary motor phenomena, syncope may also produce more complex movements (Stephenson 1990; Lempert et al 1994a; van Dijk et al 2014; Heyer et al 2016), including lip-licking, chewing, fumbling, reaching for the head, head turns, head raising, and sitting up or standing up while still being unresponsive and amnesic.{embed="pagecomponents/media_embed" entry_id="8168"} These movements are mostly short and solitary rather than repetitive. Vocalizations in the form of moaning or growling are common.

Eyes are usually open during syncope (Lempert et al 1994a; van Dijk et al 2014). Syncope often starts with downbeating nystagmus, sometimes with a horizontal component which tends to be missed in clinical settings (Stephenson 1990; Lempert and von Brevern 1996; Choi et al 2015). The most consistent ocular motor sign is an upward turning of the eyes early in the course of syncope, which may be followed by a lateral eye deviation (Lempert and von Brevern 1996).{embed="pagecomponents/media_embed" entry_id="8169"}

Hallucinations occur in both convulsive and nonconvulsive syncope. They are usually ignored if doctors do not ask and patients do not volunteer the information. Systematic studies, however, have uncovered the hallucinations with considerable regularity (Duvoisin 1962; Forster and Whinnery 1988; Lempert et al 1994a; Chiesa et al 2011). In 1 study, 60% of subjects experienced dreamlike hallucinations, which were always visual and often also auditory. In some subjects, visual hallucinations were restricted to a perception of gray haze, colored patches, or glaring lights. Others encountered more complex scenes involving landscapes, familiar situations, or people. A few individuals had out-of-body experiences. Auditory hallucinations included rushing and roaring sounds, traffic and machine noises, talking and screaming human voices, but never intelligible speech. Commonly, the emotional experience of syncope was described as detached, weightless, and peaceful, and several subjects were reluctant to return to reality (Lempert et al 1994b).

Prognosis and complications

The recurrence rate of syncope is around 35% at 1 year. Mortality is increased in patients with cardiac syncope but not in those with vasovagal (neurally mediated) syncope or syncope of unknown origin (Ungar et al 2011). Complications of syncope include self-injury due to unprotected falls and traffic accidents.

Biological basis

Localization

Nothing suggests that syncopal convulsions reflect epileptic activity of the cerebral cortex. Rather, muscle activation during syncope is subcortical and originates from abnormal firing of the reticular formation in the lower brainstem as a

consequence of hypoxic activation of reticular neurons mediated by chemoreceptors and release from cortical inhibition (Dell et al 1961). Microelectrode recordings from experimental animals exposed to total brain ischemia showed an increase of neuronal activity in the medullary reticular formation lasting up to 40 seconds, whereas cerebral cortex potentials ceased after 10 seconds (Naquet and Fernandez-Guardiola 1961).

Pathophysiology

During both convulsive and nonconvulsive syncope, the EEG shows a sequence of generalized slow waves of high amplitude, flattening of the trace, and the return of slow waves before normal background activity is restored (Gastaut and Fischer-Williams 1957; Brenner 1997). These changes are uniform and independent of the mechanism of syncope as they reflect the common final pathway of global cerebral hypoxia. A flat EEG reflects more profound cerebral hypoxia, and milder forms of syncope may present with isolated slowing (van Dijk et al 2014; Heyer et al 2016). Loss of consciousness and convulsions are related to the appearance of slow-wave activity, whereas tonic posturing, vocalizations, and slow horizontal eye movements usually appear when the EEG has flattened. Epileptic discharges are consistently absent both on ictal and interictal recordings (Gastaut and Fischer-Williams 1957; Aminoff et al 1988; Heyer et al 2016).

Differential diagnosis

Seizure phenomenology

Syncope is often misdiagnosed as epilepsy. Between 10% and 25%of patients referred to epilepsy clinics turn out to suffer from recurrent syncope (Petkar et al 2012; Xu et al 2016). The differential diagnosis of convulsive syncope includes generalized tonic-clonic seizures, complex partial seizures, psychogenic seizures, and concussive convulsions.

