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Neuroanesthetic Emergencies Critical Event Treatment Guides

The cognitive aids that follow were designed to aid in the clinical care of patients. As stated by the American Society of Anesthesiologists:

“Guidelines are systematically developed recommendations that assist in the decision making process. Practice guidelines are not intended as standards or absolute requirements, and their use cannot guarantee any specific outcome. Practice guidelines are subject to revision as warranted by the evolution of medical knowledge, technology, and practice. They provide basic recommendations that are supported by a synthesis and analysis of the current literature, expert opinion, open forum commentary, and clinical feasibility data.”

The purpose of these cognitive aids is to provide a resource for the anesthesia team when faced with a neuroanesthetic emergency. They are not a protocol that anesthesiologists are obligated to follow. Treatment may vary based on clinical decision-making.

These cognitive aids were developed by a SNACC education taskforce. Prior to publication the entire SNACC membership had the opportunity to review and revise.

#1 Acute Stroke

Alteration in level of consciousness or acute neurologic deficit (focal or global). Most common presenting symptoms: hemiparesis, visual loss, dysarthria, facial drop, vertigo, ataxia or sudden decrease in the level of consciousness.

Stabilize Patient:

ABCs

Establish time of onset (time last seen normal).

Supplemental oxygen to maintain saturation >94% (hyperoxia may be detrimental in stroke)

Brain imaging (noncontrast CT scan)

Treatment:

Intravenous thrombolysis with rtPA: indicated within 3 hours of symptoms onset, and may be considered within 4.5 hours, when no contraindication to IV tPA (SBP needs to be ≤ 185 mmHg and DBP ≤ 110 mm Hg).

Endovascular treatment (intra-arterial thrombolysis or mechanical clot extraction): indicated for major stroke within 6 hours, due to occlusion of the middle cerebral artery, especially for those with contraindications for intravenous thrombolysis.

Anesthetic management:

- Choice of anesthesia: General vs. conscious sedation.
 - No difference in neurologic outcomes and no delay in time to reperfusion when comparing both strategies
 - Decision should be individualized (eg. patient cooperation or need to secure airway)
- Hemodynamic management:
 - Preferentially use continuous invasive monitoring (do not delay intervention to obtain arterial access as it can be obtained by the neurointerventionist)
 - Avoid hypotension: maintain SBP > 140 mm Hg and <180 mm Hg.
- Oxygenation and ventilation:
 - Supplement oxygen to maintain SpO₂>92% and PaO₂> 60 mmHg
 - Maintain normocapnia to avoid cerebral vasoconstriction
- Temperature: Target temperature between 35°C and 37°.
- Glycemic management: Maintain BG between 70 to 140 mg/dL. Initiate insulin treatment if BG>140 mg/dL.

Risk Factors:

Hypertension, Diabetes, Tobacco use, Hyperlipidemia, History of CAD

#2 Aneurysm Rupture- IR

Extravasation of contrast and/or signs of increased ICP

Stabilize Patient:

Increase FiO2 to avoid hypoxia

Hyperventilate the patient

If possible, elevate head of bed (reverse Trendelenburg)

Drugs:

Protamine: Discuss dosing with Interventionalist and base on most recent ACT

- Typically 1 mg Protamine neutralizes 100 U Heparin
- Less Protamine is needed as times increased from Heparin administration
- Administer test dose and then as slow IVP: can cause hypotension, rarely can result in HTN or anaphylaxis

Mannitol: 0.25- 1 g/kg infused over 15 min

- Caution in setting of CHF and severe renal disease with anuria

Nicardipine: Infusion rate: 2.5-15 mg/h

Treatment:

Be prepared to administer Protamine. **Discuss with Interventionalist first**

Take maneuvers to decrease ICP:

- Consider giving Mannitol
- Maintain blood pressure near baseline levels until bleeding is controlled. Once hemostasis is achieved consider increasing BP to maintain cerebral perfusion pressure in context of increased ICP
- Consider switching to a TIVA

Maintain euglycemia, normothermia, and control seizures

Ensure STAT neurosurgery consult has been placed and EVD kit is being brought to IR suite

Check labs including ACT and consider converting blood status to type and cross

Ensure OR control desk aware of possible emergency case if unable to achieve hemostasis in IR

The patient will likely remain intubated after procedure/surgery

#3 Intraoperative Aneurysm Rupture (IAR)

May present clinically as sudden hypertension followed by bradycardia and arrhythmias, brain bulge or hematoma, or bleeding from the surgical site.

