CEREBRAL PROTECTION, EEG END-POINT

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Cerebral ischemic injury involves a process whereby energy supply falls short of the energy demand. Protection strategies including:

- Decrease energy demand (cerebral metabolic rate): hypothermia, barbiturates, and other anesthetics.
- Increase energy supply (cerebral blood flow): induced hypertension, hemodilution, mannitol and thrombolysis.

Administration of the various anesthetic agents including barbiturates results in a dose-related reduction in CMRO2 and cerebral blood flow. The maximum reduction occurs with the dose that results in electrocerebral silence. At this point, energy utilization associated with EEG activity has been reduced to zero, but energy utilization for cellular homeostasis persists unchanged. Additional barbiturate administration causes no further decrease in CBF or CMRO2. CMR decreases by 7% per Celsius degree of temperature reduction causing complete suppression of the EEG at about 20 degree C. However, in contrast to anesthetic agents, temperature reduction beyond that at which EEG suppression first occurs does produce a further decrease in CMR.

The burst-suppression state does not represent a uniform physiologic state and occurs just before complete suppression. EEG characteristics of the burst-suppression differ among anesthetic agents.

1) Cerebral ischemia can be classified into three types:
   a. Focal, characterized by the presence of surrounding non-ischemic region (e.g. stroke, arterial occlusion, embolization).
   b. Incomplete global, insufficient blood supply or oxygen delivery to the whole brain (e.g. hypotension or increased ICP).
   c. Complete global, characterized by absent CBF (e.g. cardiac arrest).

2) Agents
   a. Barbiturates: may slightly improve the neurologic recovery from focal or incomplete global ischemia. The protective benefit is likely achieved with an induction dose of barbiturate. In contrast barbiturates have been shown to be of no benefit following cardiac arrest.
   b. Isoflurane: which can induce an isoelectric EEG and significant lower CMR, has not been shown to be beneficial in the setting of global ischemia.
   c. Hypothermia: which can reduce metabolism for both neuronal and cellular functions, is the established protective technique for circulatory arrest procedures. Its use is limited by cardiovascular and respiratory depression, arrhythmias, tissue hypo-perfusion, and coagulopathy.
   d. Hyperthermia: worsens outcome from focal cerebral ischemia and should be avoided.
   e. Hyperglycemia: may worsen neurologic outcome following ischemic insult, probably because anaerobic metabolism of glucose produces excessive lactate, which can lead to intracellular acidosis.
   f. Nimodipine: a calcium channel antagonist, has been shown in some studies to improve outcome after stroke and attenuate cerebral hypo perfusion following global ischemia although with inconsistent neurologic recovery. Nimodipine’s beneficial effects on vasospasm after subarachnoid hemorrhage are well established.
   g. Steroids: have not been found to be beneficial after stroke or severe head injury. High dose methylprednisolone has been shown to produce modest improvement in neurologic recovery following acute spinal cord injury if the treatment started within 8 hours of the injury.
Electroencephalography

Measures electrical activity of the neurons of the cerebral cortex and is thus as a threshold marker for the detection of ischemia due to inadequate CBF. It is used frequently during procedures that jeopardize cerebral perfusion such as carotid endarterectomy or to assure electrical silence before circulatory arrest.

1. Normal CBF is 50 ml/100gm/min, when it decreases below 20-25 EEG slowing occurs; in the vicinity of 18 the EEG becomes isoelectric (flat). Sustained reduction to 8-10 ml/100gm/min result in tissue infarction. The critical level of CBF is defined as that degree of CBF below which signs of ischemia are seen on EEG, and it is roughly correlated with the extent to which each anesthetic depresses CMRO2. The level of critical CBF varies with the anesthetic agents: 20 ml/100gm/min for halothane, 18 ml/100gm/min for Enflurane, and 10 ml/100gm/min for Isoflurane.

2. The EEG may exhibit changes intraoperatively with no demonstrable neurologic deficit during postoperative examination. Cerebral ischemia can produce electrical dysfunction without causing neuronal cell damage because the blood flow threshold for electrical failure is higher than that needed to maintain cellular integrity.

3. Factors other than anesthetics that may affect the EEG include (which may limit the usefulness of EEG during cardiopulmonary bypass), hypotension, hypoxia, tumors, vascular abnormalities, and epilepsy. An abnormal EEG in patients with preexisting neurologic deficits, stroke in evolution, and recent reversible ischemic neurologic deficits can also make it difficult to interpret new changes.

4. Anesthetic effects on the EEG are generally global, which often helps distinguish them from the focal changes of ischemia. A predominance of slow activity is seen as the anesthetic depth increases. “Deep” anesthesia may cause marked EEG slowing, making detection of superimposed ischemic changes during critical periods difficult to interpret. Maintaining a constant level of anesthesia during critical periods (e.g., carotid clamping) facilitates EEG interpretation.

Reference:
