Autonomic Dysreflexia

Autonomic dysreflexia is a condition seen in chronic spinal cord injury (typically the lesion must be above T6 – 85% of patients with lesion above T5 in Barash). It is the result of loss of coordinated autonomic responses to demands on HR and vascular tone, and is characterized by diffuse vasoconstriction (below the lesion) and hypertension with exaggerated bradycardia or other dysrhythmias. Common triggers include: bladder distention, bowel impaction, pressure sores, catheterization, painful peripheral stimulus below the lesion, sexual activity.

ANESTHETIC CONSIDERATIONS

1. Avoidance of peripheral triggers (EMLA prior to starting IVs, caution with bladder catherization)
2. Availability of direct vasodilators to treat hypertensive crisis
    a. If hypertensive crisis occurs, may progress to seizures, ICH, myocardial infarction
        i. Remove offending stimulus
        ii. Deepen anesthetic
        iii. Treat hypertension with direct acting vasodilators (nitroglycerin, nitroprusside, hydralazine)
3. Preference for neuraxial anesthetic technique
4. Anesthetic considerations for chronic spinal cord injury
    a. Level of injury
    b. Respiratory dysfunction: if lesion between C5-T7 (due to loss of abdominal and intercostals support; indrawing of flaccid thoracic muscles during inspiration produces paradoxical respirations and decreased VC by 60%)
    c. Decreased ability to cough (atelectasis and retained secretions)
    d. Increased susceptibility to CAD (long term)
    e. Hyperkalemia with succinylcholine
        i. Safe within 1st 48 hours
        ii. Maximal K increases between 4 weeks and 5 months after injury
    f. Temperature control (prone to hypothermia secondary to inability to vasoconstrict)
    g. Chronic catheterization (UTI, vesicoureteric reflux, urinary calculi, renal insufficiency)
    h. Pressure sores
    i. Contractures
    j. Chronic Pain

PRIORITY RESUSCITATION HISTORY EXAM LABS/INVESTIGATIONS PFTS (SPECIAL TESTS) REGIONAL(OPTIONS)

PRIORITY

- Emergent (no time for optimization)
- Urgent (some time for optimization)
- Elective (time for preoperative optimization)
    - Remove any offending triggers
        - Management of fecal impaction
        - Urinary Catheterization
    - Application of local anesthetic to IV sites/extremities
    - Regional anesthetic techniques if indicated

RESUSCITATION

- No specific considerations
- If spinal cord injury was within 3 months, spinal shock may still be present (complete cessation of spinal cord function below the level of the lesion – flaccid paralysis, loss of visceral and somatic sensation and paralytic ileus. Vasopressor reflexes are also lost. Spinal shock may persist from a few days to 3 months)

HISTORY

- Presenting complaint and reason for presentation to surgery
- Duration since onset of spinal cord injury
- Level of injury
    - C4-5 (significant respiratory insufficiency secondary to loss of diaphragmatic innervation)
    - C5-T7 (respiratory insufficiency secondary to loss of intrathoracic and intraabdominal muscle innervation and weak cough/inability to clear secretions)
    - Above T5-T6 (loss of splanchnic innervation and high risk for autonomic dysreflexia)
- Level of function
    - Quadriplegic
    - Paraplegic
    - Motor deficits (document especially if regional technique)
    - Sensory deficits (document especially if regional technique)
- History of autonomic dysreflexia
    - Triggers
    - Symptoms
    - Treatment
- Catherization
- Associated conditions
    - Coronary artery disease
    - Contractures
    - Osteoporosis
    - Pressure sores
        - Sepsis
    - Urinary tract symptoms
    - Renal insufficiency
    - Bowel motility issues

EXAM

- Airway examination (especially if previous C spine fixation – possible difficult airway)
- Focused cardiorespiratory examination

