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1. A 14 year old male is in the operating room for scoliosis surgery. Surgeons would like to use neuromonitoring; both sensory and motor evoked potentials. All of the following have minimal effect on the latency and amplitude of the waveforms except

A. Remifentanil
B. Dexmedetomidine
C. Propofol
D. Sevoflurane
A. Remifentanil

Remifentanil is an opioid which has found an important place in neuro-anesthesia due to its ultra short acting half life and feasibility of neuromonitoring. Intravenous opioids produce minimal depression of cortical SSEPs and MEP recordings. Studies have shown mild amplitude decreases and latency increases with opioids thought secondary to the action at the μ receptor via G protein-mediated activity, resulting in depressed electrical excitability. Considering their minimal neurophysiologic effects and superior analgesic properties, an opioid-based anesthetic for scoliosis cases requiring neuro-monitoring seems beneficial.
B. Dexmedetomidine

Dexmedetomidine is a specific $\alpha_2$-receptor agonist that provides anxiolysis and analgesia without depression of respiration. At clinically relevant doses, there is little effect on neurophysiologic monitoring. This ability to minimally affect IONM (intraoperative neuromonitoring) in combination with its MAC-sparing ability for volatile agents makes dexmedetomidine an appealing adjunct when cases require IONM.

Propofol an excellent agent for total intravenous anesthesia (TIVA) due to rapid metabolism and ability for rapid titration of anesthetic depth, minimally effecting evoked potentials.

All halogenated inhalational agents produce dose-dependent decrease in amplitude and increase in latency for SSEPs with cortical responses affected to a larger degree than subcortical and peripheral nerve responses. Doses up to 0.5 minimum alveolar concentration (MAC) can be used if subcortical responses are adequate. With increasing concentrations of halogenated agents, a prominent effect on the anterior horn is noted abolishing transcranial MEPs relegating these agents suboptimal for use in cases where IONM (intraoperative neuromonitoring) is used.

2. A 38 year old male is undergoing extracranial-intracranial bypass surgery. The following would be the goals of anesthesia in this procedure except

A. To keep the blood pressure within 10% to 20% baseline
B. Hyperventilation to produce mild hypocarbia
C. Maintenance of normovolemia
D. Prevention of hypothermia
Preservation of adequate cerebral perfusion pressure is one of the most important goals during the anesthetic management of cerebral revascularization surgery. Mean arterial pressure (MAP) should be kept close to the patient's baseline so as to avoid cerebral ischemia. Insertion of arterial catheter before induction of anaesthesia is preferred for blood pressure monitoring during induction.

To maximise cerebral perfusion, normocarbia is critical. Hyperventilation results in low arterial PaCO2 levels and may induce ischemia due to cerebral vasoconstriction. Hypercapnia is also detrimental as cerebral vasculature is maximally dilated in ischemic areas and high PaCO2 levels and vasodilation will further impair perfusion to already hypoperfused areas (cerebral steal).

C. Maintenance of normovolemia

Patients can become hypotensive perioperatively because of the antihypertensive medications given preoperatively, dehydration, inadequate volume replacement during surgery, blood loss and anesthetic agents. It is important to maintain normovolemia in these patients.

D. Prevention of hypothermia

Although mild hypothermia is regarded as offering some degree of neuroprotection against cerebral ischemia due to reduction in the cerebral metabolic rate, there have been few studies and no randomized clinical trials conducted. Currently, hypothermia is only recommended in situations of cardiac arrest following return of spontaneous circulation. On the basis of the findings for the last 25 years, it can be generally agreed that normal body temperature should be maintained perioperatively.

3. Depression of cerebral oxygen requirements below the level required to create an isoelectric EEG can be achieved by

A. Administration of isoflurane
B. Administration of nimodipine
C. Use of Barbiturates
D. Hypothermia

Go to Q 4
A. Administration of isoflurane

- All volatile anesthetics including isoflurane suppress the cerebral metabolic rate (CMR) with the exception of halothane, can produce burst suppression of the electroencephalogram. At that level, the CMR is reduced by approximately 60%.

- Volatile anesthetics have dose-dependent effects on CBF. In doses less than the minimal alveolar concentration (MAC), CBF is modestly decreased. In doses larger than 1 MAC, direct cerebral vasodilation results in an increase in CBF and cerebral blood volume.

Ref: Cerebral Physiology and the Effects of Anesthetic Drugs
Piyush M. Patel, John C. Drummond and Brian P. Lemkuil
Miller's Anesthesia, Chapter 17, 387-422.e9
B. Administration of nimodipine

Nimodipine is a systemic vasodilator and can also produce cerebral vasodilation. It does not reduce the cerebral oxygen requirements and can increase intracranial pressure.
Barbiturates, etomidate, and propofol can produce burst suppression of the electroencephalogram by decreasing the cerebral metabolic rate (CMR). At that level, the CMR is reduced by approximately 60%. They preserve the blood flow metabolism coupling. However, they are still not able to achieve suppression of CMR below the level required to produce an isoelectric EEG.

