Video-EEG in syncopal attack due to ocular compression in an adolescent mistreated for epilepsy

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ABSTRACT – Vasovagal syncope can be confused with epileptic seizure. In this situation, eye compression may be performed during EEG. We present a video-EEG of a patient in whom eye compression produced a typical syncope. Before this recording, the patient had been treated for five years with sodium valproate because of a misdiagnosis of epilepsy. The diagnosis of neurally-mediated syncope was not difficult on the basis of the clinical history, but the reproduction of syncope reinforced the correct diagnosis. Ocular compression is now only indicated in exceptional circumstances and should be performed with precautions. The video of this episode in an adolescent should be of considerable educational value to those who are confronted to diagnostic work-up of transient loss of consciousness.

[Published with video sequences]

Key words: eye compression, syncope, EEG, ECG, seizure, epilepsy, adolescent, epilepsy misdiagnosis

Eye compression is performed in order to initiate vagal hyperactivity. In individuals presenting vagal hyperactivity, eye compression induces bradycardia or sinus arrest with clinical symptoms of varying intensity. We present a patient in whom eye compression produces syncope.

This 14-year-old adolescent experienced a first, brief loss of consciousness at age 9 years while he was standing up. The family history was negative for epilepsy, but both parents and a grand-mother had had syncopes in the past. His EEG disclosed high voltage delta waves with sharper-contoured waveforms during hyperventilation. A diagnosis of epilepsy was retained and he was treated with valproate. Two years later, he had a similar episode. The patient mentioned that he could feel it coming on. A third episode was characterized by dizziness, a warm feeling followed by a fall, with recovery of full consciousness soon after. He was still on valproate. A misdiagnosis of epilepsy was suspected by his neurologist and he was referred for assessment. The semiology of the attacks was suggestive of syncope and ocular compression was performed during EEG. Ocular compression triggered a 22.5 second cardiac arrest (figures 1-4). The syncope (ocular
Figure 1. Recording at 30 mm/s; 10 µV/mm (T1, T2, TA1, TA2, supplementary inferior/anterior temporal electrodes). The patient is awake, eyes closed. The doctor presses the eyes. Five seconds later a cardiac arrest occurs. At this moment, eye compression should be stopped but due to the lack of experience of the doctor and the nurse, the procedure is continued.

Figure 2. Compression is stopped. Note the progressive slowing of the EEG with diffuse slow waves at the end of the figure.
Figure 3. At the beginning, the breathing is regular and there is no change of the face allowing us to suggest that the patient is still conscious. He loses consciousness (syncope) when the EEG demonstrates no cerebral activity. At the end of the figure, resumption of the ECG activity (*).

Figure 4. Three seconds after the start of this trace, the isoelectric phase of the EEG is interrupted by a large downward positive deflection which signals the return of the slow activity that normalises within 4 seconds, coinciding with the return of consciousness. This feature, at the end of the flat-phase of the syncopal EEG, has been illustrated by Stephenson (1990).
revulsion, snorting) starts at the 17th second of cardiac arrest. The EEG demonstrated no cerebral activity for 13 seconds despite resumption of ECG activity. A diagnosis of vasovagal syncope was made. Valproate treatment was stopped. Cardiologic assessment was normal and it was decided not to treat him given the rarity of his syncopes.

Distinguishing epilepsy from syncope can be challenging (Spanaki et al. 2006). Epileptic seizures are sometimes confused with syncopes because of hypertonia and some jerks (Gastaut and Fischer-Williams, 1956, 1957). The main characteristic symptoms for the differentiation from a generalized tonic-clonic seizure are the duration of loss of consciousness (several minutes in generalized tonic-clonic seizure), absence of post-ictal confusion, headache, aching of musculature and tongue biting. Eye compression during EEG and ECG may exceptionally be used to reinforce the diagnosis or aid the management of neurally-mediated syncope (Stephenson and McLeod, 2000), but some precautions are recommended (Stephenson, 1980, 1990). This procedure is contra-indicated if the individual has undergone cataract surgery or has severe eye disorders. Contact lenses must be removed prior to eye compression. If the individual has a history of severe cardiac disorders, eye compression should be performed by a cardiologist in a specialized unit. Atropine must be kept near at hand, and the procedure explained to the patient. He is asked to close his eyes. The clinician then presses firmly on the closed eyes for 10 seconds. The pressure must be strong enough to obtain whitening of a 2 mm spot on the clinician’s fingernail. Intensity of eye compression must be maximal from the onset.

It has been suggested on the basis of a receiver operating curve that an abnormal response is six seconds (Stephenson, 1990). However, the development of symptoms recognized by the subject as similar to the spontaneous event, in association with bradycardia or asystole of three to five seconds may be considered positive (Thomas and Arzimanoglou, 2003). It has been suggested that if a sinus arrest is obtained, eye compression must be stopped to avoid syncope (Thomas and Arzimanoglou, 2003). It is important to continue the recording even in the absence of response concomitant to eye compression because bradycardia can be delayed, appearing several seconds after termination of the eye compression (Gastaut and Fischer-Williams 1956, Crespel and Gelisse 2005).

EEG in patients with cardiac arrest was documented many years ago (Gastaut and Fischer-Williams 1956, 1957). The sequence is a gradual slowing activity with slow theta waves followed by diffuse delta waves. If the cardiac arrest continues, the EEG is isoelectric with no cerebral activity and can show muscle movement activities due to the tonic muscular contraction. Gastaut and Fischer-Williams (1956, 1957) investigated 20 syncopes due to ocular compression (15 seconds), and gave a particularly clear description. Until six seconds, the cardiac arrest had no electroencephalographic effect. After six seconds, generalized slow waves appear the amplitude and duration of which quickly increase to their maximum in a few seconds. If the cardiac arrest does not exceed eight to ten seconds, the electroencephalographic manifestations are limited to this “hypersynchrony” with a more or less severe obnubilation (confusion). If the cardiac arrest lasts more than 13 seconds, one or two bilateral jerks appear, preceded by a contraction in opisthotonos, often associated with a deviation of the head, and sometimes followed by one or two jerks but without a regular clonic convulsive phase as in an epileptic seizure. These phenomena are associated with such remarkable electrocerebral inactivity that it abruptly replaces the phase of hypersynchrony. Resumption of heart beat occurs during this 10 to 20 second phase. Immediately after, a new burst of slow waves appears, the amplitude and duration of which decrease progressively. Ten to 30 seconds later, the slow hypersynchrony waves are replaced with an alpha rhythm and the subject becomes lucid again.

In conclusion, eye compression is a good procedure to confirm vasovagal hyperactivity but should be performed with precautions.

References