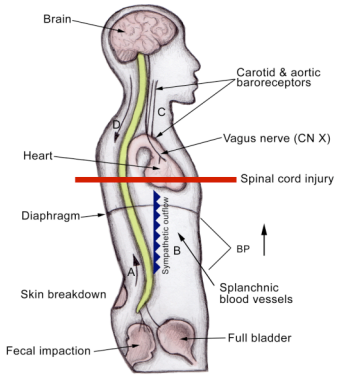
- Documentation of motor and sensory deficits
- IV access – peripheral examination

INVESTIGATIONS
- CBCcDiff (urosepsis, skin/soft tissue sepsis from pressure sores), lytes, BUN, Cr (renal insufficiency), INR, PTT

SPECIAL TESTS
- As indicated

ANESTHETIC OPTIONS
- None
- MAC
- Sedation
- GA (LMA – gastroparesis) vs ETT
- Regional
  - Neuraxial
  - Peripheral nerve blockade

ANESTHETIC SETUP
- Standard CAS monitors
- Consider 5 lead ECG if history of cardiac ischemia
- EMLA for IV insertion
- Standard emergency drugs (atropine, ephedrine, phenylephrine)
  - **NO SUCCINYLCHOLINE**
    - (may be ok after 5 months (Barash); conservative practitioners would say never)
  - Prefer direct acting vasoactive drugs
- Direct vasodilators to treat hypertensive crisis
  - Hydralazine
  - Nifedipine
  - Nitroglycerin
  - nitroprusside



PATHOPHYSIOLOGY:
This phenomenon occurs after the phase of spinal shock in which reflexes return. Individuals with injury above the major splanchnic outflow may develop autonomic dysreflexia (AD). Below the injury, intact peripheral sensory nerves transmit impulses that ascend in the spinothalamic and posterior columns to stimulate sympathetic neurons located in the intermediolateral gray matter of the spinal cord. The inhibitory outflow above the SCI from cerebral vasomotor centers is increased, but it is unable to pass below the block of the SCI. This large sympathetic outflow causes release of various neurotransmitters (norepinephrine, dopamine-b-hydroxylase, dopamine), causing piloerection, skin pallor, and severe vasoconstriction in arterial vasculature.1 The result is sudden elevation in blood pressure and vasodilation above the level of injury. Patients commonly have a headache caused by vasodilation of pain sensitive intracranial vessels.

Vasomotor brainstem reflexes attempt to lower blood pressure by increasing parasympathetic stimulation to the heart through the vagus nerve to cause compensatory bradycardia. This reflex action cannot compensate for severe vasoconstriction, explained by the Poiseuille formula, where pressure in a tube is affected to the fourth power by change in radius (vasoconstriction) and only linearly by change in flow rate (bradycardia). Parasympathetic nerves prevail above the level of injury, which may be characterized by profuse sweating and vasodilation with skin flushing.

Cameron and colleagues have found that site-directed genetic manipulation of fiber sprouting in the spinal dorsal horns in a cord compression rat model could alter the extent of hyperreflexia after bowel distention, indicating that endogenous spinal cord circuitry/neural sprouting plays a role in the pathophysiology of AD.

SUMMARY:
- **complete transection of spinal cord above T5-6 (above major splanchnic outflow)**
  - **peripheral stimulus (or noxious visceral stimulus) causes sympathetic discharge**
  - **hypertension ensues (due to systemic release of catecholamines)**
  - **carotid baroreceptors sense hypertension and exhibit supraspinal inhibitory outflow causing parasympathetic (reflex) response above level of lesion**
  - **supraspinal inhibitory outflow and parasympathetic response unable to descend below level of spinal cord transection**
  - **overall:**
    - **above level of lesion: hypertension and bradycardia (parasympathetic response) with vasodilation and sweating**
    - **below level of lesion: cutaneous vasoconstriction (sympathetic predominance)**

REFERENCES:
Barash 6th Edition Chapter 53 and Chapter 39
EMedicine: Autonomic Dysreflexia in Spinal Cord Injury
Stoelting's Anesthesia and Coexisting Disease