Topics Covered Today

- Intracranial Pressure
- Intracranial Compliance
- Cerebral Metabolism
- Cerebral Blood Flow
- Evoked Potentials
Neuroanesthesia at Upstate

- Everyone does a Neuro rotation
- Please read a textbook prior to neurorotation
- Neuroanesthesia is not complex; however small errors lead to big price to pay in terms of poor outcome
Intracranial Pressure

- The cranial vault is a rigid structure.
- It consists of the brain (80%), blood (12%) and CSF (8%)
- Any increase in one component is offset by an equivalent decrease in another or else ICP increase
- Normal ICP is 0-10 mmHg
FIG. 5-10  Ventricular and intraparenchymal pressure monitoring systems. The stopcock on the ventricular monitoring system (right) is fixed and must be positioned at the same height as the tip of the intraventricular catheter to properly “zero” the system. The graduated cylinder may be adjusted vertically. The height at which the CSF fluid column just spills over into the cylinder, with respect to the zero value of the stopcock, is the ventricular fluid pressure. The intraparenchymal monitor (left) is “zeroed” before insertion, and the monitoring box position will not affect readings.
Use of ICP monitor

- Fiberoptic ICP monitor
- Standard ICP monitor hooked to our HP monitors
- Ask for help on how to use the equipment
- Sometimes the ICP is to be left open
- Overdrainage as well as underdrainage has been a problem at SUNY
- Do not inject anything into the catheter
- Please aspirate SLOW if asked by surgeons
Intracranial Compliance

• As intracranial pressure increases, the body compensates:
  • Shift CSF to spinal canal
  • Increase CSF absorption
  • Decrease CSF production
  • Decrease Ce vv capacitance

Can Compliance be measured??
• Saline Test … 1:4 ratio
Herniation Sites

- Sustained increases in ICP can lead to herniation and cerebral ischemia/infarction
  - Cingulate gyrus
  - Uncinate gyrus
  - Cerebellar tonsils thru foramen magnum
  - Any skull defect as in trauma
• Intracranial Pressure
• Intracranial Compliance
• Cerebral Metabolism
• Cerebral Blood Flow
• Blood Brain Barrier
• Evoked Potentials
Cerebral Metabolism

- $\text{CMRO}_2$ parallels activity of brain cells
- $\text{CMRO}_2$ is greatest in the grey matter of CCx
- $\text{CMRO}_2$ is 3-5 ml/100gm/minute in adults = 50ml/minute
- Brain uses 20% of total body oxygen consumption, most of it for ATP production
FIG. 1-5  Oxygen requirements of the normal brain. Values are those obtained in the canine. (From Michenfelder JD: The hypothermic brain. In Michenfelder JD: Anesthesia and the brain, New York, 1988, Churchill-Livingstone.)
Cerebral Metabolism

- 90% of brain metabolism is aerobic
- O2 reserves are low, consumption is high
- Narrow margin of safety with hypoxia
- Primary fuel is glucose and oxygen
- Hypoglycemia is not well tolerated
- Hyperglycemia leads to cellular acidosis during ischemia
• Intracranial Pressure
• Intracranial Compliance
• Cerebral Metabolism
• Cerebral Blood Flow: A sometimes confusing topic
• Evoked Potentials
CeBF and $\text{CMRO}_2$

- CeBF is proportional to $\text{CMRO}_2$
- CeBF: Grey matter versus white matter
- Total CeBF is about 20% of CO = 1 liter/min
- EEG is used clinically to judge adequate CeBF
CeBF and Children

- Newborn: lower than adults
- Infants and above CeBF: higher than adults
- Big Children (adults) = Average BF is 1 liter/min; Grey matter is 1.2 l/min and white matter is 0.3 l/min
Measuring CeBF

- Inhale RA inert gases: N₂O, Krypton and Xenon
- Intraarterial injection of inert gas: 133 Xe
- PET Scanning (Positive emission tomography)
  - Using radionucleotide that emits particles called positrons (11C, 15O, 13N, 18F)
  - Injection or inhalation
  - Need cyclotron; a very expensive piece of equipment
<table>
<thead>
<tr>
<th>Anesthetic /PHYSIO EFFECT</th>
<th>CMRO2</th>
<th>CeBF</th>
<th>CeBV</th>
<th>ICP</th>
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D=Decrease  I= Increase N=No change

