

Transient Eyelid Movements associated with Alpha-Coma following Cardiopulmonary Resuscitation

Despite the development of emergency medicine over the last three decades, coma after out-of-hospital cardiac arrest still presents frequently and leads to important ethical, economic, social and legal consequences. In addition to clinical examination, electrophysiological tests, neuroimaging and laboratory parameters are used to predict outcome^{1,2}. Since the first report concerning the prognostic importance of electroencephalography after cerebral hypoxia by Groenqvist in 1952³, various electroencephalographic (EEG) patterns for outcome prediction have been described.⁴ An EEG pattern of unreactive alpha-like activity with widespread distribution (predominantly over the anterior regions with absent or minor fluctuations in amplitude) appearing in deeply comatose patients without any spontaneous or reactive motor phenomena has been described as 'alpha-coma'.^{5,6} Depending on the etiology⁷, alpha-coma has been considered a temporary antemortem stage sometimes following a burst-suppression pattern.^{5,8} We report a patient presenting with alpha-coma after cardiac arrest, accompanied by transient spontaneous eyelid movements. Although spontaneous eyelid movements have been reported in burst-suppression EEG patterns, to our knowledge this phenomenon has not been described before in alpha-coma.

CASE REPORT

A 57-year-old woman with long-standing type II diabetes mellitus and aortic stenosis had an out-of-hospital cardiac arrest and was resuscitated by the local emergency medical team. On presentation to the emergency team, the patient was found to be in ventricular fibrillation. Advanced cardiac life support was instituted and after primary stabilization the patient was admitted to the Intensive Care Unit (ICU). Body posture was flaccid, there was no reaction to acoustic stimuli or pain and pupils were dilated without response to light (equivalent to zero points on the Innsbruck Coma Scale^{9,10} or three points on the Glasgow Coma Scale¹¹). The time from collapse to the start of cardiopulmonary resuscitation was estimated to be 10 minutes. The interval between start of cardiopulmonary resuscitation and return of spontaneous circulation was another 10 minutes.

During the stay on the ICU mean arterial blood pressure

was between 70 and 94 mmHg. She had a sinus tachycardia with signs of left ventricular hypertrophy on electrocardiography. Chest X-ray showed concentric left ventricular enlargement together with pulmonary infiltration.

The patient was deeply comatose without any reaction to exogenous stimuli. She was artificially ventilated without any sedating drugs. Pupils were unequal and remained unreactive to light. The doll's head phenomenon could not be elicited to the right and only minimally to the left. She had a flaccid tetraparesis, loss of tendon-reflexes and Babinski's signs were positive bilaterally. Shortly after admission facial myoclonic jerks occurred in a periodic fashion for several hours. On the second day after admission the patient showed intermittent eye opening and spontaneous eyelid movements. Continuous eyelid closure and opening appeared with a frequency of about 15 to 20 per minute. EEG demonstrated a monotonous rhythm in the alpha frequency band without any response to painful stimuli. There was a precentral accentuation (Fig.1) and eyelid artefacts without change in the alpha activities (Fig.2).

Otherwise the patient's condition remained unchanged. This phenomenon persisted until death on the third day after cardiac arrest.

DISCUSSION

To our knowledge this is the first report of spontaneous eyelid movements in a patient with alpha-coma. Various abnormalities of eye position and eye movement can be of diagnostic value in an unconscious patient. Typical findings of hypoxic encephalopathy are sustained upgaze with eyes remaining deviated upward for several days before returning to horizontal position; ocular dipping, which is characterized by slow downward eye movement followed by a rapid return to midposition; and so-called reverse ocular bobbing with fast upward gaze, followed by slower return to horizontal.¹² Spontaneous eyelid movements which could be observed in our patient are a very unusual sign in comatose patients, as coma is defined as unarousable unresponsiveness, in which the patient lies with the eyes closed¹³.

Cerebral ischemia due to cardiac arrest, excessive hypotension or mechanical interruption of cerebral blood

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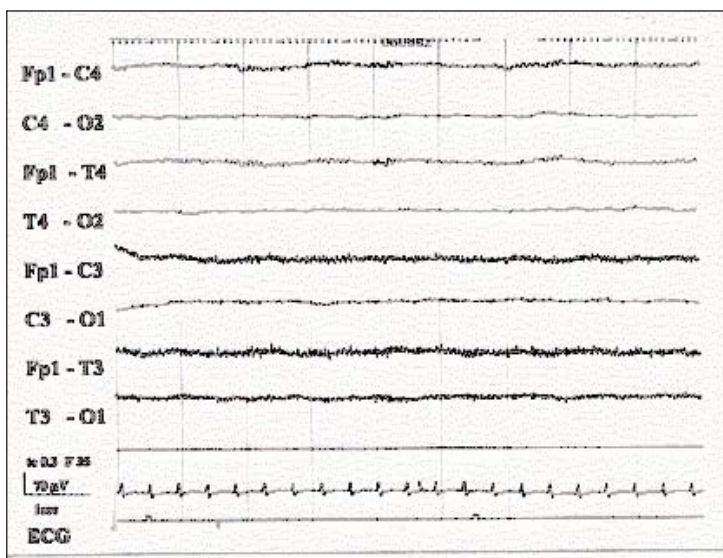


Figure 1: 57-year-old patient, comatose without brainstem reflexes. No reaction to exogenous stimuli. Continuous 10-12/sec activities over the anterior regions. Note muscle artifacts over Fp1.

