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1. A 20-year-old man sustained complete spinal cord transection 3 years ago from a gunshot wound. He has a sensory level of T4 and is scheduled for a cystoscopy. If he develops autonomic hyperreflexia, which of the following is MOST likely to occur prior to treatment?

A. Hypotension  
B. Tachycardia  
C. Hypertension  
D. Mydriasis

Go to Q 2
Autonomic hyper-reflexia leads to uncontrolled sympathetic response secondary to noxious stimuli resulting in a sudden rise in blood pressure (hypertension not hypotension) with dangerous consequences. It occurs in patients with an injury at level T6 or higher. The AH reaction is provoked by a noxious stimulus entering the spinal cord (like a surgical stimulus) below the level of injury. It usually occurs within the first six months after injury, but can occur up to 13 years later.

B. Tachycardia

The autonomic hyper-reflexia reaction usually causes bradycardia not tachycardia as a reflex response to hypertension. Besides bradycardia, other cardiac abnormalities may be encountered, such as cardiac arrhythmias (atrial fibrillation, premature ventricular contraction, and atrioventricular conduction anomalies). Like chronic hypertension, AH can lead to cardiovascular damage.
C. Hypertension

A noxious stimulus below the level of the lesion produces an afferent impulse that generates a generalized sympathetic response, which in turn results in vasoconstriction, most significantly in the splanchnic vasculature, which causes an increase in peripheral resistance and a shunting of blood into the general circulation. The combination of the increased vasoconstriction and the increased fluid load in the vascular space causes a potentially catastrophic increase in blood pressure (BP).

As the brain detects the hypertensive crisis through intact baroreceptors the parasympathetic nervous system is stimulated in an attempt to lower BP. The parasympathetic overactivity (and lack of sympathetic tone) above the level of lesion is responsible for the headache, flushing and sweating in the head and neck region and pupillary constriction.
2. Which of the following would be appropriate management in the setting of autonomic hyperreflexia.

A. Ask the surgeon to stop the stimulus
B. Put the patient in the trendelenberg position
C. Lighten the anesthetic
D. Administration of a beta blocker like metoprolol
A. Ask the surgeon to stop the stimulus

Early recognition of signs and symptoms of AD is a major key to immediate and appropriate treatment of this urgent condition. Inappropriate management may result in severe hypertension and complications such as seizures, intracranial and retinal hemorrhages, myocardial irregularities, coma, and even death. Since the reflex is initiated by noxious stimulus, the first thing to do would be to stop the stimulus.

The initial management of an episode of Autonomic Dysreflexia involves placing the patient in an upright position to take advantage of any orthostatic reduction in blood pressure. The next step in managing acute Autonomic dysreflexia must be to loosen any tight clothing. These procedures allow further blood pooling in vessel beds below the level of injury.
C. Lighten the anesthetic

Deepening the general anesthesia if already administered or prevention of the episode altogether by spinal anesthesia may be a good choice in these patients. The effectiveness of different anesthesia methods relies on blocking the nociceptive signals from the surgical site below the level of injury of the spinal cord.

Antihypertensive medication should preferably have a rapid onset and short duration of action. Direct vasodilators like nitroglycerin, nitroprusside, hydralazine, are all recommended. Isolated beta blockers in the setting of severe bradycardia would not be advisable.
3. A 55 year old male is undergoing asleep-aware craniotomy for tumor resection. During the awake phase he has developed seizures. What is the next best step?

A. Ask the surgeon to instill normal saline on the brain
B. Administer IV lidocaine
C. Administer thiopental
D. Administer ketamine

Go to Q 4
A. Ask the surgeon to instill normal saline on the brain

Seizure in the operating room is managed with immediate cessation of the cortical stimulation and gentle irrigation of the brain surface with a small amount of cold saline. Low dose propofol or midazolam can be used in addition for seizure control making sure to maintain a patent airway, adequate ventilation and cardiovascular stability.

B. Administer IV lidocaine

There are some studies reporting the use of intravenous lidocaine infusion for refractory status epilepticus resistant to conventional anti-epileptic therapy in neonates and pediatric age group (ref). Lidocaine by itself can cause seizures in toxic doses and would not be suitable in the setting of intraoperative seizures during awake craniotomy.

Sodium thiopental being a barbiturate has been used in refractory status epilepticus for seizure control. However, it is the best choice in this situation. Thiopental may delay emergence, interfere with further awake neurological testing, necessitate mechanical ventilation and cause cardiovascular depression.
Ketamine which is a NMDA antagonist has been used for treatment of refractory seizures. Although it maintains spontaneous ventilatory drive, it would not be the best option in the clinical setting described.
4. All the following regarding the use of dexmedetomidine as an adjunct for neurosurgical procedures are true except:

A. Reduces the hemodynamic response to laryngoscopy
B. Reduces postoperative pain
C. Useful for awake fiberoptic intubation
D. Has a context sensitive half life similar to remifentanil

Go to Q5
A number of medications like IV lidocaine, opioids, inhaled anesthetics, esmolol have been used for this purpose, dexmedetomidine has emerged as an effective drug when used for this purpose. Hypertensive response during intubation in neurosurgical patients may be associated with an increase in the intracranial pressure, intracranial bleed and prevention is important to maintain cerebral homeostasis. Dexmedetomidine is a highly selective and specific alpha two adrenergic agonist and decreases central adrenergic flow and decreases the response to laryngoscopy and intubation.
Effective analgesia after craniotomy remains one of the greatest challenges because analgesics must be administered judiciously in order to avoid opiate-related upper airway obstruction, PONV, and respiratory depression. An intraoperative infusion of dexmedetomidine has an opioid-sparing effect as demonstrated by reduced verbal pain scores post craniotomy, prolonged time to analgesic request and reduced amounts of opioids to control postoperative pain (ref).

Ref: J. Song et al: The Opioid-sparing Effect of Intraoperative Dexmedetomidine Infusion After Craniotomy: J Neurosurg Anesthesiol. 2015 Apr 30. [Epub ahead of print]
Adequate sedation in addition to topicalization of the airway is key to performing successful awake fiberoptic intubation in deserving cases. Many sedatives pose a significant risk of respiratory depression. Dexmedetomidine has the advantage of producing adequate analgesia and sedation without a significant decrease in respiratory drive. Dexmedetomidine has a unique sedative property, which is more like natural sleep. It allows patients to respond and co-operate despite sedation.

Dexmedetomidine has an onset-of-action of approximately 15 minutes after intravenous administration. Plasma concentrations typically stabilize after about an hour of constant drug infusion. Over the manufacturer-suggested dose ranges of 0.2-0.7 μg/kg/hr an elimination half-life ($t_{1/2}$) of between 2.0 and 2.5 hours. In contrast, remifentanil has a context-sensitive half-life of about 3 min, and an elimination half-life between 12 to 30 min.

5. Predictors of post operative cognitive dysfunction include all the following except

A. **Male sex**
B. **Lower educational group**
C. **Previous stroke without residual impairment**
D. **Higher age**
A. Male sex

Male sex has not been found to have a higher incidence of postoperative cognitive dysfunction.
Lower educational level is associated with prolonged cognitive decline (ref). There seem to be protective effects of higher educational level or better intellectual endowment against the development of cognitive decline. The mechanism of this protection may be an ability to compensate for acquired cognitive difficulties.
C. Previous stroke without residual impairment

Asymptomatic patients with a history of stroke have been reported to be more likely to have postoperative cognitive dysfunction.

D. Higher age

Age is an important factor. In addition harboring the apo Lipoprotein E allele according to some studies have shown increased incidence. Older patients with less education, higher American Society of Anesthesiologists physical status, more complicated surgery, and longer hospital stays were also more likely to exhibit POCD at hospital discharge.