ANESTHESIA FOR NEUROVASCULAR SURGERY

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CONFLICT OF INTEREST AND DISCLOSURES

• Professor, but very busy clinical anesthesiologist
• I do not accept honoraria for lectures
• Consulting fees (case review, industry FDA activity) are paid directly to charity (e.g. Oregon Food Bank, FAER etc)

PRESENTATION OBJECTIVES

• Gain the knowledge to provide state-of-art perioperative anesthetic management for patients requiring neurovascular surgery
• Gain the knowledge to optimize conditions to optimize outcomes following neurovascular surgery

TOPICS COVERED:

• Anesthesia for carotid endarterectomy
• Anesthesia for cerebral endovascular clot retrieval
• Anesthesia for cerebral aneurysm surgery
• Anesthesia for cerebral AVM resection

CEREBRAL ISCHEMIA:

CAROTID ENDARTECTOMY
POST-STROKE CLOT RETRIEVAL

79 Y.O. MAN WITH 85% LEFT CAROTID STENOSIS. HISTORY OF TIAS.

• Will you do the case awake or under GA?
• How will you monitor the patient?
• Will you recommend that the surgeons use a shunt or not?
• What anesthetic agents will you use?
PREOPERATIVE

Optimize CV System to minimize risk of perioperative are MI

- Poor reserve of high energy phosphates
- Poor reserve of metabolic substrates
- High metabolic requirement
- Poor functional redundancy (functional deficit, even with relatively little damage in “eloquent” areas of brain)

RA VS GA

- Biggest advantage of RA is neurologic exam during clamp/shunt. There may be some reduced CV risk.
- Decision should be collaborative between surgeon, anesthesiologist and patient

PREVENTING INJURY FROM ISCHEMIA

- Monitoring for ischemia
- Shunt: plastic bypass catheter can maintain perfusion during removal of plaque but may also cause plaque emboli during placement
- Precise BP and PaCO2 management
- Temperature control
- Glucose management
- Pharmacologic treatment

CESSATION OF BLOOD FLOW TO BRAIN (CEREBRAL ISCHEMIA)

Appropriate monitoring for ischemia
- Neuro-monitoring (EEG, SSEP, MEP)
- CBF: Xe, TCD (MCA flow and emboli detection), laser Doppler
- Stump Pressure
- Neurologic exam during awake surgery (RA)
- Near-infrared reflected spectroscopy (NIRS)
- Combined stump pressure and NIRS
To shunt or not to shunt?

- During GA: Selective shunting with EEG monitoring is associated with better outcome than shunting all patients or no patients (Salvian AJ et al., Cardiovasc Surg 5:481, 1997; Plestis KA, J Vasc Surg 25: 620, 1997)
- During GA: Selective shunting with stump pressure plus NIRS (Findlay JM et al., Can J NS 44: 692, 2017)
- During RA: Selective shunting based on the Neurologic Examination is best.
- Critical to engage surgical strategies to minimize plaque emboli during shunt placement

PREVENTING ISCHEMIA (2)

The association of the difference between baseline mean arterial pressure (MAP) and average MAP during GA (ΔMAP) with the score on the modified Rankin Scale (mRS) at 90 days.

- Maintain MABP no less than normal baseline (ephedrine may be better than PE (from NIRS studies))
- Maintain adequate oxygen carrying capacity (Hb and PaO2)
  - Hb 11-12 g/dl is associated with less vasospasm & better outcomes after aSAH (Sun J et al., NeuroRpt 26:263, 2015; Stein M et al., J Clin Neurosci 22:234, 2015)
- Avoid hyperventilation

PREVENTING ISCHEMIA (3)

- Standard ASA monitors; Timing of a-line dependent on pt’s CV situation
- CV Stable induction (e.g. Etomidate, Roc/Sux, PE/Ephedrine, Esmolol)
- Neuromonitoring needs (TIVA, no prolonged NMJ blockade for MEPs?)
- Plan for increased BP during carotid clamp: reduced BP during reperfusion (suture line pressure, excessive brain perfusion)
- Anesthetic allowing rapid emergence for Neuro-Exam; Deep extubation may prevent coughing and suture line stress
- Prepare for acute BP manipulation during surgical dissection of carotid bifurcation secondary carotid baroreceptor manipulation (Rx with lidocaine by surgeon in bifurcation)
HANDY TO HAVE IMMEDIATELY AVAILABLE

