

Asynchronous Burst-Suppression on EEG in Severe, Diffuse Axonal Injury

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A 25-year-old male presented after a car accident, ejected from the back seat after hitting a truck. Initial Glasgow Coma Scale was 3, with a fixed mydriasis on right. Initial CT head showed signs of increased intracranial pressure (ICP) with a small subarachnoid hemorrhage. An external ventricular drain was put in place. Despite aggressive ICP-lowering treatment, the patient remained deeply comatose. On day four, somatosensory evoked potentials were absent over the right hemisphere and difficult to interpret on left because of artifacts. An electroencephalogram (EEG) done the same day showed generalized severe slowing without epileptiform activity. Blood tests were unremarkable.

Since the patient did not awaken from coma, the EEG was repeated on day nine, after the propofol infusion the patient needed to support the ventilator was stopped (Figure 1A). It showed a burst-suppression pattern without epileptiform discharges. Most of the bursts were totally asynchronous between both hemispheres. Bursts had a duration between two to five seconds and practically no activity over $10\mu\text{V}$ was seen between them. This suggested a corpus callosum lesion, which was confirmed by MRI (Figure 1B to 1J). Magnetic resonance image also showed multiple cortical and subcortical lesions, including the brainstem and the upper cervical cord, secondary to cytotoxic oedema. The corpus callosum was particularly involved in the process, the whole structure showing an abnormal signal on the MRI. The MRI was described as an extreme diffuse axonal injury (DAI) by the attending radiologist. After discussion with the family, it was decided to withdraw life support the following day. The patient died shortly after stopping the ventilator.

DISCUSSION

Burst-suppression was first described by Swank and Watson in 1949 after observing the effect of amytal on the dog brain.¹ Most experts consider deafferentation of the cortex from thalamus as the pathophysiologic cause of this EEG pattern. This can be provoked by various conditions, some reversible (usually anesthetic drugs such as midazolam, propofol,

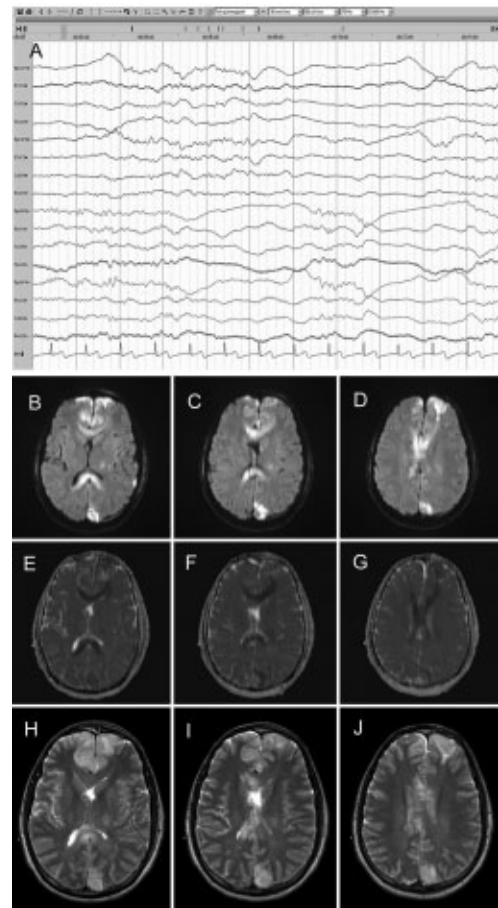


Figure: A. EEG with bipolar longitudinal montage, showing asynchronous burst suppression on day nine after the accident (see text for discussion). B - J. MRI showing multiple cytotoxic oedema lesions compatible with DAI, particularly in left frontal, left occipital and in the corpus callosum, with an increased signal in diffusion (B-D), decreased signal in apparent diffusion coefficient (E-G) and hypersignal in T2 (H-J).

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anesthetic barbiturate, etc., but also profound hypothermia), some irreversible (as in traumatic brain injury and anoxic-ischemic encephalopathy after cardiac arrest).²

Asynchronous burst-suppression is a well known abnormality in the pediatric Aicardi's syndrome, characterized by the triad of chorioretinal lacunae, infantile spasms and partial or complete agenesis of the corpus callosum in a female patient.³ However, this is a rare condition in other settings, even if Swank had already observed asynchronous burst-suppression in dog under barbiturates with a previous transection of the corpus callosum rostrum.⁴ To our knowledge, there are only three case reports in the literature of such a condition in a different setting than Aicardi syndrome, all three involving the corpus callosum.⁵⁻⁷ Our case gives further support to the idea of the concept that the corpus callosum is an essential part in the synchronisation of EEG electrical activity, particularly during burst-suppression.

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