LETTER TO THE EDITOR

Syncope and electroencephalography

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Sir,

van Dijk and colleagues have made an impressive scientific analysis of the relation between the clinical features during head-up-tilt induced syncope in adults and the simultaneous EEG findings (van Dijk et al., 2014). In particular, these authors have drawn attention to the semiology associated with the ‘classical’ slow-flat-slow EEG pattern and with simple EEG slowing without EEG flattening. I would like to add some observations to their excellent study, based on personal experience (Stephenson, 1990) and perusal of the literature.

If syncope is sufficiently severe the slow-flat-slow EEG appearance seems to be independent of age—in that it is also observed in young children—and is seen in all types of syncope, however induced. However, the exact clinical semiology seems to vary with different methods of syncope generation. For example, in Gastaut and Fischer-Williams’s (1957) predominantly adult group who had 14–15 s or more cardiac standstill induced by ocular compression while sitting, the resultant flat EEG was accompanied by sudden opisthotonus, whereas this was not usual in my series of children who had ocular compression supine (Stephenson, 1990) and was not seen at all in the latest series of adults with head-up-tilt induced syncope (van Dijk et al., 2014).

The argument that myoclonic jerks are of cortical origin because they are not seen when the EEG is flat (van Dijk et al., 2014) is not convincing. Other authors have noted such jerks during the flat EEG phase (Lombroso and Lerman, 1967; Lempert et al., 1994) and I have published evidence that some of these jerks may be spasms rather than true myoclonus (Stephenson, 1990), suggesting a subcortical origin.

Some neurologists may not be familiar with anoxic-epileptic seizures in which a syncope triggers an epileptic seizure (Horrocks et al., 2005) but a rare example of ictal EEG in anoxic-epileptic seizures is worth some study in the light of the present discussion. In Fig. 1 it is evident that rhythmic brief jerk artefacts appear in both the EEG and the ECG channels while the EEG is still flat, and some seconds before rhythmic spike-wave in the EEG signals the return of cortical function.

Regarding signs supposedly ‘rarely reported’, oral automatisms were described in the video study of Lempert et al. (1994), albeit precise numbers were not published. What they wrote was:

‘Motor activity other than myoclonus was observed in 33 syncopes (79%)… Some patients performed repetitive purposeless movements such as lip-licking, chewing, or fumbling, which resembled epileptic automatisms.’

I also described oral automatisms in more than one case example (Stephenson, 1960).

I realize that limb automatisms would have been outside the field of the video camera in van Dijk et al. (2014), but this was somewhat of a pity insofar as these automatisms might be the clinical expressions of the activity of subcortical central pattern generators (Brigo, 2011).

One other minor point is that the English translations of French papers in PubMed are not necessarily correct. For example, in the French original, Gastaut et al. (1956) did not describe ‘150 cases of vago-vagal syncope’ but rather ‘150 sujets présentant des syncopes vaso-vagales’. Vago-vagal syncope is rare enough to be described in individual case reports (Lewis et al., 1988)—150 would be too many even for the clinic of the great Henri Gastaut!
Figure 1 EEG/ECG recording showing anoxic-epileptic seizure induced by ocular compression. Each panel lasts 20 s. In the upper panel ocular compression induces instant asystole and the EEG becomes flat within 12 s (the fast deflections in the 9th and 11th channels correspond to downbeat nystagmus). In the centre panel the tonic phase of the anoxic seizure is shown on the ECG line as black ‘fuzz’ due to tonic EMG activity. Midway across the centre panel while the EEG is still iso-electric, rhythmic 2–3/s jerk artefacts are seen on EEG and ECG channels. By the lower panel, rhythmic EEG spike wave is visible, slowing towards the conclusion. Jerking lasted 28 s but the child was unresponsive for a further 8 min (reproduced with permission from Stephenson 1990, Mac Keith Press, Fig. 11.4).
References