• Blood pressure raising:
  • Bolus: Phenylephrine (50-100 mcg), Ephedrine (5-10 mg), Vaso (1-2 units)
  • Infusions: PE (0.2 mcg/kg/min), NE (0.02 mcg/kg/min), Vaso (1-6 U/hr)

• Blood pressure lowering:
  • Bolus: Labetalol (5 to 10 mg every 5 minutes), Nicardipine (100 to 500 mcg), or esmolol (10 to 20 mg).
  • Infusions: labetalol (0.5 to 2 mg/minute), nitroprusside (0.1 to 4.0 mcg/kg/min), nitroglycerin (0.1 to 4 mcg/kg/min), nicardipine (initial 3 to 6 mg/h titrated to a max of 15 mg/h), or esmolol (50 to 300 mcg/kg/min)

79 Y.O. MAN WITH 85% LEFT CAROTID STENOSIS. HISTORY OF TIAS.

• Should this be done as a CEA or Stent? Carotid stenting is associated with a higher incidence of procedural stroke/death (Muller MD et al., 49:2715, 2018). If stent, deploying stent may be painful, as typically performed with sedation.
• Will you do the case awake or under GA? Primarily up to the surgeon: ETT for CEA; LMA for stenting
• How will you monitor the patient? ASA routine monitors plus A-line; Neuromonitoring up to surgeon
• Will you recommend that the surgeons use a shunt or not? I prefer shunt guided by EEG change during clamping.
• What anesthetic agents will you use? Tight control of BP, PaCO2, glucose, HR (high incidence of post-op MI) and tailored to optimize neuromonitoring

MECHANISTIC TREATMENT TO PREVENT BRAIN INJURY FROM CEREBRAL ISCHEMIA (TEMPORARY CLIP PLACEMENT)

- Reduce brain metabolism
- Calcium: blocking of VDCC, AOCC (EAA inhibitors) or release from ER
- Sigma receptor agonist (decrease release of EAA or impair their ability to act at receptor)
- Oxygen radical scavengers
- Inhibitors of nNOS and iNOS
- Stimulators of eNOS
- Protease inhibitors
- Anti-inflamatory/adhesion agents

MECHANISTIC TREATMENT TO PREVENT BRAIN INJURY FROM CEREBRAL ISCHEMIA

Mechanistic treatment only effective in rodents

EXPERIMENTAL PHARMACOLOGIC TREATMENT TO PREVENT INJURY FROM CEREBRAL ISCHEMIA DURING CEA

- If anesthetics were neuroprotective during CEA, we would expect better outcomes following GA vs RA
- Melatonin: (Zhao Z et al., J Pineal Res 65::e12521, 2018)
  • Decreased brain injury and increased brain anti-inflammatory capacity in rats subjected to transient focal ischemia
  • Double blind RCT in patients treated with oral Melatonin (6 mg/day) for 3 days prior to and 3 days following CEA decreased evidence of brain injury (S100-Beta) and increased systemic anti-inflammatory capacity
POSTOPERATIVE COMPLICATIONS

• Cerebral micro-emboli from plaque disruption
• BP Control: Instability due to baroreceptor injury and pain; SBP goal 100-150
  - HTN could lead to “cerebral hyperperfusion syndrome (cerebral edema, ICH) and suture line
    rupture
  - Hypotension: Stroke
• Slow emergence (anesthesia, hypothermia, residual NMJ blockade vs. Stroke)
• Neck Hematoma: residual anticoagulant +/- HTN, acute airway emergency
  - Associated with increased incidence of post-op MI and Stroke
• Vocal Cord Paralysis: Surgery associated recurrent laryngeal nerve injury or
  compression. Results in VC adduction, hoarseness and airway obstruction (if
  previous injury on contralateral side)

ANESTHESIA FOR CLOT RETRIEVAL

65 YEAR OLD MAN WITH RIGHT MCA OCCLUSION

• History of atrial fibrillation on aspirin treatment
• PMH: Hypertension, Type 2 diabetes, high cholesterol, inactive
• PE: BMI 32, MP 3 airway, NPO for 6 hours after a
  light meal
• Interventional radiologist wants to proceed with clot retrieval

Additional preoperative assessment?
GA vs Sedation?
Specific plans for neuroprotection?
RETROSPECTIVE STUDIES CANNOT EXCLUDE THE LIKELIHOOD THAT SICKER PATIENTS WERE CHOSEN FOR GA AND NO STUDY WAS CONTROLLED TO ADDRESS DIFFERENCES IN BLOOD PRESSURE OR PACO2.