Stabilize Patient:

Communicate with the surgeon regarding anticipated blood loss and surgical visibility

Initiate vasopressor support, infuse volume, increase FiO2 as needed, and consider transfusion

Verify blood products availability, consider MTP

Call for extra help

Drugs:

Adenosine: 0.3-0.6 mg/kg provides flow arrest of 12-50 seconds

Mannitol: 0.25- 1 g/kg infused over 15 min

- Caution in setting of CHF and severe renal disease with anuria

Propofol: 20-60 mg bolus, >125 mcg/kg/min infusion

Treatment:

Rupture before dural opening (IAR mainly occurs during hemodynamic swings and rapid changes in transmural pressure):

- Control abrupt increases in ICP with modest hyperventilation
- Immediate blood pressure control
- Minimize CMRO2 using IV anesthetic agents (propofol, barbiturates)
- Surgical decompression
- Osmotherapy with mannitol or 3% saline

Rupture after dural opening (IAR mainly occurs during dissection or clip placement):

- Reduce MAPs to ~50s acutely to decrease bleeding, improve visualization and soften aneurysm neck for clipping
- Consider Transient Flow Arrest with Adenosine
- Minimize CMRO2 using IV anesthetic agents (propofol, barbiturates)

* Hypothermia is not recommended regardless of Hunt and Hess Grade

* Reduction in MAPs must be balanced with maintaining perfusion of ischemic regions of the brain with impaired auto regulation

Common Causes:

Anesthetic factors: Response with intubation and emergence, coughing/gagging
Hypertension

ICP (sudden decrease): hyperventilation, use of large dose mannitol, CSF drain

Maneuvers: Valsalva, application of PEEP

Comorbidities: COPD, CAD, and hyperlipidemia

#4 Autonomic Hyperreflexia

Hypertensive episode, which can escalate to hypertensive crisis with reflexive bradycardia and dysrhythmias
Patients with spinal cord injury at T6 or higher are at increased risk (can appear 2-3 weeks after injury, typically will manifest within 1 yr)

Stabilize Patient:

Immediately inform the surgeon and ask the surgeon to stop operating
Deepen level of anesthetic
Increase FiO₂ to avoid hypoxia

Drugs:

Nitroglycerin: Consider bolus 50-100 mcg and titrate to effect. Infusion rate: 10-100 mcg/min

Nicardipine: Consider bolus of 0.1-0.2 mg and titrate to effect. Infusion rate: 2.5-15 mg/h

Treatment:

Administer short-acting, rapid onset anti-hypertensive

- Consider Nitroglycerin, Nicardipine, or Nitroprusside

Eliminate stimulus if applicable (i.e. empty bladder or bowel) and maintain deeper plane of anesthesia

Position patient with head up

Monitor for signs of: MI, hemorrhage, seizure, dysrhythmias (can progress to heart block) and treat accordingly

- Consider invasive monitoring

Differential Diagnosis:

Pain, inadequate anesthetic depth, uncontrolled baseline HTN, pre-eclampsia

Common Causes:

Visceral stimulus: bladder or bowel distention

Surgical stimulus

Infection: UTI, Pressure ulcers

Uterine contractions

#5 Bleeding During Spine Surgery

Verify Diagnosis: Increase in Blood Loss, Hypotension, Tachycardia, Oliguria

Stabilize Patient:

Control bleeding (surgical)
Replace plasma volume
Maintain BP with vasopressors as needed
Verify blood product availability, consider MTP

Drugs:

Tranexamic Acid: *Institutional protocols may vary and should be followed

- Low Dose: Load 1 mg/kg Infusion 1 mg/kg/hr
- High Dose: Load 30 mg/kg Infusion 1-10 mg/kg/hr

Treatment:

Confirm blood loss (suction canister, sponges, under drapes, etc.) and exclude other causes for hemodynamic problems
Ensure adequate vascular access if not secured already, communicate with blood bank re need for blood products
Consider use of red cell salvage techniques
Check labs: (ABG, Hct/Hb, thromboelastography)

- Correct deficiencies as indicated
- Goal Hb >8.0 (may vary based on patient co-morbidities)

Consider temporary wound packing until patient stabilized
Restore plasma volume and temporize with vasopressors
Ensure normothermia 36.0-37.9 °C
Consider antifibrinolytics (Tranexamic Acid) and Factor VIIa

Differential Diagnosis:

Overdose of anesthetic, pulmonary embolism, anaphylaxis, other forms of shock, or transfusion reaction.

Common Causes:

Multilevel spine surgery, tumor resection, trauma, infection
Revision surgery
Patient age, obesity, known coagulation defects

#6 Delayed Emergence after Craniotomy

Failure of emergence or resumption of baseline mental status after expected time frame of recovery from anesthetics has elapsed

Stabilize Patient:

Immediately correct hemodynamic instabilities (hypotension, hypoxia, hypercapnia, etc.) or hypothermia

Notify surgeon, perform neurological assessment in OR

Drugs:

Naloxone: 0.04 mg-0.1 mg, IV every 2-3 minutes PRN for reversing an opioid overdose

Flumazenil: 0.2 mg IV, repeat at 1 min intervals PRN. usual dose: 0.6-1 mg for reversing a benzodiazepine overdose (caution for seizure precipitation)

Treatment:

Check and reverse residual anesthetics if possible:

- Opioid: reverse with naloxone
- Benzodiazepines: reverse with flumazenil
- Nondepolarizing neuromuscular blockade: reverse with anticholinesterases or sugammadex (for rocuronium and vecuronium)
- Volatile agent, barbiturates, propofol, other sedatives like ketamine, lidocaine: allow adequate time for recovery

Correct metabolic/endocrinological derangements: send blood gas, drug screen or other relevant tests

- Hypo-/hyper-glycemia, hyponatremia, hypermagnesemia, severe acid-base disturbances, etc.
- Hyperammonemia, uremia, severe hypothyroidism, central anticholinergic syndrome, drug intoxication, etc.

If neurosurgical causes are suspected:

- Prepare for transporting patient for emergent CT/MRI or angiogram
- Keep OR available for potential emergent re-exploration or angiogram
- Consider EEG to rule out seizures

Differential Diagnosis:

- Residual anesthetics (see "Treatment" section)
- Metabolic/endocrinological derangements (see "Treatment" section)
- Neurosurgical causes: cerebral ischemia and vascular occlusion, intracranial hemorrhage, elevated intracranial pressure, cerebral edema, hydrocephalus, postictal state after intraoperative seizures, tension pneumocephalus, surgical trauma, etc.

#7 Management of Intraoperative Increased ICP

Verify clinical picture. **Subjective** (surgeon's visible judgment), **objective** (Hypertension, bradycardia, ICP monitoring) and **radiological** evidence(CT scan) of cerebral edema are the most common indications to institute intraoperative ICP lowering treatment

Stabilize Patient:

Maintenance of cerebral perfusion pressure with mild hypertension as indicated.

Ensure adequacy of oxygenation: PaO₂ >100mmHg

Ensure adequacy of ventilation: EtCO₂ ~35 mmHg

Glucose control: BG 140-180 mg/dL

Avoidance of hyperthermia: Temp <38C

Drugs:

20% Mannitol (0.25-2g/kg) in IV boluses, typical dose is 1g/kg

Hypertonic saline(1.6-7.5%) continuous infusion (0.1 to 1.0 mL/kg/hr)

IV Furosemide (5-20mg)

Steroids (tumors/vasogenic edema)

Barbiturate coma- only in special circumstances.