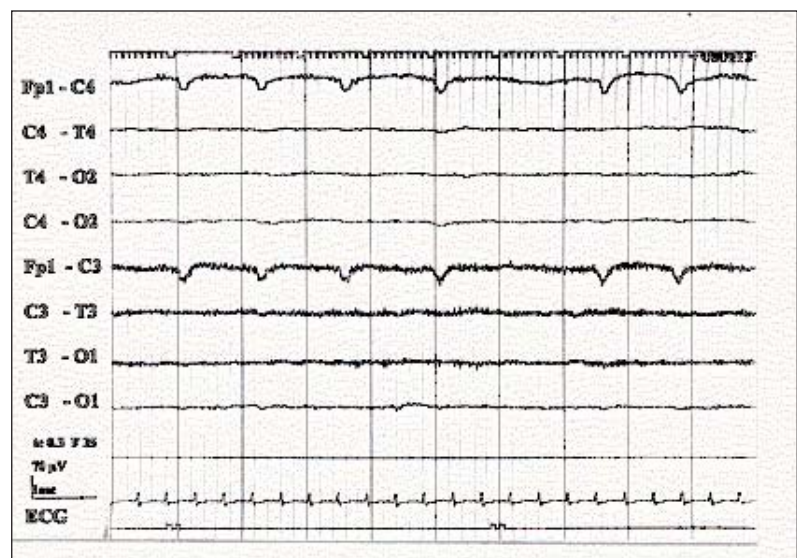


Figure 2: Same record as in figure 1. Frequent artifacts over frontal regions due to spontaneous eyelid movements.

flow leads to various electroencephalographic abnormalities, which may be of prognostic importance.¹⁴ In burst-suppression EEG pattern spontaneous movements have been described by some authors.^{15,16} McCarty and Marshall¹⁵ reported four patients with severe anoxic encephalopathy, in whom eyelid opening followed by slow eyelid closure corresponding to the onset and termination of EEG suppression was observed. This phenomenon, which was not associated with major body movements, is predictive of early death. Reeves et al.¹⁶ described 12 cases of comatose patients with burst-suppression EEG patterns, who manifested complex or tonic oro-facial-lingual movements during the burst portion of the recordings. Other movements associated with the burst portion of a burst-suppression EEG recording include nystagmus¹⁷, pupillary changes¹⁸, chewing¹⁹ and myoclonus¹⁴. The presence and relevance of these movements is important, as they may give false impressions to family members. In our patient myoclonic jerks in the face could be observed shortly after admission.

Burst-suppression activity and the so-called alpha-coma are ominous prognostic signs of hypoxic encephalopathy. The term 'alpha-coma' denotes deep coma accompanied by an EEG pattern resembling that of normal wakefulness. Important differences to normal alpha rhythm concern amplitude, frequency, spatial distribution, variability and reactivity to exogenous stimuli⁶. In unresponsive patients, activities in the alpha-band can be found in hypoxic coma, but also in comatose states due to intoxications and transtentorial herniation.⁵ In severe metabolic coma alpha activities may be observed as an epileptic phenomenon.⁵ Alpha activities in these conditions have to be differentiated from alpha rhythm in patients with pontine lesions, who are often not comatose but locked in. In these 'locked-in' patients the EEG is characterised by a reactive alpha rhythm, sometimes accompanied by underlying temporal slowing.⁵

The specific neuronal structures generating the alpha-coma pattern still remain unidentified. However, there are

theoretical, experimental, and clinical observations suggesting that alpha pattern coma may result from significant thalamo-cortical disruption.^{20,21}

EEG-recordings can be very helpful in the evaluation of patients with altered states of consciousness. Nevertheless, the prognostic impact of EEG patterns such as alpha-coma and burst-suppression depends on the etiology. With hypoxia these patterns carry an extremely poor prognosis, whereas complete recovery can be seen in cases secondary to drug ingestion. According to this, Kaplan et al.²² showed that although the cause of alpha-coma predicts outcome, EEG reactivity in alpha-coma predicts survival; most patients in their series with reactivity awoke, most of those without reactivity died.

The patient presented in this report was deeply comatose without brainstem reflexes. Nevertheless, she developed a state of continuous eyelid closure and opening. These movements were not triggered by exogenous stimuli. Although the patient's appearance resembled, in some aspects, that of persistent vegetative state, this was excluded by the time course and by clinical findings such as the lack of brainstem reflexes or arousal by painful stimuli.¹³ Similar to the reports of burst suppression EEG pattern accompanied by spontaneous eyelid movements^{15,16}, the pathophysiology of the reported phenomenon in our patient remains unclear. The simultaneous appearance of spontaneous eyelid movements and alpha-coma EEG pattern might give evidence that the responsible hypoxic lesion was localized rostral the mesencephalon²⁰ possibly interrupting thalamo-cortical connections.²¹ Hypothetically, such an interruption can lead to disinhibition of the rostral interstitial nucleus of the medial longitudinal fasciculus, which is involved in the control of coordinated lid movements.²³ Unfortunately magnetic resonance imaging for exact localisation of the lesion could not be performed due to cardiac instability.

Our observation makes clear that spontaneous eyelid movements in a posthypoxic patient must not be misinterpreted as a sign of cerebral recovery and do not alter the unfavorable prognosis of alpha-coma pattern.

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