ANESTHESIA FOR CLOT RETRIEVAL

- Consider sedation rather than GA
- Avoid hyperventilation
- Avoid hypotension
- Consider low dose (0.5 mg/kg; single dose) ketamine
- If GA: Desflurane for anesthetic maintenance
CEREBRAL ANEURYSM SURGERY

ACUTE SUBARACHNOID HEMORRHAGE

SACULAR CEREBRAL ANEURYSMS (THE ISSUES)

• Different types: presenting signs and symptoms
• Natural course
• Rupture
• Cerebral vasospasm: pathophysiology and treatment
• Surgical treatment; anesthesia issues, suggested approach
• Endovascular treatment; anesthesia issues, suggested approach
• Common associated problems; hyponatremia, hydrocephalus
• Neuromonitoring optimization, neuroprotection

TYPICAL PATIENT PROFILE FOR CEREBRAL (SACULAR) ANEURYSM

• 3% of general population; 30% of these have multiple aneurysms
• Present at age 40 to 60
• No gender preference at 50 or younger but much more common in women, older than 50
• Most (particularly small) aneurysms do not rupture: rupture rate approximately 10 per 100,000
• Only 1/3 of patients with aneurysmal SAH (A-SAH) have a good outcome.
• 85% occur at vascular branch point in anterior circulation.
• Inc. incidence of A-SAH with HTN, ETOH, Smoke, advanced age in women (dec estrogen)
• Dec incidence of A-SAH with high chols and exercise (statins may increase risk of SAH)
• Variety of genetic diseases predispose to presence of cerebral aneurysm and increased risk of rupture.

ANEURYSM RUPTURE

• Risk of rupture is higher as the aneurysm gets bigger, particularly more than 7 mm in diameter
• Posterior circulation aneurysms rupture with a higher frequency than anterior circulation aneurysms
• No clear association with activity or stress
• Surgeon decision regarding intervention vs. observation relates to size, location and patient age/co-morbidities

SAH CONSEQUENCES

• Increased ICP and immediate death (most severe), severe headache/altered LOC or minimal symptoms
• Delayed hydrocephalus from adhesions or reduced CSF resorption in arachnoid granulations; often require permanent LP/VP shunt
• Delayed cerebral ischemia (AKA vasospasm)
• Stress cardiomyopathy (myocardial ischemia in distribution of sympathetic nerves, rather than vascular territory)
• Hyponatremia

Etiologies of thunderclap headache

Most common causes of thunderclap headache:
- Subarachnoid hemorrhage
- Reversible cerebral vasoconstriction syndrome (RCVS)

Conditions that less commonly cause thunderclap headache:
- Cerebral infection (e.g., meningitis, acute complicated sinus)
- Cerebral venous thrombosis
- Cerebral artery dissection
- Spontaneous intracranial hypertension
- Acute hypertensive crisis
- Posterior reversible leukoencephalopathy syndrome (PRES)
- Intracranial hemorrhage
- Ischemic stroke

Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage • UpToDate
CAUSES OF SAH

- Ruptured cerebral aneurysm
- Trauma
- AVM/fistulae
- Vasculitides
- Arterial dissection
- Amyloid angiopathy
- Bleeding diatheses
- Illicit drugs (Cocaine/Amphetamines)

REBLEEDING

- Approximately 20% chance of rebleeding within first 24 hours (particularly first 6 hours)
- Acute rebleeding increases risk of death 12 fold
- Only effective treatment to prevent rebleeding is surgery/endovascular treatment
- Rebleeding during anesthesia induction is usually fatal
- Antifibrinolytic therapy (e.g., TXA) decreases risk of rebleeding but increases risk of vasospasm
- Higher rates in patients after endovascular treatment compared to surgery
SURGERY VS. ENDOVASCULAR

• For patients who fit the specific criteria (based on previous randomized studies) for endovascular treatment, outcomes are better than with surgery
• Endovascular more often associated with rebleeding (early and late)
• Endovascular associated with lower rate of post event seizures
• There is no long-term benefit of endovascular vs. surgery for patients with high grade SAH

Table 1: Intraoperative factors contributing to intraoperative aneurysm rupture

<table>
<thead>
<tr>
<th>Factors</th>
<th>Controversies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Upper limit of blood pressure</td>
</tr>
<tr>
<td></td>
<td>Poorly controlled BP/controlled BP</td>
</tr>
<tr>
<td></td>
<td>Chronic/excessive hypertension</td>
</tr>
<tr>
<td>Anesthetic factors</td>
<td>Sympathetic responses</td>
</tr>
<tr>
<td></td>
<td>(intubation/ventilation)</td>
</tr>
<tr>
<td>ICP</td>
<td>Coughing/gagging</td>
</tr>
<tr>
<td></td>
<td>Sudden decrease in ICP during hyperventilation, use of large dose mannitol, and CSF drain</td>
</tr>
<tr>
<td>Maneuvers</td>
<td>Valvular, application of PEEP (upper limit)</td>
</tr>
<tr>
<td>Concomitivities</td>
<td>COPD, CAD, and hyperalimentation</td>
</tr>
</tbody>
</table>

Table 2: Diagnosis of IAR

<table>
<thead>
<tr>
<th>Method</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Clinical</td>
<td>Hypertension, bradycardia, and arrhythmias</td>
</tr>
<tr>
<td></td>
<td>Blown pupil</td>
</tr>
<tr>
<td>(2) Surgical</td>
<td>Increase more from surgical incision</td>
</tr>
<tr>
<td></td>
<td>Brain bulge, Hematoma</td>
</tr>
<tr>
<td>(3) Monitoring</td>
<td>Sudden rise in ICP, presence of pathological waves</td>
</tr>
<tr>
<td>ICP</td>
<td>No diastolic flow to reversal of diastolic flow</td>
</tr>
<tr>
<td>TCD</td>
<td>Cerebral oximetry</td>
</tr>
<tr>
<td></td>
<td>Sudden decrease in values</td>
</tr>
</tbody>
</table>

SURGICAL DISSECTION

• If surgeons place lumbar drain for brain relaxation, will that increase transmural pressure and result in aneurysm re-rupture?
• Re-rupture is less likely but also may occur with early, aggressive brain relaxation with mannitol or hypertonic saline
• Will hyperventilation result in cerebral ischemia?
• Define BP goals with surgeon for period of dissection, during clip (temporary) placement and following placement of permanent clip
• Will neuromonitoring be used and if so which modalities? Will TIVA be required?
• Risks associated with surgical position; assess for VAE risk and aggressive head turning
• Effects of acute increase ICP on cardiac function
• Hyponatremia common: SIADH vs. Cerebral Salt Wasting; dehydration associated with more frequent vasospasm-associated cerebral ischemia

ANESTHETIC MANAGEMENT FOR SURGICAL ANEURYSM TREATMENT
Cardiovascular complications are common after brain injury and are associated with increased mortality and morbidity. Neurogenic cardiac injury is related to brain injury-induced catecholamine and inflammatory responses. The neurogenic stunned myocardium (NSM) syndrome is caused by local release of norepinephrine from myocardial sympathetic nerve terminals. The NSM syndrome is characterized by ECG changes, cardiac arrhythmias, release of biomarkers of cardiac injury, and left ventricular dysfunction. Neurogenic cardiac abnormalities are often transient and management should focus on general supportive care and treatment of the injured brain.

NEUROGENIC CARDIAC DYSFUNCTION

- No history of cardiac problems
- Temporal relationship between brain injury and cardiovascular abnormalities
- ECG changes in isolation
- Modest elevations in cardiac troponin (cTnl)
- New onset LV dysfunction
- Cardiac wall motion abnormalities that do not correspond with coronary vascular territories,
- Inconsistency between echocardiographic and ECG findings,
- Inconsistency between cTnl and LV ejection fraction (cTnl <2.8 µg litre$^{-1}$ in association with LV ejection fraction <40%).