The most common problem in clinical practice is differentiation from generalized tonic-clonic seizures. One should remember that it is not the occurrence of tonic or myoclonic activity per se, but its specific phenomenology that discriminates between the two. In contrast to epileptic muscle activity, syncopal myoclonus is arrhythmic; it is more commonly multifocal than generalized, and it is only rarely sustained for more than half a minute. Tonic muscle activity is often absent or mild, a distinguishing feature that can be obtained from an eyewitness reliably (Thijs et al 2008).The eyes are open and often deviated both in syncope and epileptic seizures. Unlike syncopal eye turns, however, epileptic eye deviations tend to last longer than just a few seconds.

Asking about premonitory symptoms may aid in the differential diagnosis. Characteristic aura symptoms of syncope include bilateral tinnitus, decreased hearing, and "blackening-out," a transient amaurosis while consciousness is still preserved that is caused by the early collapse of retinal perfusion. Lightheadedness, confusion, abdominal discomfort, warmth, and faintness are equally common but less specific as patients may also use these terms to describe an epileptic aura or the sensation that precedes a psychogenic seizure. Some well-known epileptic aura phenomena such as tastes, smells, déjà-vu experiences, speech disturbances, and unilateral paresthesia do not occur before syncope (Benke et al 1997). Palpitations, although not specific for syncope, are suggestive of a tachycardia that compromises cardiac output.

Several postictal features may be equally useful to discriminate between syncope and an epileptic seizure. An important factor is postictal confusion as observed by an eyewitness (Hoefnagels et al 1991). Reorientation is usually immediate in syncope and does not exceed 30 seconds even after extended attacks (Aminoff et al 1988). Thus, any postictal disorientation lasting longer than 30 seconds suggests an epileptic seizure. Tongue bites point likewise to an epileptic event, but there are rare exceptions to this rule (Hoefnagels et al 1991; Lempert et al 1994a; Brigo et al 2012). In contrast, urinary incontinence and head injuries appear to be common both in syncope and generalized tonic-clonic seizures (Hoefnagels et al 1991; Brigo et al 2013). Exhaustion, sleepiness, vomiting, headaches, and muscle aches may all occur after syncope, but they tend to be more frequent and severe after generalized tonic-clonic seizures.

Table 1. Distinctive Features of Syncope and Generalized Tonic-Clonic Seizures

	Syncope	Generalized tonic-clonic seizures
Duration	Usually less than 30 seconds	1 to 2 minutes

Precipitation event	50%	None
Falls	Flaccid or stiff	Stiff
Convulsions	80% mostly brief, arrhythmic, multifocal or generalized Usually little or no tonic activity	Always 2 to 3 minutes, rhythmic, generalized Intense tonic stiffening
Eyes	Open, transient upward or lateral deviation, downbeat nystagmus at onset	Open, often sustained deviation
Hallucinations	Late in the attack	May precede generalized seizure in focal epilepsy
Color of the face	Pale	Cyanotic
Hypersalivation, frothing	Absent	Common
Incontinence	Common	Common
Tongue bite	Rare	Common
Postictal confusion	Less than 30 seconds	2 to 30 minutes
Creatine kinase	Normal	Often elevated

Epileptic seizures almost always occur spontaneously, whereas syncope is provoked by specific actions or circumstances, which in about half of the cases can be unearthed by careful history taking. Common precipitants of syncope include prolonged standing, violent coughing, micturition, exertion, intake of antihypertensive drugs, nitrates or alcohol, blood loss, venipuncture or other invasive medical procedures, and even attending rock concerts. Identification of precipitating factors may provide valuable clues to the underlying pathophysiological mechanisms.

Table 2. Precipitants of Syncope: Clues for Etiologic Diagnosis

Precipitant	Pathophysiological mechanism
Standing up	Orthostatic hypotension due to autonomic failure, dehydration, or drugs or as an idiopathic disorder in adolescents; anemia
Carbohydrate meals	Postprandial hypotension of the elderly
Prolonged standing, micturition, pain, invasive medical procedures, glossopharyngeal neuralgia, swallowing, unpleasant sights and smells, psychological shock	Vasovagal (neurally mediated) syncope with reflex vasodilatation or bradycardia or asystole
Heat, alcohol, antihypertensive drugs	Vasodilatation
Coughing, blowing a trumpet, screaming, weight-lifting	Valsalva maneuver resulting in decreased cardiac output
Lying supine in advanced pregnancy	Venous obstruction
Hyperventilation (”stuffy air”)	Hypocapnic cerebral vasoconstriction
Exertion	Cardiac or pulmonary obstruction, eg, valve stenosis, myxoma, pulmonary hypertension
Changing body position	Atrial myxoma
Rock concert	Combination of dehydration, prolonged orthostasis, hyperventilation, and Valsalva (pressing crowd, screaming)

