Cerebral physiology and Cardiopulmonary Bypass

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Content

- *Neurologic morbidity*
- *CBF and cerebral physiology*
- *Cerebral physiology during CPB*
- *Pathophysiology behind neurologic morbidity*
- *Intraoperative management*
- *Cerebral Monitoring*
- *Deep hypothermic Circulatory arrest*
- *Hypothermia*
  - *Alfa-stat/pH-stat*

Is there a problem ? Yes the incidence of major neurologic morbidity is 1-6% and up to 16% in high risk patients

- *Type 1 (cerebral death, nonfatal stroke, TIA and coma)*
- *Type 2 (intellectual defects, confusion, agitation,seizures)*
  - *Post op 10-80%*
  - *3 to 6 months 5-20%*

Type 1

Ann Thorac Surg 2003;75:472.16184pt
4.6% stroke
CABG 3.8%
OPCAB 1.9%
Aortic valve surg 4.8%
Mitral valve surg 8.8%
CABG+Valve surg 7.4%
Double or triple valve surg 9.7%

Western Danish Heart Registry 7304 pt.
2.2 % stroke/TIA

Cognitive dysfunction 20-30 %, due to CPB ?

- No meaningfull cognitive outcome differences at 3 and 12 month in age- and health-matched cardiology and cardiac surgery patients
  - Ann Thorac Surg 2003; 75: 1377-84
- Incidence 25-30% in older patients undergoing major surgery
  - Lancet 1998;351:857-61

Cognitive decline is a function of the elderly brain and perioperative factors rather than specific intraoperative events due to bypass

Normal CBF/$CMRO_2$ physiology

- Brain = 2% of body weight
  - 15% of Cardiac output (splanchnicus =25%, Kidneys= 20%)
  - CBF ≈ 50 ml/100gr/min
- Energy
  - CMRO2 ≈ 3.5 mL/100gr/min
  - Glucose

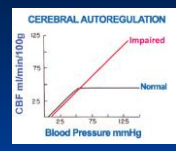
Normal CBF/CMRO2 physiology

Regulation

- Autoregulation
- CO2
- Metabolic
- Temperature
- Neurogen

Normal CBF/$CMRO_2$ physiology

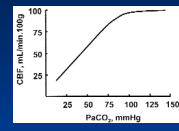
- Regulation af CBF



  - Autoregulation
    - The ability to maintain normal CBF despite changes in arterial blodpressure.
    - Autoregulation may be impaired by intracerebral disasters

Normal CBF/$CMRO_2$ physiology

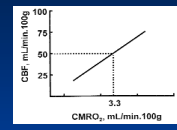
- Regulation af CBF



  - CO_2 reactivity
    - CBF changes linearly with $PaCO_2$ ≈ 20-80 mmHg
    - 2-4% change in CBF /mmHg
    - CO_2 changes tonicity in the vessels by changes in extracellulary pH
    - Hypotension reduces CO_2 reactivity

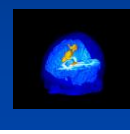
Normal CBF/$CMRO_2$ physiology

- Regulation of CBF



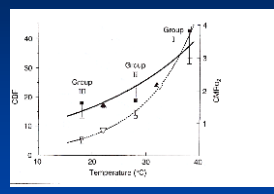
  - Metabolic regulation
    - $CBF - CMRO_2$ relation
      - CBF changes according to the metabolic demand of the brain

Pain-stimulus



Normal $CBF/CMRO_2$ physiology

CBF / CMRO2 and Temperature



CBF : solid line, CMRO2 : dashed line

Normal $CBF/CMRO_2$ physiology

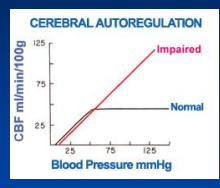
- Regulation of CBF

  - *Neurogen regulation*
    - *Less importance*
      - *Stimulation by sympaticus and parasympaticus fibers only lead to discrete changes i vessel tonicity.*
      - *Molecules which influence on CBF: NO, Endothelin, prostaglandines, free radicals ……*

Anesthesia: influence on $CBF/CMRO_2$

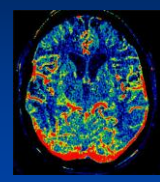
- *Inhalational anesthesia (isofluran/sevoflurane)*
- ↓ $CMRO_2$ → ↑ *CBF*
- *Intravenous anesthesia (propofol/barbiturates)*
- ↓ $CMRO_2$ ↓ *CBF*
- *Opioids (fentanyl/sufentanil)*
  - ↓ $CMRO_2$ ↓ *CBF*

CPB: MAP



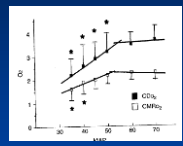
Cardiopulmonal bypass

- *Variables affecting CBF/CMRO$_2$*
  - *Blodpressure MAP*
  - *PaCO$_2$*
  - *Hct*
  - *Temperature*



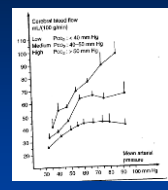
CPB :MAP & CO2

MAP



CBF is assured by MAP between 50 – 150 mmHg

CO2



During bypass an elevated PaCO2 Is associated with a higher CBF for any given MAP,

CPB : Flow, Pulsatility, ECC-duration

- CBF flow maintenance is not sufficient to quarantee cerebral perfusion if MAP is low
- No convincing evidence of beneficial physiologic effect of pulsativity
- No evidence of decrease in CBF with bypass time