BP GOALS DURING SURGERY IN PATIENTS WITH CEREBRAL ANEURYSM

- Include surgeon in discussion
- Is elevated BP secondary to elevated ICP; BP treatment results in cerebral ischemia?
- Pts with unruptured aneurysm: SBP less than patient's normal BP
- CPP of 60 or greater if monitoring ICP; treatment should focus on avoiding acute BP spikes from patient's preoperative values.

PATIENTS WITH CEREBRAL VASOSPASM

- Occurs in 20-30% of patients following Aneurysm SAH
- Symptoms start around day 3 following SAH and peak at day 7/8; usually gone by 14 days
- Structural thickening of arterial wall (media); not true spasm
- Diagnosis made by TCD and signs/symptoms (vs. Hydrocephalus)
- Treatment is oral nimodipine (only agent known to improve outcomes in patients); should be continued intraoperatively
- Normovolemia, avoiding hypotension, removal of SAH blood (surgery); intraluminal treatment (balloon or vasodilators)
- No benefit of HHH treatment (Gathier C et al., Stroke 49:76, 2018)
INTRAOPERATIVE ANESTHETIC MANAGEMENT

- Continue nimodipine via OG tube
- Hb 8 to 11.5 (higher Hb associated with less vasospasm)
- Be prepared for administration of vasoactive medications, including adenosine for cardiac pause during clip placement
- External pads if adenosine anticipated
- Standard monitors plus arterial line (placement prior to or following induction of anesthesia?)
- Two large bore (16 ga or larger) IVs
- Minimize inhalated anesthetics if neuromonitoring.
- BIS/EEG if plan TIVA and/or burst suppression

CRITICAL INTRAOPERATIVE EVENTS

- Anesthesia induction: Lower BP is better
- Skull pinning and incision: Avoid HTN: Scalp block (0.5% ropivacaine), Esmolol, Short-acting Opioid (500 mcg Alfentanil), Propofol
- Dissection: Avoid movement (NMB) blockade or GA/norepi
- Temporary clipping: Increase BP per surgeon with pressor; discuss "neuroprotection" Propofol neither helps or hurts
- Adenosine for flow reduction during clip placement: 0.4 mg/kg for 20-40 seconds of flow cessation (can be repeated if necessary). TC pacing pads should be placed prior to adenosine administration
- Intraoperative rupture: call for help, massive transfusion, lower BP, Adenosine?, brain protection?
- Confirmation of correct placement: Angi, micro Doppler, indocyanine green (artifactual detail)
- End of case: plan for emergence for neuro-exam regardless of plans for extubation

VENOUS AIR EMBOLISM DURING CRANIOTOMY: Rapid overview

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Clinical signs</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Craniotomy in the sitting position</td>
<td>Air visible on TEE</td>
<td>Notify surgeon</td>
</tr>
<tr>
<td>Surgery involving major intracranial venous sinuses; air entry possible from any venous opening above the level of the heart</td>
<td>Change in precordial Doppler tone</td>
<td>Flood field with saline</td>
</tr>
<tr>
<td></td>
<td>Decrease in ETCO</td>
<td>Repair site of air entry</td>
</tr>
<tr>
<td></td>
<td>Decrease in SPO</td>
<td>Lower the head</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td>May result in bleeding from air entry site</td>
</tr>
<tr>
<td></td>
<td>Increase in CVP</td>
<td>May limit surgical access to operative site</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Discontinue NO and administer 100 percent O</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aspirate air from central venous catheter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Discontinue PEEP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular support</td>
</tr>
</tbody>
</table>

TEE: transesophageal echocardiography; ETCO: end-tidal carbon dioxide; SPO: oxygen saturation; CVP: central venous pressure; NO: nitrous oxide; O: oxygen; PEEP: positive end-expiratory pressure; VAE: venous air embolism.

* Clinical signs depend on severity of VAE.