* These are beneficial; Antiseizure, role in preventing focal not Global

** All change if hypoventilate

***Causes myoclonus not seizures; It can cause seizures in patients with
t

† except trimethapam

†† only secondary effects
• Volatile Anesthetics
  • CeBF increases (CeVR decreases) even though CMR02 decreases \( \Rightarrow \) luxury perfusion
  • CeBF increases with volatile anesthetics
  • ICP increases with volatile anesthetics
  • CO2 responsiveness maintained

Nitrous increases CeBF, CMR02 and ICP
Regulation of CeBF

- Cerebral Perfusion Pressure
- Autoregulation
  - Myogenic, metabolic (CMRO2)
- Extrinsic Mechanisms
  - $\text{PaO}_2$, $\text{PaCO}_2$, Temp, and viscosity
Cerebral Perfusion Pressure

- CeBF remains constant between MAP of 50-160 mm Hg despite changes in CePP (CePP = MAP - ICP or CVP)
- Beyond these blood flow is pressure dependant
- Normal ICP = 10 mmHG
- CPP < 50 mm Hg EEG slowing (w/ no anesthesia)
- CPP 25-40 mm Hg EEG flat (w/ no anesthesia)
Autoregulation

- Mechanism for autoregulation: thought to be myogenic and metabolic
- Myogenic involves the intrinsic property of cerebral blood vessels to control blood flow
  - Property of cerebral blood vessels to keep blood flow constant between a MAP of 60-160 mm Hg
  - Beyond these, the blood flow becomes pressure dependant
FIG. 2-4 Idealized depiction of pressure autoregulation in terms of CBF, cerebrovascular resistance, and arteriolar diameter. See text for further explanation. (From Young W: Clinical neuroscience lectures. Munster, Ind, 1991, Catbemart Publishing.)
Hypertension and CeBF/ICP

- Untreated HTN --> autoregulatory curve shifted to right
- If MAP > 150 --> “Autoregulatory breakthrough”
  - As MAP increase, CeBF goes up as it is now pressure dependant
  - BBB is disrupted and cerebral edema or bleeding can ensue
Autoregulation Loss

- Hypoxia
- Hypercapnia
- Ischemia
- Trauma
- CVA
Extrinsic Mechanisms for control of CeBF: Blood Gas Tensions

- \( \text{PaCO}_2 \): Direct proportion between 20-80
- CeBF changes 1-2 ml/100gm/1mm change in \( \text{PaCO}_2 \)
- Marked Hyperventilation: Cerebral Ischemia
- Marked Hypoventilation: pressure dependant CeBF
- Limitation of hyperventilation: after 6 hours, hyperventilation will not be effective in decreasing blood flow
Extrinsic Mechanisms for control of CeBF: PaO$_2$

- PaO$_2$ < 50 causes a rapid increase in CeBF
- Hyperoxia has little effect on CeBF
- Spinal cord reacts the same way as the cortex
FIG. 2-8 Influence of oxygen content (CaO₂) and PaO₂ on CBF. A, CBF is inversely proportional to CaO₂. B, Replotting the straight line in A by applying a sigmoid O₂ dissociation curve and taking the reciprocal produces the more familiar asymptotic curve of PaO₂ vs. CBF, which disguises the dependence of CBF on CaO₂. 5 kPa is approximately 40 mm Hg. (Redrawn by Lesser PJA, Jones JG. In Scarr C, Fehltman S, Sani N, editors: Scientific foundations of anaesthesia: the basis of intensive care, ed 2. Chicago, 1990, Year Book Medical Publishers, p 205, from original data by Brown MM, Wade JPH, Marshall J: Brain 108(pt 1):81, 1985.)
Extrinsic Mechanisms for control of CeBF: Temperature

- For every one degree decrease in body temp, $\text{CMRO}_2$ decreases 5% --> leads to fall in CeBF
- Brain temp of 20deg C --> isoelectric EEG
Extrinsic Mechanisms for control of CeBF: Viscosity