Pentobarbital, 10 mg/kg over 30 minutes (load), 5 mg/kg/hr for 3 hours, then 1 mg/kg/hr.

Treatment:

Optimize patient position to improve venous drainage (elevate head)

Maintenance of adequate anesthetic depth and analgesia

- IV anesthetic preferred: Propofol
- Adequate muscle relaxation in an intubated patient and prevention of coughing, bucking or straining on the end tracheal tube

Hyperventilation to lower ICP

- Should not be prophylactic

Check labs to evaluate for metabolic causes

CSF drainage

- Consider Intraoperative drainage of CSF with a ventriculostomy

Treat seizures

- Cold saline irrigation by surgeon

Assessment for treatable surgical causes

Common Causes:

Intracranial hemorrhage: Epidural, subdural, intracerebral hemorrhage

Subarachnoid hemorrhage from an aneurysm rupture

CSF flow obstruction: hydrocephalus

Increase in venous pressure: venous thrombosis, heart failure

Metabolic causes: hyponatremia, hypoosmolality, hepatic, or uremic encephalopathy

#8 Loss of Evoked Potentials in Spine Surgery

Threshold for alarm is based on monitoring modality and surgery being performed (It is a team decision) However for SSEP a decrease in amplitude >50% or an increase in latency >10% is generally considered significant. MEP >80% amplitude decrease is considered significant.

Stabilize Patient:

Notify all members of the OR team

Neurophysiology technician should check lead placement to make sure nothing has become dislodged

Anesthesia team will verify that no sudden changes to the anesthetic have been made and will ensure the patient is not hypoxic

Surgical team should consider reversal of any high-risk maneuvers performed prior to loss of signals

Treatment:

Increase MAP \geq 85mmHg for adolescents and adults. For younger children, the MAP should be 20% above their baseline.

Turn off volatile anesthetics/switch to TIVA (notify neurophysiology team you are doing this)

Ensure normothermia 36.0-37.9 °C

Ensure normocarbia

Check patient limb positioning

Check an ABG

- Correct acidosis if present
- Transfuse pRBCs if Hb is <8.0

Re-evaluation of evoked potentials should be ongoing while corrective actions are being made.

If abnormality remains consider:

- Wake-up test
- Emergent spinal cord imaging

Common Causes:

Surgical: distraction of spinal cord, derotation or deformity, screw misplacement, osteotomy, patient positioning

Anesthetic: Drug boluses, deep stage of anesthesia

Physiologic: hypotension, hypothermia

Technical: electrode disconnection

#9 Seizure During Craniotomy

Awake craniotomy: loss of consciousness, fluttering eyelids, repetitive/rhythmic jerking movements of head or limbs, stiffness and convulsions. Characteristic EEG or ECoG changes during GA

Stabilize Patient:

100% O₂, may perform bag-mask ventilation with oral airway or place LMA if necessary (for awake craniotomy)
Notify surgeon, halt surgical manipulations, place ice-cold saline on the cortical surface
Treat hemodynamic instabilities (hypotension, hypoxia, hyperthermia, etc.)

Drugs:

Midazolam 1-5 mg, diazepam 5-10 mg, lorazepam 2-5 mg IV
Propofol: IV in 0.5-1 mg/kg increments
Phenobarbital: 10-20 mg/kg IV, max 30 mg/kg, rate < 60 mg/min
Phenytoin: 20 mg/kg IV at up to 50 mg/min
Fosphenytoin: load 15-20 mg PE/kg IV at up to 150 mg PE/min

Treatment:

If seizure is mild and self-limited => surgery may be continued with cautions. Supplement O₂ and closely monitor airway patency, ventilation/oxygenation, consciousness (if awake) and vital signs

If seizure is not self-limited, administer incremental doses of IV propofol, midazolam or barbiturate to halt seizures

If airway patency or adequate ventilation/oxygenation are not maintained:

- Place LMA or endotracheal tube (if awake), consider video laryngoscope or fiberoptic bronchoscope to help with airway visualization
- Suction oropharyngeal area and consider OGT to prevent aspiration
- Place bite blocker and protect patient from traumatic injury

