Recognizing syncope: pitfalls and surprises

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SUMMARY

Loss of consciousness and falling are the key features of syncope. Common accompaniments include tonic and myoclonic muscle activity, eye deviations, automatisms, vocalizations and hallucinations which may render the distinction from epileptic seizures difficult. Differential diagnosis is based on the specific features and not the mere presence of these phenomena. Recognition of syncope depends also on accurate information about precipitants, premonitory symptoms and postictal events: the absence of postictal confusion has been identified as the single most powerful factor discriminating syncope from epileptic seizures whereas incontinence and head injury are common in both conditions. Investigations such as electroencephalogram, tilt testing and postictal prolactin or creatine kinase levels may be helpful but are never diagnostic in isolation. Exceptionally, hypoxic and epileptic mechanisms interact within a single attack.

INTRODUCTION

Syncope is diagnosed in two steps: first, identifying a transient loss of consciousness as syncope; and, secondly, establishing its underlying cause. Comprehensive reviews[1,2] offer guidance for the latter, i.e. the aetiological work-up of syncope. This article will focus on the former, the recognition of syncope and its differentiation from epileptic seizures, which is a common dilemma in clinical practice.

It appears that the most frequent source of error is not inaccurate accounts of symptoms from patients or relatives but misconceptions held by doctors[3]. Textbook descriptions of syncope tend to recall the melodramatic faints of Hollywood movies: the actress/patient sighs, sinks to the ground, lies motionless with eyes closed and finally recovers wondering 'Where am I?'. Research into the semiology of syncope has contested every single element of this stereotype.

To elucidate the phenomenology of syncope we recently videotaped and analysed attacks that were induced by hyperventilation and the Valsalva manoeuvre in healthy volunteers[4]. Previous investigators studied syncope induced by various means such as the Valsalva manoeuvre alone[5], exposure to acceleration on a centrifuge[6], venipuncture and blood loss[7], ventricular arrhythmia[5] and ocular compression[8–10]. The clinical phenomenology proves quite consistent, irrespective of the induction procedure.

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FALLS

We were concerned to study the motor symptoms of syncope as naturally as possible. Therefore, we did not constrain our subjects but allowed them to fall onto a mat of foam rubber and move freely. Surprisingly, only half of them collapsed flaccidly while the others fell with knees and hips extended. Thus, a stiff fall does not necessarily herald a generalized tonic clonic seizure.

CONVULSIONS

The term 'convulsive syncope' implies that there is a peculiar variant of syncope complicated by myoclonic or tonic muscle activity. Moreover, it suggests that an epileptic mechanism may be at work. Both presumptions are erroneous. Convulsions are an integral component of the brain's response to hypoxia; they represent the rule rather than the exception. Reported frequencies vary from 12%[7] to 100%[11] but most investigators observe them in between 70 to 90% of syncopal episodes[4–6,8,9,12]. High rates were usually obtained from prospective studies and when events were recorded on film or video[4–6,12].

That convulsions are less often recounted by an ordinary eyewitness reflects their fleeting nature and variable intensity. Syncopal myoclonus may manifest itself as anything from a single twitch of the mouth to a storm of violent jerks affecting the whole body. It is often multifocal, with asynchronous muscle jerks in different parts of the body. Alternatively, it may be generalized with bilateral synchronous muscle activation. Both forms of myoclonus may occur during an attack[4]. In contrast to epileptic muscle activity, syncopal myoclonus is not rhythmic and is only rarely sustained for more than half a minute.

Tonic muscle activity during syncope typically consists of head and body extension with either flexion or extension of the arms[5,7]. Usually it is only mild and does not resemble the forced extensor posturing of a generalized tonic clonic seizure. However, a brief but intense opisthotonic stiffening is a common accompaniment of breath-holding attacks and other forms of childhood syncope[9].

There is nothing to suggest that syncopal convulsions reflect epileptic activity of the cerebral cortex. During an attack the electroencephalogram (EEG) shows a quite uniform sequence of generalized slow waves of high amplitude, flattening of the trace and return of slow waves before normal background activity is restored. Epileptic discharges are consistently absent[5,8,12] both on ictal and interictal recordings. Muscle activation during syncope is subcortical and probably originates from abnormal firing of the reticular formation in the lower brainstem[13]. Microelectrode recordings from laboratory animals exposed to total brain ischaemia showed preservation and even increase of neuronal activity in the medullary reticular formation lasting up to 40 s whereas cerebral cortex potentials ceased after 10 s[14].

EYE MOVEMENTS

As a rule, eyes are open during syncope, a feature that is shared by epileptic but not by psychogenic seizures[15]. Syncope often starts with a vertical downbeating nystagmus[9,16], which tends to be missed in everyday circumstances. The most consistent ocular motor sign is an upward turning of the eyes early in the course of syncope[7,16,17]. This may be followed by a lateral deviation[16] which can further complicate the distinction of syncopal and epileptic eye movements. Unlike syncopal eye turns, epileptic eye deviations tend to last longer than just a few seconds.

AUTOMATISMS

Automatisms are complex movements performed during an impairment of consciousness. They have only rarely been reported in syncope[9], and yet we observed them in 80% of our subjects[4]. Typical are lip-licking, chewing, fumbling, reaching for the head, head raising, sitting up or even standing up while still being unresponsive and amnesic. In contrast to epileptic automatisms these movements were mostly short and solitary rather than repetitive. Occasionally, however, prolonged complex movements during syncope may render the differentiation from complex partial seizures difficult[9]. Similarly misleading may be growling or moaning vocalizations which we noticed in 40% of subjects.

