Basics of Neuroanesthesia

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Disclosures:
No commercial interests
No conflict of interest

Goals for Neuroanesthesia

- Rapid induction and emergence
- Hemodynamic stability
- Maintenance of favorable cerebral hemodynamics
- Permit brain relaxation
- Permit electrophysiologic monitoring
- Lack of inherent toxicity
- Brain protection

Case presentation

- 55 yr male with glioma
- Neurologic exam
  - Mild LUE weakness
  - Mild headache
  - LOC normal
- Hypertension 146/94
- Diabetes BG 205 mg/dl

Management?

- Anesthetic choice
- Target blood pressure
- Hyperglycemia
- Brain protection

How do you proceed?
Selection of agents for Neuroanesthesia

- Should be influenced by an understanding of:
  - Rapid induction and emergence
  - Effects on CBF, CMR, CBV and ICP
  - Tolerance of brain to cerebral ischemia
  - Inherent toxicity:
    - Cellular injury
    - Epileptogenesis

Selection should be based on outcome studies

CBF effects of volatile anesthetics

Comparative Effects of Anesthetics on Cerebral Vasodilation
Cerebral blood flow and volume relationship

Effects of Sevoflurane, Propofol and Nitrous Oxide on Cerebral Hemodynamics

A Comparison of Remifentanil and Fentanyl in Patients Undergoing Surgery for Intracranial Mass Lesions
Comparison of propofol and volatile agents for maintenance of anesthesia during elective craniotomy procedures: systematic review and meta-analysis

- Cerebral hemodynamics
  - ICP
  - CPP
- Brain relaxation score
  - Slack
  - Mild herniation
  - Moderate herniation
  - Severe herniation
- Recovery
  - Extubation
  - Aldrete discharge score
  - 5 mm less with propofol
  - Slightly greater with propofol
  - No difference
  - No difference


Clinical Evaluation of Intracranial Hypertension

- Symptoms
  - Headache
  - Nausea and vomiting
  - Visual symptoms
  - Neurologic deficits
- Signs
  - Altered LOC
  - Pupillary dilation
  - Contralateral hemiparesis
  - Neurologic deficits
  - BP, HR

  Herniation

  - Radiologic findings
    - Mass lesion
    - Effacement of lateral ventricle
    - Midline shift
    - Full basal cisterns
    - Herniation of brain tissue

Blood pressure

What is the optimal blood pressure?
Cerebral Autoregulation

MAP, PaCO₂, PaO₂ (mmHg)

CBF (ml 100 gm⁻¹ min⁻¹)

MAP and Collateral Flow

Penumbra
Autoregulation abolished
rCBF reduced
Autoregulation attenuated
Normal brain
Autoregulation intact

Blood flow to penumbra dependent on perfusion pressure

Collateral Cerebral Circulation

MCA
Variations in Circle of Willis

Variable or incomplete Circle in ~ 30% of patients

Cerebral Autoregulation and Hypertension

Hypertension may encroach on cerebral reserve

Arterial Carbon Dioxide Tension

What is the optimal level?
Beneficial Effects of Hyperventilation

- Reduction of brain bulk
- Permits less aggressive brain retraction
- Reduction in ICP
- Redistribution of blood flow (inverse steal)
- Reduction of cerebral acidosis

Can hyperventilation decrease cerebral blood flow and increase ischemic neuronal injury?

PaCO₂ and Collateral Flow

Hyperventilation can potentially reduce collateral blood flow to the penumbra

Hyperventilation

- Use as any drug
- Define indications
- Minimize PaCO₂ reduction
- Withdraw when it is no longer needed

“Routine hyperventilation” should be discouraged
Most anesthetics in use today have been shown to reduce ischemic cerebral injury. How much protection?

Effect of Etomidate on Ischemic Brain

Effect of Etomidate on Ischemic Brain

CEA GALA Trial

- Stroke rate within 30 days of CEA
- Local anesthesia: 3.7%
- General anesthesia: 4.0%

Question:
If anesthetics reduce vulnerability to ischemia, why are strokes rates between GA and LA same?
No Association between Intraoperative Hypothermia or Supplemental Protective Drug and Neurologic Outcomes in Patients Undergoing Temporary Clipping during Cerebral Aneurysm Surgery

Findings from the Intraoperative Hypothermia for Aneurysm Surgery Trial

- Temporary clip 441
  - Hypothermia 208
  - Normothermia 233
- Additional protection
  - Thiopental 158
  - Etomidate 20

No benefit of “neuroprotective” anesthetics

Hindman B et al, Anesthesiology 2010;112:86-101

Hyperglycemia

Should hyperglycemia be treated?

Glucose Levels and Stroke

- EEG suppression at levels less than 20-30 mg/dl
- Significant neuronal loss after 30 min of EEG silence

Auer Metab Brain Dis 2004;19:169-175
Poststroke Hyperglycemia
Natural History and Immediate Management
Christopher S. Gray, MD, Anthony J. Hildreth, MPH, George K.M. Alberti, PhD, Janice E. O’Connell, MB, ChB, on behalf of the GISP Co-investigators

- 452 patients randomized to GKI or saline
- Target glucose 72 - 126 mg/dl
- Rapid control of glucose with GKI
- 9% had hypoglycemia (glucose < 72 mg/dl)

No difference in outcome

Gray et al. Stroke 2004;35:122-134

Summary
- Choice of anesthetics
  - No significant differences between volatile and IV anesthetics
  - Use regimen according to preference
  - Switch to TIVA if brain “tight”
- Blood pressure
  - 10 – 20% below baseline
  - May need to increase if indicated
- Hyperventilation
  - Use as drug; no prophylaxis
- Blood glucose
  - Liberal target; treat if > 180 mg/dL