

When patients with epilepsy or “epilepsy” might need a pacemaker

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Ictal asystole means cardiac standstill during an epileptic seizure, as in the woman described by Guldiken *et al.*, (2015) in this issue. If the asystole lasts long enough – more than 6 seconds – then a syncope results (Bestawros *et al.*, 2015). This is a situation in which a patient with definite epilepsy might need a cardiac pacemaker, but only after careful thought (Benditt *et al.*, 2015).

Much more common is the scenario in which a patient is treated for “epilepsy” but instead has non-epileptic reflex syncope with or without cardiac asystole (Stephenson, 1990; Petkar *et al.*, 2012).

It seems worthwhile to discuss how one should make one or other diagnosis, how one should treat, and especially whether a cardiac pacemaker might be indicated.

The first question for the clinician is whether the patient has pure syncope, an uncomplicated epileptic seizure or a combination of the two. Although an important consideration in early life (Horrocks *et al.*, 2005) we may leave the phenomenon of anoxic-epileptic seizures – in which a syncope induces an epileptic seizure – because these are extremely rare in adults. The converse – syncope induced by an epileptic seizure – is of course the focus of the report (Guldiken *et al.*, 2015) accompanying this editorial.

There are many descriptions of the phenomenology of syncope (such as Stephenson, 1990; Lempert *et al.*, 1994; Jackson *et al.*, 2015) but for most doctors the best review is Lempert (1996) which aims to teach clinicians how to recognise syncope, particularly in contrast to a generalised tonic-clonic epileptic seizure.

As Lempert (1996) points out, loss of consciousness and falling are the key features of pure syncope, albeit the falling may be either flaccid or stiff. Common accompaniments include tonic and arrhythmic myoclonic muscle activity, eye deviations, automatisms, vocalizations and hallucinations (in the recovery period). Flushing at the end is also common (Stephenson, 1990).

When a syncope is induced by an epileptic seizure – that is, ictal syncope – then the seizure type is complex partial, commonly with origin in the temporal lobe or specifically the insula (Catenox *et al.*, 2013).

Such patients are not expected to fall when the seizure does not induce syncope. Thus, in epilepsy with complex partial seizures, falling with or without bilateral limb movements suggests ictal syncope (Ghearing *et al.*, 2007).

Ictal syncope has features very similar to non-epileptic syncope including early and late myoclonus and flushing at the conclusion (Nguyen-Michel *et al.*, 2014). Almost all reported ictal syncopes have been associated with ictal asystole but there is one convincing published example of ictal syncope with no asystole: in this case vasoplegia with systemic arterial hypotension was presumed (Nguyen-Michel *et al.*, 2014). A pacemaker would not help here (Benditt *et al.*, 2015).

After asystole EEG activity commonly disappears (flat EEG) so it is not surprising that when there is ictal syncope the duration of the seizure is less than when there is only bradycardia (Moseley *et al.*, 2011b). Thus, ictal syncope may paradoxically be of benefit as a seizure-terminating mechanism (Benditt *et al.*, 2015).

In patients with ictal asystole cardiac pacing has been reported to lead to a marked reduction in the number of falls (Moseley *et al.*, 2011a), but in some centres most such patients are not paced (Nguyen-Michel *et al.*, 2014). If there was evidence that ictal asystole is a causal precursor of SUDEP (sudden unexpected and unexplained death in epilepsy) that would be an argument for pacing. However, no such evidence exists (Nguyen-Michel *et al.*, 2014, Benditt *et al.*, 2015).

In conclusion, it may be wise to regard ictal syncope with or without asystole as being as benign as non-epileptic reflex syncope (Nguyen-Michel *et al.*, 2014) and to manage accordingly.

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