CPB and hematocrit

- Hematocrit
  - Hemodilution reduces Hgb 1/3
  - ↓ viscositet , ↓ PVR

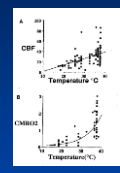
= ↑ CBF and CDO_2 → 

- Progressive hemodilution: CBF and O_2 extraction can no longer compensate reduced transport capacity

CPB: Temperature

↓C° increases ischemic tolerance

CMRO2 is reduced app.7% / C°

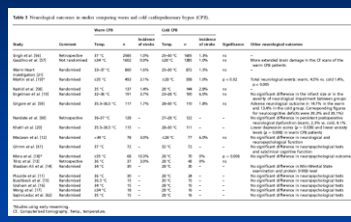


Autoregulation is ceased by profound hypothermia (15-20 c°)

Does temperature have any effect on neurologic outcome ? ??

CPB: Temperature

- Temperature : 20 studies Warm bypass: 33-37°
  Cold bypass: 23-32°



No clear evidence !!!!

CPB: Cerebral ischemia

- Cerebral ischemia:
  - When CBF is too low to meet cerebral O2 and glucose demand

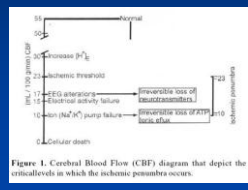
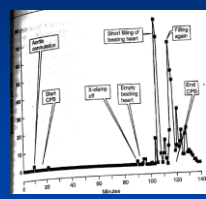


Figure 1. Cerebral Blood Flow (CBF) diagram that depict the criticallevels in which the ischemic penumbra occurs.

CPB and neurologic damage

- Embolism
  - Macro (focal defects)
    - Atheromas and debris
  - Micro (cognitive defects)
    - Air
    - Lipids
    - Cellular aggregates
    - Emboli from HLM
- Inflammatory response
    - Platelet aggregation an degranulation
    - Neutrophil and complement activation
    - Cytokine release
    - Endothelium dysfunction
- Cerebral Hypoperfusion

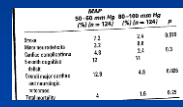
Number of embolic events per minute from cannulation to after end of bypass



CPB and neurologic damage

Surgical and technical interventions

- *Epiaortic scan*
- *Single clamp technique*
- *Off-bypass CABG*
- *Echo assesment of ventricular deairing*
- *Insufflation of CO2 in chest wound*
- *CPB circuit management*
  - *heparin coated*
  - *biocompatibility*
  - *filters*
- *Minimazation of cardiotomi suction*
  - *main source of lipid embolism*
- *MAP >50 mmHg*
  - *higher when pt.with DM or/and HA*
  - *1 RCT 1995*
- *Hct (ECC >15-18,post ECC > 30)*



J Thorac Cardiovasc Surg 1995;1110:1302

CPB and neurologic damage

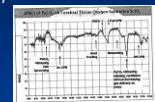
Cerebral monitoring

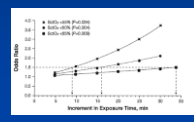
- *Jugular bulp $SjvO_2$*
  - *Focal events undetected*
  - *Research tool*
- *EEG*
  - *Only cortical activity*
  - *CPB and anesthesia artefacts*
  - *No clear relation to outcome*
- *Visual/somato evoked potentials*
  - *As for EEG*
- *Bi-spectral index (BIS)*
  - *Affected by temperature and anesthetic agents*
- *Near-infraread spectroscopy (NIRS)*

NIRS (INVOS,FORE-SIGHT,EQUANOX)

Continous, noninvasive transcutaneous assessment of regional brain oxygenation

- *Tissue oxygen saturation at microvascular level*
- *Extracerebral contamination is minimized*
- *Not influenced by depth of anaesthesia*
- *Pulsatility independent*
- *No ECC and temperature artefacts*
- *Outcome studies are evolving*
  - *Thresholds has to be defined*





Seminars in Cardiothoracic and vascular anesthesia 2008;12:60

Cardiopulmonal bypass

- Conclusion
  - Neurologic morbidity frequent on CPB
  - CPB variables which influence on CBF:
    - Temperature, $PaCO_2$, Hct, MABP
  - Choice of anesthesia has no documented effect on neuroprotection
  - No evidence on neurologic outcome on "warm" vs "cold" ECC

Deep hypothermic circulatory arrest

- Brain protection
  - Studies show evidence of decreased IQ
  - Other studies demonstrate no adverse effect on intellectual capacity
- Minimal adverse effects on psychomotor results with arrest times of about 35 min at 18°C (NEJM,1995;32:549)

Hypothermia: Alpha-stat, pH-stat

- Decreasing temp ⟶ increased solubility of gas ⟶ decread partial pressure
  - Temp. 40 ⇨ $PaCO2\downarrow \rightarrow$ pH 7.35, $CO2$ content unchanged
  - Temp. 20 ⇨ $PaCO2\downarrow \rightarrow$ pH 7.65, $CO2$ content unchanged
  - Alpha stat ($-CO_2$):
    - » Advantage: normal enzyme ativity, autoregulation, flow-metabolisme coupling
  - pH stat ($+CO_2$):
    - » Advantage : Increased CBF, more homogeneous cooling, increased oxygen delivery