Syncope with automatisms may mimic complex partial seizures. A diagnosis of syncope is supported by the preceding fall, the short duration of the automatisms, and the rapid reorientation of the patient. Lesser degrees of cerebral hypoperfusion, sometimes called presyncope, may likewise resemble complex partial seizures by rendering the patient

confused and unresponsive while still being able to maintain upright posture.

The distinction of epileptic seizures and syncope can be achieved with 94% sensitivity and 94% specificity with a 9-item score that includes: (1) waking with cut tongue (2 points), (2) abnormal behavior such as jerking (1 point), (3) abnormal posturing or witnessed unresponsiveness (1 point), (4) loss of consciousness with emotional stress (1 point), (5) postictal confusion (1 point), (6) head turning (1 point), (7) déjà vu or jamais vu (1 point), presyncope (-2 points), (8) prolonged standing or sitting (-2 points) and (9) diaphoresis before a spell (-2 points). A score of 1 or more points indicates a diagnosis of seizures rather than syncope (Sheldon et al 2002).

Rarely, both syncopal and epileptic mechanisms may interact within 1 attack. Thus, syncope may provoke an epileptic seizure and vice-versa. Only a few EEG- or video-documented epileptic seizures evolving from syncope have been reported. Most of them occurred in children in whom syncope was followed by an absence (Guerrini et al 1991) or a generalized clonic seizure (Stephenson 1991; Horrocks et al 2005). A complex partial seizure triggered by syncope appears to be even rarer (Bergey et al 1997). In contrast, innumerable other accounts of "syncope followed by a seizure" have been poorly substantiated and obviously reflect misinterpretation of hypoxic convulsions. Conversely, cardiac arrhythmia may accompany epileptic seizures, especially those of temporal lobe origin, which may lead to syncope in the course of a complex partial seizure. Such secondary syncopes are characterized by asystole for more than 6 seconds, loss of muscle tone or asymmetric posturing, or jerking evolving from a typical temporal lobe seizure (Ghearing et al 2007; Schuele et al 2007; Bestawros et al 2015). When a patient's history contains elements of both epilepsy and syncope, ictal EEG and ECG recordings are usually required to establish the diagnosis (Kouakam et al 2009; Jackson et al 2015). Ictal syncope can be prevented by antiepileptic treatment or pacemaker implantation (Bestawros et al 2015; Kohno et al 2016). Epileptic asystole may occur as an isolated ictal phenomenon, rendering clinical distinction from other forms of syncope impossible (Ruppert et al 2015).

The most common nonepileptic differential diagnosis of syncope is psychogenic seizure. Both syncope and psychogenic seizures may be triggered by emotional upset, may go along with convulsions, and may lead to injury due to an abrupt fall. Distinctive features that are characteristic of psychogenic seizures include provocation by suggestion, duration of several minutes up to an hour, closed eyes, and long-lasting tonic, clonic, or complex movements. Preserved consciousness can often be demonstrated by nonverbal responses during the attack and partial memory of ictal events.

“Concussive convulsions” is a term for generalized tonic and myoclonic activity appearing immediately after closed-head injury (McCrory et al 1997). Concussive convulsions may last up to 4 minutes. Recovery is rapid, and the outcome is favorable with respect to neurologic and neuropsychologic function. Epilepsy does not develop in affected individuals. The underlying mechanism is still unclear, but the immediate appearance of convulsions within 2 seconds after the impact argues against cerebral hypoxia. Similarly, an epileptic mechanism seems unlikely regarding the benign nature of these convulsions.

Underlying disorders

Convulsions may occur with any type of syncope, no matter if it is orthostatic, vasovagal, or cardiac in origin. Convulsions reflect the final common pathway of syncope: global cerebral hypoperfusion.