Ref: Cerebral Physiology and the Effects of Anesthetic Drugs
Piyush M. Patel, John C. Drummond and Brian P. Lemkuil
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D. Hypothermia

Hypothermia can cause complete suppression of the EEG (at approximately 18°C to 20°C. However, in contrast to anesthetic drugs, temperature reduction beyond that at which EEG suppression first occurs does produce a further decrease in the CMR. This decrease occurs because anesthetic drugs reduce only the component of the CMR associated with neuronal function, whereas hypothermia decreases the rate of energy utilization associated with both electrophysiologic function and the basal component associated with the maintenance of cellular integrity.

4. A patient is undergoing surgery for a cerebellar hemangioblastoma. He has a smoking history and associated chronic obstructive pulmonary disease. All of a sudden, the airway pressure is increased and the ETCO2 is high. Which of the following statements is true regarding the relationship between cerebral blood flow (CBF) and PaCO2?

A. CBF changes 1 to 2 mL/100 g/min for each 1 mm Hg change in PaCO2
B. The changes in CBF caused by Paco2 is due to increase in the CMRO2
C. The CBF decreases as long as the hypocapnia is maintained.
D. Hypocapnia can cause cerebral steal

Go to Q 5
Cerebral blood flow (CBF) varies directly with PaCo$_2$ especially within the range of physiologic variation of PaCo$_2$. CBF changes 1 to 2 mL/100 g/min for each 1 mm Hg change in PaCo$_2$ around normal PaCo$_2$ values. This response is attenuated at a PaCo$_2$ less than 25 mm Hg.

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Piyush M. Patel, John C. Drummond and Brian P. Lemkuil
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The changes in CBF caused by PaCco2 are dependent on pH alterations in the extracellular fluid of the brain. NO(nitric oxide), in particular NO of neuronal origin, is an important although not exclusive mediator of CO2-induced vasodilation. The vasodilatory response to hypercapnia is also mediated in part by prostaglandins.

This is false because despite the maintenance of an increased arterial pH, CBF returns toward normal over a period of 6 to 8 hours because the pH of cerebrospinal fluid (CSF) gradually returns to normal levels as a result of extrusion of bicarbonate.
Hypercapnia has the potential to cause intracerebral steal. Ischemic areas of the brain are already maximally dilated and further increase in PaCO2 can cause blood to flow away from these areas. Despite some support for the occurrence of a favorable so-called Robin Hood or inverse steal, hypocapnia has not generally proved effective in either laboratory or clinical settings. Pending further information and in the absence of a means of verifying the perfusion response to the manipulation of PaCO2, normocapnia remains standard practice in situations where potential for cerebral ischemia exist.

5. A patient with a known difficult airway has received topical benzocaine for an upcoming fiberoptic intubation. Several minutes later, the patient appears cyanotic with an SpO2 value of 85% with no apparent respiratory distress. Which of the following is the most appropriate treatment for this clinical scenario?

A. Administration of sodium nitrite
B. Administration of methylene blue
C. Administration of sodium thiosulphate
D. Administration of suxamethonium for urgent intubation
A. Administration of sodium nitrite

Administration of sodium nitrite like benzocaine can cause methemoglobinemia. Methemoglobinemia is a known complication of many pharmacologic and naturally occurring agents. These include topical anesthetic agents (benzocaine), nitrates, and antibiotics (dapsone).
The diagnosis of methemoglobinemia should be suspected whenever there is cyanosis that does not improve with increased fraction of inspired oxygen (F I O 2), abnormal coloration of blood, physiologically appropriate paO 2 on blood gas sample with low pulse oximeter saturation—"saturation gap" and new-onset cyanosis and/or hypoxia after ingestion of an agent with oxidative properties (benzocaine, nitrites). The diagnosis of MetHb is made using multiwavelength Co-oximetry.

First-line therapy for a patient with methemoglobinemia is supportive care and discontinuation of the offending agent. Definitive treatment is returning MetHb back to its unoxidized state by reducing it with IV methylene blue which serves as a cofactor for the enzyme NADPH MetHb reductase.

C. Administration of sodium thiosulphate

Sodium thiosulphate is used for treatment of cyanide toxicity and not for methemoglobinemia.
D. Administration of suxamethonium for urgent intubation

Despite the diagnosis of MetHb, not all patients will require treatment. Healthy patients will tolerate higher levels of MetHb, while those with pre-existing cardiovascular disease or anemia will not. Decisions to treat patients with intravenous methylene blue should be made after consideration of medical history, degree of symptomatology, and individual patient physiology. However, all patients who show signs of cellular hypoxia should be treated. These signs include presence of clinical symptoms, a metabolic acidosis, or low mixed venous oxygen saturation.

Administration of suxamethonium and intubation will not reverse the methemoglobinemia.