HALLUCINATIONS

Another feature that links syncope with complex partial seizures is hallucinations. They are usually ignored as doctors do not ask and patients do not volunteer them. Systematic studies, however, have uncovered them with considerable regularity[4,5,17,18]. In our study, 60% of subjects experienced dreamlike hallucinations which were always visual and often also auditory. In some, visual hallucinations were restricted to a perception of grey haze, coloured patches or glaring lights. Others encountered more complex scenes involving landscapes, familiar situations or persons. Four 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. Unlike epileptic auras, syncopal hallucinations do not precede the attack but rather extend into the reorientation period[4,17].

Commonly the emotional experience of syncope was described as detachment, weightlessness and peace, so that the subjects were reluctant to return to reality. Some compared it to drug or meditation experiences and two were reminded of a previous near-death experience. This similarity led us to speculate that near-death experience may reflect hypoxic disinhibition of the limbic system rather than entry into a transcendental domain[18].

PRECEDENTS

As a rule of thumb, epileptic seizures 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 include prolonged standing, violent coughing, micturition, defecation, exertion, intake of antihypertensive drugs, nitrates or alcohol, blood loss, venipuncture or other invasive medical procedures and even attending rock concerts[19,20]. Psychological shock is another frequent precedent of syncope which may render the distinction from hysterical seizures difficult.

The premonitory symptoms of syncope include bilateral tinnitus and 'blackening-out'—a transient amaurosis while consciousness is still preserved—which is caused by the early collapse of retinal perfusion. Lightheadedness and faintness are equally common but less specific as patients may use these terms also to describe an epileptic aura or the sensation that precedes a hysterical seizure.

POSTICTAL PHENOMENA

Several postictal features may help to discriminate between syncope and an epileptic seizure. The single most powerful factor is postictal confusion as observed by an eyewitness[21]. Reorientation is usually immediate in syncope and does not exceed 30 seconds even after extended attacks[12]. Thus, any postictal disorientation lasting longer than that suggests an epileptic seizure. Tongue bites point likewise to an epileptic event, but there are exceptions to this rule[4,9,21]. In contrast, urinary incontinence and head injuries appear to be equally

Table 1 **Clinical features of syncope and generalized tonic clonic seizures**

	Syncope	*GTCS*
Precipitating event	~50%	None
Falls	Flaccid or stiff	Stiff
Convulsions	~80%, usually <30s, arrhythmic, multi-focal and/or generalized	Always 1–2 min, rhythmic, generalized
Eyes	Open, transient upward or lateral deviation	Open, often sustained deviation
Hallucinations	Late in the attack	May precede seizure in focal epilepsy
Incontinence	Common	Common
Tongue bite	Rare	Common
Postictal confusion	<30 s	2–20 min
Prolactin, creatine kinase	Normal	Elevated

common in syncope and generalized tonic, clonic seizures (GTCS)[21]. Exhaustion, sleepiness, vomiting, headaches and muscle aches may all occur after syncope[9,21] but tend to be more frequent and severe after GTCSs. Table 1 summarizes the main features differentiating syncope and GTCS.

INVESTIGATIONS

Although a careful history remains indispensable for differentiating seizures and syncope, additional investigations may sometimes help to settle doubtful cases. Prolactin plasma concentration is usually elevated within the first hour after a GTCS and creatine kinase from two hours onward. Both are unchanged after syncope[22,23]. Obviously, a negative test on its own is insufficient to diagnose syncope. False negative results and other non-epileptic attacks such as hysterical seizures have to be taken into account[24].

The diagnostic power of the electroencephalogram is often overestimated[25]. Epileptic discharges on an interictal recording certainly support a diagnosis of epilepsy but 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 all the more after seizures related to drug or alcohol withdrawal[26].

Reproduction of syncope in the laboratory by tilt testing[27], eyeball pressure[8,9], or hyperventilation[26] has been advocated to confirm the diagnosis. A positive response, however, does not necessarily imply that the patient's habitual attacks are also syncopal in nature[28]. Therefore, a relative of the patient should witness the event, or review it on video, to confirm its similarity to previous episodes.

INTERACTION OF SYNCOPAL AND EPILEPTIC MECHANISMS

I have emphasized that many features of syncope may look epileptic but are non-epileptic with regard to their underlying pathophysiology. Exceptionally, however, both syncopal and epileptic mechanisms are active within one attack. Thus, syncope may provoke an epileptic seizure and vice versa.

Cerebral hypoxia has potent epileptogenic effects, not only after causing structural damage to the cortex[29] but also at earlier stages. This is why hyperventilation (leading to cerebral vasoconstriction) is routinely used during EEG recordings to activate epileptic discharges. Nevertheless, only about a dozen EEG documented epileptic seizures evolving from syncope have been reported. Most of them occurred in children in whom syncope was followed by an absence[30–33] or a clonic seizure[9]. In contrast, innumerable other accounts of 'syncope followed by a seizure' have been poorly substantiated and obviously reflect misinterpretations of hypoxic convulsions.

Cardiac arrhythmia is a common accompaniment of epileptic seizures, especially those of temporal lobe origin[34,35]. Only rarely however are changes in heart rhythm severe enough to provoke syncope in the course of a complex partial seizure[36,37]. Ictal EEG/electrocardiogram recordings are required to establish the diagnosis and to discriminate atonic drop attacks, another rare complication of temporal lobe epilepsy[38].

CONCLUSION

Tonic and myoclonic convulsions, automatisms, vocalizations, eye deviations and hallucinations are all common manifestations of transient cerebral hypoxia. It is not their presence or absence but their specific phenomenology that distinguishes syncope from an epileptic seizure. Hypoxic and epileptic mechanisms may interact within one attack but this seems to be exceedingly rare.

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