

THE IMPORTANCE OF ELECTROENCEPHALOGRAPHY IN DEVELOPING COMA AND BRAIN DEATH

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Abstract

Electroencephalography (EEG) has long been used in evaluating comatose patients, and is being increasingly found to uncover patterns of prognostic significance, reveal subclinical seizure activity and provide data during treatment in which patients are paralyzed. Some EEG patterns reveal increasing degrees of cerebral compromise with progressive slowing of the background frequencies, while others can be explored for reactivity to external stimuli for prognostic purposes. With some etiologies, particular patterns carry grave import such as flat or highly suppressed patterns, or unreactive alpha, delta or burst-suppression patterns. Others including beta and triphasic patterns may herald a good prognosis, depending on cause.

Keywords: Coma, ischemic stroke, intracerebral hemorrhage, traumatic brain injury, hypoxic encephalopathy, metabolic status, brain death.

Introduction

Coma is an eyes-closed state of unresponsiveness with severely impaired arousal and cognition. It represents a failure of neurologic function resulting from damage of a critical number of brainstem and diencephalic pathways, which regulate the overall level of cortical function. Coma has been identified as a major predictor of death and poor neurofunctional outcomes in patients with a variety of critical illnesses, including ischemic strokes, intracerebral hemorrhage, traumatic brain injury, hypoxic encephalopathy after cardiac arrest, and metabolic derangements or sepsis. Besides ventilator dependency and infectious complications, coma is one of the major critical conditions leading to prolonged intensive care and increased mortality. Cerebral electrographic patterns allow distinction of coma from normal sleep and other causes of confusion or unresponsiveness. Some EEG patterns reflect a deepening or lightening of mental status, though progression of coma through various EEG patterns is inconsistent. Several EEG patterns indicate the type of cerebral impairment, while others may suggest favorable or unfavorable prognoses. This review presents different abnormalities of EEG patterns and background activity seen in coma, along with those that indicate deepening coma and have particular prognostic significance.

The prognostic value of EEG in coma is contingent on the underlying cause. A patient's medications, metabolic status, and core body temperature must be taken into account before a prognosis is based on particular EEG findings because these factors can affect the EEG independently of underlying cerebral disease. A characteristic EEG feature in coma is a background that is nonreactive to external stimuli. In severe coma due to cortical lesions, such as





severe intracranial mass effect or hypoxic ischemic encephalopathy, the EEG may show generalized suppression. Monorhythmic coma patterns have been described in which frequency is predominant, with corresponding names (ie, alpha coma, theta coma, beta coma, and spindle coma patterns). These patterns are most commonly seen in severe hypoxic ischemic encephalopathies but may also be seen in posttraumatic coma. Beta coma may be seen in pharmacologic coma.

Although EEG was increasingly looked at to provide objective evidence of brain dysfunction, it became evident that it provided little in the way of diagnostic specificity to an underlying cause. From another perspective, however, when used in specific etiologies of coma encountered in the intensive care unit, EEG has been progressively seen as providing a helpful tool in prognosis, revealing subclinical seizure activity, and tracking brain activity while patients are paralyzed. To date, EEG is of greatest value in prognostication following closed traumatic brain injury and cardio-respiratory arrest (CRA) with consequent hypoxic-ischemic encephalopathy. Recently, good outcome in comatose patients after CRA was shown to correlate well with EEG background variability and reactivity to stimulation during or after mild therapeutic hypothermia (MTH), or conversely herald poor outcome when evidence of reactivity to noxious stimuli was absent. Advances in quantitative EEG during MTH after CRA recently identified subgroups of patients with distinct evolutions of qEEG “burst-suppression ratios” that were likely to have good neurofunctional recovery.

The burst suppression pattern consists of intermittent bursts of polyphasic, sharply contoured or spiky activity interspersed with periods of suppression, each lasting several seconds. When the burst suppression pattern is seen with anesthesia and hypothermia, it may be reversible. However, the burst suppression pattern in a patient with hypoxic ischemic encephalopathy generally portends a very poor prognosis.

Brain death should be based on clinical examination and cannot be diagnosed from EEG alone. If a clinical brain death examination cannot be performed in its entirety and if reversible causes have been eliminated, EEG may be useful for establishing support for or against brain death. The following EEG criteria for brain death are used for adult and pediatric patients:

A full set of standard electrodes is recommended.

The montage used should allow ≥ 10 cm between any 2 electrodes.

Placement and integrity must be verified by tapping.

Impedance must not be 10,000 Ω

The high-frequency filter must not be set < 30 Hz, and the low-frequency filter must be set at ≤ 1 Hz.

A sensitivity of 2 $\mu\text{V/mm}$ must be used for ≥ 30 minutes of the recording.

There should be electrocerebral silence without reactivity to stimulation throughout the recording.

The recording must be performed by a qualified technologist.

The EEG is supportive of brain death if the above criteria are met in the appropriate clinical context. If true electrocerebral silence is doubtful, the study should be repeated. Although strict criteria do not exist, EEG should not be repeated before 24 hours in the evaluation of brain death in children.





A characteristic EEG feature in coma is a background that is nonreactive to external stimuli. Monorhythmic coma patterns are named according to the predominant frequency or waveforms present (ie, alpha coma, theta coma, beta coma, and spindle coma patterns).

Monorhythmic coma patterns are most commonly seen in severe hypoxic ischemic encephalopathies but may also be seen in posttraumatic coma.

Brain death should be based on clinical examination and cannot be diagnosed from EEG alone.

In conclusion, Electroencephalography (EEG) is a useful diagnostic and prognostic tool for evaluation of patients with epilepsy, encephalopathy, focal lesions, coma, or brain death. Early studies on stupor and coma have correlated decreases in mental status and deepening levels of coma with particular EEG patterns and suppression of EEG reactivity. Electroencephalography (EEG) is frequently used to assist the diagnosis of brain death. To determine brain death, electrocerebral inactivity (ECI) should be demonstrated on EEG at a sensitivity of 2 μ V/mm using double-distance electrodes spaced 10 centimeters or more apart from each other for at least 30 minutes, with intense somatosensory or audiovisual stimuli.

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