NEUROANESTHESIA

BP CONTROL
- contributes to CPP
- decrease when working around aneurysm
- increase to enhance collateral circulation during temporary clipping

CO2 is the most potent cerebral vasodilator
Hyperventilation 🡪 decreased PaCO2 🡪 decreased CBV and CBF
Goal: end tidal CO2 25-30mm Hg = PaCO2 30-35mm Hg
Hypothermia: each 1 degree C drop in T reduces cerebral metabolic rate of oxygen (CMRO2) by 7%
Reducing CMRO2 helps protect again ischemic injury
Lowering the head increases arterial blood flow and increases ICP by impairing venous outflow

MEDICATIONS

Inhalational agents
- most reduce cerebral metabolism (except nitrous oxide) by suppressing neuronal activity
- disturb cerebral autoregulation and cause cerebral vasodilation which increases CBV and can increase ICP
- if administred for > 2 hours 🡪 increase CSF volume 🡪 elevation in ICP
- increase CO2 reactivity of cerebral blood vessels
- affect intraoperative EP monitoring

 Nitrous oxide (N2O)
 - potent vasodilator
 - markedly increases CBF
 - minimally increases cerebral metabolism
 - contributes to post-operative nausea/vomiting
 - solubility is ~34 times that of nitrogen
 - when it comes out of solution can convert pneumocephalus 🡪 tension pneumocephalus
 - may aggravate air embolism
 - risk of tension pneumocephalus may be reduced by filling cavity with fluid and turning off nitrous 10 minutes before dural closure

 Halogenated agents:
 - all suppress EEG activity
 - provide some degree of cerebral protection
 - Isoflurane
 - Desflurane: cerebral vasodilator, increase CBF and ICP, decreases CMRO2 🡪 compensatory vasoconstriction
 - Sevoflurane: mildly increases CBP and ICP, reduces CMRO2, mild neg inotrope (CO not as well maintained)

Intravenous agents
- Propofol: may be used for induction and continuous infusion during total IV anesthesia (TIVA); causes dose dependent decrease in MAP and ICP
- Barbiturates: reduce CMRO2; produce dose-dependent EEG suppression; minimally affect EPs; most are anticonvulsant; myocardial suppression and peripheral vasodilation can cause hypotension and compromise CPP; sodium thiopental (most common agent)
- Etomidate: anesthetic, amnestic, NO analgesic properties; may produce myoclonus which may be confused with seizures

Narcotics
- increase CSF absorption
- can slow EEG but will not produce isoelectric tracing
- dose-dependent respiratory depression 🡪 elevated ICP
- morphine: does not significantly cross BBB
- fentanyl: crosses the BBB; reduces cerebral metabolism/CBV/ICP
- precedex: alpha 2 adrenergic receptor agonist

Paralytics
- Succinylcholine: depolarizing agent; may transiently increase ICP; dose: 1-1.5mg/kg (20mg/ml) ~ 3.5-5cc for 70kg; onset 60-90 seconds, duration 3-10 minutes, may repeat x 1
- Rocuronium: non-depolarizing
- Vecuronium: not approved for rapid sequence intubation

ANESTHESIA AND IOM
- All volatile agents produce dose-dependent reduction in SSEP peak amplitude and increase peak latency
- TIVA is ideal
- if inhalational is used, use < 1 MAC (minimal alveolar concentration), ideally < 0.5 MAC
- non-depolarizing agents have little effect on IOM
- propofol has mild effect
- benzodiazepines have mild-to-moderate effect
- SSEP can be affected by hyper/hypothermia and BP changes
- AED do not affect SSEP

MALIGNANT HYPERTHERMIA
- hypermetabolic state of skeletal muscle by block of Ca re-entry into sarcoplasmic reticulum
- multifactorial genetic predisposition
- total body O2 consumption increases 2-3 times
- 50% had previous anesthesia without MH
- associated with halogenated agents and succinylcholine
- 30% mortality
- presentation:
 - increase in ETCO2
 - tachycardia
 - progression: DIC, metabolic acidosis, decreasing PO2, pulmonary edema, elevated T (113⭘F), limb rigidity, rhabdomyolysis
 - terminal: hypotension, bradycardia, cardiac arrest
- treatment:
 - eliminate offending agent (stop OR, d/c agent, change tubing on anesthesia machine)
 - dantrolene 2.5mg/kg IV, infuse until symptoms subside up to 10mg/kg
 - hyperventilation with 100% oxygen
 - cooling
 - bicarbonate 1-2mEq/kg for acidosis
 - IV insulin and glucose (lowers potassium, glucose acts as energy substrate)
 - procainamide for arrhythmias
 - diuresis
- prevention:
 - identify at risk patients (muscle biopsy)
 - family history
 - pts with heavy musculature, Duchenne MD, scoliosis
 - pts with masseter spasm following succinylcholine
 - in pts at risk avoid succinylcholine and use non-halogenated anesthetics
 - prophylactic oral dantrolene: 4-8mg/kg/day for 1-2 days (last dose 2 hours before anesthesia)