- Polycythemia is detrimental to CeBF and can cause a CVA
- HCTs less than 30 improve CeBF but at the expense of decrease O2 carrying capacity
- Studies suggest the optimal HCT to be between 30 and 40
Luxury/Steal Perfusion

$\text{CMRO}_2$ parallels activity of brain cells; volatile agents uncouple metabolism from CeBF needs. This is called *Luxury Perfusion*

*Cerebral Steal:*

Blood is shunted from an area of that is ischemic (and maximally vasodilated) to normal area. Setting: patient with CeVascular disease getting isoflurane dilates vasculature. Ischemic area gets blood shunted away from it
Review
Regulation of CeBF

- Cerebral Perfusion Pressure
- Autoregulation
  - Myogenic, metabolic (CMRO2)
- Extrinsic Mechanisms
  - $\text{PaO}_2$, $\text{PaCO}_2$, Temp, and viscosity
Last Topic: Evoked Potential
Neurolophysiologic Monitoring

- EEG
- Evoked Potentials (AEV, VEP, SSEP, MEP w/ tcMEP)
Neuroophysiologic Monitoring

- Cooperation important
- Confrontation not useful with the monitoring techs
EEG as a neuro monitor

- EEG monitors post-synaptic potentials in cerebral cortex
- Monitors CNS when clinical exam is not practical
- EEG depressed by: Hypoxia, hypotension, hypothermia and deep anesthesia
EEG Activation/Depression

- EEG Activation
  - Light Anesthesia
  - Seizures

- Most anesthetics initially activate and then depress EEG as the concentration increases; a clear biphasic response

- EEG Depression
  - Deep Anesthesia
  - Poor Cerebral Perfusion
EEG and Volatile Anesthesia

- 2 MAC Isoflurane --> Isoelectric EEG
- 1.2 MAC Desflurane --> Burst Suppression
- 1.5 Enflurane --> Burst Suppression
- 4 MAC Halothane --> Isoelectric EEG
- N₂O is unusual; it increases both frequency and amplitude; other volatile agents dec freq and amplitude
EEG and IV Anesthetics

- Benzodiazepines give a biphasic response; need large doses to get silence
- Barbiturates, Etomidate, Propofol --> Biphasic response then electrical silence
- Opioids --> resistant but eventual depression
- Ketamine --> EEG activation
Evoked Potential

**SSEP**

- SSEP tests the integrity of the sensory cortex and dorsal spinal columns
- Used in:
  - Resection of spinal tumors
  - Spine instrumentation
  - Carotid endarterectomy
  - Aortic Surgery
- 50% N20, 1 MAC VA, narcotics or TIVA with Propofol
44 yo W male undergoing cervical discectomy suddenly loses SSEP signal. What are the causes?

- 1. Real neural injury
- 2. Hypoxia and hypercarbia
- 3. Is patient hypotensive?
- 4. Was there a sudden bolus of anesthetic drugs or change in concentration of volatile agent?
BAEP

- Measures the integrity of cranial nerve 8
- Used in Posterior fossa craniotomies
Visual Evoked Potential

- VEPs measure the integrity of the optic nerve and upper brainstem
Volatile Anesthesia and EPs

- Resistance decreases: BAER, MEP>VEP>SSEP, tcMEP
- Keep volatile agents between 0.5-1 MAC; supplement with narcotics (not tcMEP)
- N₂O decreased the amplitude and increases latency of SSEPs
- Preexisting Myelopathy is a problem
Cortical Motor Evoked Potentials

- What is it??
- Anesthetic Technique:
  - N₂O is acceptable
  - Volatile anesthetic Ok but less than 0.2 MAC !!!
  - Narcotics and Propofol are agents of choice
Cortical Motor Evoked Potentials

- Technique: TIVA with propofol, ketamine, narcotics, No NMBs, No volatile agents, N20 fine
Non- Cortical Motor Evoked Potentials

- Any anesthetics
- Avoid NMB
- Does not mean using TIVA
IV anesthesia and EPs

- EP’s are preserved with barbiturates even at doses causing EEG silence
- Opioids: well tolerated
- Ketamine: Increases amplitude