VENOUS AIR EMBOLISM DURING CRANIOTOMY: Up to date 2018

PREVENTING INJURY FROM CEREBRAL ISCHEMIA

- Monitoring for ischemia: Neurologic exam, EEG, SEPs, Near Infrared spectroscopy Jugular bulb oxygen saturation, paratrend, micro-dialysis
- Facilitating perfusion: BP control (particularly in a non-autoregulating vascular bed)
- Avoiding unnecessary hyperventilation
- Avoiding hyperthermia
- Avoiding hyperglycemia and hypoglycemia

MECHANISTIC TREATMENT TO PREVENT BRAIN INJURY FROM CEREBRAL ISCHEMIA (TEMPORARY CLIP PLACEMENT)

- Reduce brain metabolism
- Calcium: blocking of YDCC, AOCC (EAA inhibitors) or release from ER
- Sigma receptor agonist (decrease release of EAA or impair their ability to act at receptor)
- Oxygen radical scavengers
- Inhibitors of nNOS and iNOS
- Stimulators of eNOS
- Protease inhibitors
- Anti-inflammatory/adhesion agents

43 Y.O. MOTHER OF THREE WITH MULTIPLE CEREBRAL CEREBRAL ANEURYSMS

- As the surgeons approach the aneurysm they indicate that they are going to use a temporary clip and ask you to institute "brain protection": What are you going to do?
  A. Propofol!
  B. Desflurane?
  C. Hypothermia?
  D. Hypertension?
  E. Ketamine?
MECHANISTIC TREATMENT TO PREVENT BRAIN INJURY FROM CEREBRAL ISCHEMIA

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- Protease inhibitors
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Mechanistic treatment only effective in rodents

HYPOTHERMIA?

INHALED ANESTHETICS?

HYPOTHERMIA IS NO LONGER INDICATED AS AN TREATMENT TO PREVENT POST-ISCHEMIC NEUROLOGIC INJURY (EXCEPT IF YOU ARE TAKING THE 2015 ACLS RECERTIFICATION EXAMINATION). EFFICACY OF REGIONAL CEREBRAL HYPOTHERMIA (MICROCATHETERS) IS CURRENTLY UNDER INVESTIGATION.
**DESLURANE PREVENTS ISCHEMIA-INDUCED ACIDOSIS IN HUMANS**

**Improved Collateral Flow?**

Hoffman We Anesthesiology 88:1188, 1998

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**PROBLEMS WITH HIGH DOSE DESFLURANE**

Cost
Prevents neuromonitoring

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**PROPOFOL?**

- Antioxidant (free rad scavenger) (Aarts et al., FBS Lett 357:83, 1995)
- Decrease in CMRO2 and CBF/ICP
- Prevents neuronal injury in an In Vitro model of anoxia-reoxygenation (Dia-Cri et al., Brain Res 830:16, 1998)
- Prevents neuronal injury (as compared to isoflurane) in a model of transient anoxia reoxygenation (Young et al., Eur J Neuroscience 4:322, 1996 but not permanent MCAO in rats (Cox et al., Acta Anesthesioloica 33:99, 1994)
- Does not improve neurologic outcome in cats exposed to incomplete global ischemia (Yokota et al., Brain Res 288:199, 1983)
- At burst suppression doses, does not improve neurologic outcome following valve surgery (Robert, Anesth 90: 1255)

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**NITROUS OXIDE**

- Decreases efficacy of Barbiturates as a neuroprotectant (Warner et al., Anesthesiology 73:686, 1990)
- Decreases efficacy of isoflurane as a neuroprotectant (Baughman et al., Anesthesiology 70:767, 1989)
- May worsen outcome because of an increase in CMRO2
- Alone provides inadequate anesthesia - high catecholamine concentration may contribute to worse outcome
- Increases transient neurologic deficits in humans exposed to temporary clipping for aneurysm surgery (Pästernak J et al., Anesthesiology 110: 563, 2009)

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In summary, use of nitrous oxide in a group of patients at high risk for cerebral ischemia had no detrimental effect on long-term gross neurologic or neuropsychological function. Nitrous oxide use was associated with an increased risk of developing DIND, but this did not correlate with long-term outcome. …There is no evidence to support the unconditional avoidance of nitrous oxide in patients at risk for cerebral ischemia.
Alpha 2 agonists do not protect human brain from ischemia induced injury. May prevent post-op delirium following deep GA.