Diagnostic workup

The diagnosis of syncope is made in 2 steps: first, identifying an attack with loss of consciousness as syncope, and second, establishing its underlying cause. Although a careful history remains indispensable for differentiating seizures and syncope, additional investigations may sometimes help to settle doubtful cases. Creatine kinase plasma concentrations usually rise within 24 hours after a generalized tonic-clonic seizure but remain normal after syncope (Neufeld et al 1997). Serum lactate measured 2 hours after the event is usually elevated in patients with tonic-clonic seizures but rarely in those with syncope. A cut-off concentration of 2.45 mmol/l provided a sensitivity of 0.88 and a specificity of 0.87 in a single study (Matz et al 2016). Prolactin levels rise within the first hour after a generalized tonic-clonic seizure and may increase or remain unchanged after syncope; therefore, they are not helpful for the differential diagnosis (Chen et al 2005). Serum neuron-specific enolase rises in most patients after a generalized tonic-clonic seizure but not in patients with syncope, but sensitivity for identification of a seizure is only 58% at a cut-off of 11.5 ng/mL (Lee et al 2010).

The diagnostic power of the EEG is often overestimated. Epileptic discharges on an interictal recording certainly support a diagnosis of epilepsy, but they do not rule out additional syncopal attacks. A negative EEG does not settle the matter either. Epileptic discharges may be absent in a single interictal EEG even in chronic epilepsy and especially after seizures related to drug or alcohol withdrawal. The routine use of EEG in the evaluation of syncope is not recommended (Dantas et al 2012).

Reproduction of syncope in the laboratory by tilt testing, eyeball pressure, or hyperventilation has been advocated to confirm the diagnosis. A positive response may demonstrate the patient's propensity for vasovagal (neurally mediated) syncope, but it does not necessarily imply that the patient's habitual attacks are also syncopal in nature (Landau and Nelson 1996). Therefore, testing should be recorded by video, EEG, and ECG (LaRoche et al 2011) and a relative of the patient should review the video to confirm its similarity to previous episodes.

Once a diagnosis of syncope has been established, the etiologic workup should be tailored to the individual patient (Sutton et al 2012). When history clearly points to the underlying mechanism, no further investigations other than an ECG are required. A diagnosis of orthostatic hypotension can be confirmed when systolic blood pressure drops by at least 20 mm/Hg and typical symptoms appear during the first 3 minutes after changing from supine to upright stance. In syncope of unknown origin, tilt testing may occasionally be helpful in reproducing neurocardiogenic syncope under laboratory conditions. More important, however, is the identification or exclusion of cardiac arrhythmia, especially in elderly patients. The relatively low yield of 24-hour Holter monitoring may be increased with prolonged monitoring using portable or implantable loop recorders, which are activated only after the event has occurred (Petkar et al 2012). Only selected patients will require electrophysiological testing for identification of arrhythmias.

Management

Anticonvulsants have no place in the management of convulsive syncope. Instead, the management of convulsive (and nonconvulsive) syncope is directed at the underlying pathophysiological mechanism (Sutton et al 2012). In vasovagal (neurally mediated) and Valsalva-induced syncope, avoidance of precipitating factors is often sufficient. Currently, there is no evidence that drugs may prevent recurrent vasovagal syncope (Brignole 2003). Patients with orthostatic syncope may benefit from increased fluid and sodium intake, sleeping with the head of the bed raised, compressive stockings, and drugs such as fludrocortisone. Treatment of cardiac syncope should address the specific disorder and may include medication or implantation of a pacemaker or an automatic defibrillator.

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ICD and OMIM codes

ICD codes

ICD-10:
Syncope: R55

Profile

Age range of presentation

01-23 months
02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

Sex preponderance

Female ˃ male

Family history

Family history may be obtained in some forms of cardiac syncope.

Heredity

Heredity may be a factor.

Population groups selectively affected

None

Occupation groups selectively affected

None

Associated disorders

Carotid sinus syncope

Other topics to consider

Breath-holding spells
Cough syncope
Drug-induced syncope
Micturition syncope and defecation syncope
Neocortical temporal lobe seizures
Neurocardiogenic syncope
Reflex anoxic seizures
Swallow syncope
Syncope