Work on the underlying causes:

- Metabolic derangements: hypoglycemia, hyponatremia, alkalosis/hyperventilation, severe hypocalcemia or hypomagnesemia
- Drug toxicity or withdrawal, inadequate serum anticonvulsant levels
- Surgical insults: cortical stimulation, brain injury, cerebral hemorrhage or ischemia, etc.
- Anesthetics with epileptogenic potential should be avoided

Consider EEG or ECoG monitoring if feasible

Discuss administration of antiepileptic drugs (e.g. phenytoin, keppra) with surgical team

Prepare for delayed emergence and prolonged mechanical ventilation after surgery

Differential Diagnosis:

If awake: dystonia, shivering (hypothermia), nonepileptic seizures, anxiety spells, migraine headaches, syncope, etc.

If under GA (not paralyzed): dystonia, shivering (hypothermia), etc.

#10 Cerebral Vasospasm

Changes in neurological exam after SAH, often blood pressure dependent. Diagnosis via TCD followed by CT angiography

Stabilize Patient:

Avoid hypotension, hyperthermia, hyperglycemia, hypovolemia, and hyponatremia.

Ensure euvolemia

Consider securing the airway

Drugs:

Nimodipine 60mg PO q 4hrs x 21 days *standard of care

Nicardipine 0.075 – 0.15 mg/kg/hr IV *consider if unable to give nimodipine

Treatment:

Modified Triple H therapy if vasospasm confirmed and aneurysm secured, otherwise risk of re-bleed.

- Hypertension by volume expansion and possible addition of vasopressors (Phenylephrine, Norepinephrine, Vasopressin, etc.).
- Hemodilution with either crystalloids or colloids, aim for Hct 30 – 35% (optimal balance between low viscosity and O2 carrying capacity)
- Euvolemia in combination with induced hypertension

Vasorelaxation:

- Calcium Channel Blockers: (Nimodipine 1st line, Nicardipine 2nd line)
- Magnesium: no difference in outcome

Experimental and less efficacious medical therapies:

- Endothelin Receptor Antagonists: Clazosentan (conflicting results and increased risk of pulmonary edema, hypotension and anemia)

Free Radical Scavengers: Tirilazad, Nicaraven, Ebselen

Thromboxane Inhibitors: Ozagrel

Anti-Inflammatory Treatment: Nafamostat, Methylprednisolone

Interventional Therapies:

- Intra-arterial medical therapies: Papaverine, Verapamil
- Mechanical treatment

Differential Diagnosis:

Aneurysmal rebleed, acute stroke, hydrocephalus, seizures

Common Causes:

Day 2 – 15 after subarachnoid hemorrhage, usually resolving between weeks 2 – 5 post hemorrhage

#11 Venous Air Embolism

Sudden decrease in EtCO₂, hypotension, decreased oxygen saturation, mill-wheel murmur on Doppler. Dyspnea/respiratory distress if awake.

Stabilize Patient:

Switch to 100% O₂

Notify surgeon, flood field with saline

Treatment:

Attempt to halt further air entry by getting the surgical site below the level of the heart

- Identify source (IV or central line vs. surgical source)
- Trendelenburg and/or left lateral decubitus (beware of Mayfield stabilizer in sitting cervical cases)
- Valsalva or bilateral jugular compression have been used to halt ongoing embolism

Chest compressions may help break up air lock

Turn PEEP off (may facilitate paradoxical embolism in patients with PFO)

Maintain systemic arterial pressure with fluid and inotropic agents as needed

Cardiac Collapse should be treated with ACLS protocols

Consider:

- Central line with attempted aspiration of air
- Arterial line placement (if not already present)
- TEE

Differential Diagnosis:

In the absence of Doppler or TEE confirmation of air:
Anaphylaxis, Acute MI, Bronchospasm, and Pulmonary Embolism.

Common Causes:

Caused by air entering the venous system potentially leading to RVOT obstruction.