Importance of Metabolic Substrate

- Severe hypo- or hyper-glycemia is detrimental to neurologic outcome following ischemia (Sieber et al)
- Alteration in glucose value effects metabolic supply and intracellular pH (Hurn et al)
- Tight control of blood glucose has never been shown conclusively to prevent ischemic brain injury in humans
- My practice: maintain glucose >80 and <180 mg/dl.

MY APPROACH FOR INTRAOPERATIVE CARE FOLLOWING A-SAH

- Pre-oxygenate
- "Modified" Rapid Sequence Induction
- Dose induction drugs (Propofol, fentanyl, rocuronium) to avoid hypertension
- DL with deep GA (+/- LTA) to avoid BP spikes
- 2 Large Bore (16 ga or larger) IV and arterial line (placed after induction)
- If CVP required for access (large bore not available), consider femoral or axillary/subclavian
- +/- Scalp block
- Cardiac pacing pads applied if plan intraoperative adenosine
- Maintenance with Neurmonitoring friendly agents (TIVA+); Propofol or Alfentanyl for pin placement; BIS or EEG to facilitate timely emergence
- OG tube for nimodipine; push syringes to increase or decrease BP
- Plan for emergence for neurologic examination and extubation as indicated

INTRAOPERATIVE ADMINISTRATION OF ANTICONVULSANTS

- Lack of consistent efficacy to reduce post-operative seizures
- CV depressant effects of anticonvulsants are worse during GA
- Change effective half life of NMJ blocker
- Acute administration inhibit Ach release at the nerve terminal resulting in acute increased sensitivity to NDMR
- After 2 weeks of therapy they cause increased Ach receptor number
- Induce liver metabolism of NDMR
- Increase release of acute phase reactant proteins that bind to many drugs (changing the volume of distribution of the NDMR drugs)

CEREBRAL ARTERIO-VENOUS MALFORMATION (AVM)

- Presenting signs and symptoms (HA, Seizures, Hemorrhage)
- Natural Course (Bleeding)
- Associated cerebral aneurysms
- Embolization, Gamma knife and/or open surgery
- Anesthesia issues
- Normal Pressure Breakthrough
AVM TREATMENT

- Surgery is standard for most
- Radiosurgery (Gamma Knife) should be considered for deep, small AVM without bleeding; cure can occur in 1 to 3 years
- Embolization is not a treatment for cure. It is often implemented prior to surgery to decrease surgical risks

ANESTHETIC MANAGEMENT OF SURGICAL REMOVAL OF CEREBRAL AVM

- Same as for cerebral aneurysm PLUS very large bore access if incompletely embolized preoperatively
- Not usually associated with vasospasm or hyponatremia

NORMAL PRESSURE BREAK THROUGH (COMPULSIVE AND COLLABORATIVE MANAGEMENT OF BP)

- “The occurrence of multifocal areas of hemorrhage associated with cerebral edema in the postoperative of totally-resected high-flow arteriovenous malformations (AVMs)” Mattei TA BJNS 26:786, 2012
- Impaired autoregulation
- Abnormal capillaries
- Impaired venous drainage

TAKE HOME POINTS

- Anesthesia for carotid endarterectomy
  - Avoid poor perfusion during clamping; anesthetic tailored for optimal neuro-monitoring; compulsive management of CV variables to avoid perioperative MI and post-op bleed; post-op recognize and treat neck hematomas
- Anesthesia for cerebral endovascular clot retrieval
  - Timely implementation of anesthetic; avoid hyperventilation; adhere to anesthetic request (MAC vs GA) of proceduralist
- Anesthesia for cerebral aneurysm surgery
  - Avoid HTN prior to clip placement; CV control in compliance with surgeon goal during temporary clipping; anesthetic tailored for neuromonitoring; vascular access to support rupture; close control of serum electrolytes; vigilance regarding VAE
- Anesthesia for cerebral AVM resection
  - Similar consideration as aneurysm care plus BP control to avoid normal pressure break through