Quiz 31

Hemodynamic Complications in Neuroanesthesia

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Start
1. A 36 y/o female patient is scheduled for resection of temporal-occipital tumor. She presented with headache, weakness and tremors. Possible causes of intra-op hypotension, bradycardia could be all EXCEPT:

A. Trigeminal cardiac reflex.
B. Venous Air Embolism.
C. Sudden decrease in ICP.
D. Anaphylaxis.

Go to Q2
A. TRIGEMINAL CARDIAC REFLEX.

This is true. Trigeminal Cardiac Reflex (TCR) should be considered whenever surgeons are working in the distribution of the trigeminal nerve. Patients may develop bradycardia and hypotension.

B. VENOUS AIR EMBOLISM (VAE).

This is true. VAE can occur either during trephination or when a sagittal sinus is opened during dissection of a cranial tumor. This can cause sudden hypotension, bradycardia/asystole depending on the amount and speed of air entrapment.

C. SUDDEN DECREASE IN ICP.

This is false. Sudden decrease in ICP would lead to hypotension tachycardia and not bradycardia, as the Cushing reflex dissipates.

This is true. Anaphylaxis can result in hypotension and bradycardia secondary to intra-op medications (latex, colloids, antibiotics and muscle relaxants most commonly). Usually anaphylaxis is associated with tachycardia, but histamine release can cause constriction of the coronaries causing ischemia of the SA node leading to slowing of the heart rate.

2. A 69y/ female patient undergoes a microvascular decompression for trigeminal neuralgia. Sudden bradycardia and hypotension is noted during surgery. Trigeminal cardiac reflex (TCR) is suspected. All are true regarding TCR except:

A. Occurs with peripheral or central stimulation of trigeminal nerve.
B. Hypocarbia is a significant risk factor for the reflex to occur.
C. Can present as bradycardia and hypertension.
D. Beta blockers can potentiate the reflex.

Go to Q3
A. OCCURS WITH PERIPHERAL OR CENTRAL STIMULATION OF THE TRIGEMINAL NERVE.

This is True. Trigeminal Cardiac Reflex can occur with manipulation of the trigeminal nerve anywhere in its entire course (intracranial or extracranial). Central TCR occurs with stimulation of the intracranial course of the trigeminal N- from Gasserian ganglion to the brain stem. Peripheral TCR occurs with stimulation of the trigeminal N outside the cranium to Gasserian ganglion.

B. B. HYPOCARBIA IS A SIGNIFICANT RISK FACTOR FOR THE REFLEX TO OCCUR.

This is False. Hypercarbia rather than hypocarbia is a risk factor for the development of TCR. Other risk factors are light anesthesia, hypoxia and acidosis.


C. CAN PRESENT AS BRADYCARDIA AND HYPERTENSION

This is True. The clinical importance of the TCR lies in the fact that it can present as sudden onset of sinus bradycardia, bradycardia terminating to asystole, asystole with no preceding bradycardia, arterial hypotension, apnea. However, there is coactivation of the parasympathetic and sympathetic system upon stimulation of the trigeminal pathway, which is more prominent in peripheral TCR resulting in bradycardia and hypertension, sometimes even tachycardia and ventricular ectopy. Central TCR is more of a vagal response.

BETA BLOCKERS CAN POTENTIATE THE REFLEX

This is True. Beta blockers potentiate the reflex by reducing the sympathetic response of the heart and hence augmenting the vagal response.

3. 55 YEAR OLD FEMALE UNDERGOING DEEP BRAIN STIMULATOR (DBS) PLACEMENT SURGERY UNDER MAC ANESTHESIA, SUDDENLY BECOMES ASYSTOLIC. BEZOLD-JARISCH REFLEX (BZR) IS SUSPECTED. PRECIPITATING FACTORS INCLUDE ALL EXCEPT:

A. Hypovolemia.
B. Beta blockers.
C. Epinephrine.
D. Sitting position.
A. HYPOVOLEMIA.

This is true. Hypovolemia sensed by the carotid sinus receptors, causes compensatory increase in heart rate, vasoconstriction and contractility. A hypercontractile empty ventricle stimulates the intramyocardial nonmyelinated type C vagal fibers potentiating a sudden withdrawal of sympathetic outflow, increasing vagal tone and causing bradycardia and hypotension.

This is false. Beta blockers may actually reduce the incidence of the reflex or have no/minimal reduction. Beta blockers have been suggested to diminish the adrenergic surge and ventricular mechanoreceptor activation and hence the hypercontractility of the empty ventricle that may trigger the reflex.

**BZR----** Triggering factors stimulate cardiopulmonary chemoreceptors and mechanoreceptors of LV wall which communicate with the unmyelinated vagal afferent type C fibers leading to Inhibition of medullary vasomotor center and increased parasympathetic tone--Triad of – bradycardia, hypotension, peripheral vasodilatation

This is True. Exogenous epinephrine absorbed from the local anesthetic/epinephrine mixture in the setting of hypovolemia leads to increased cardiac contractility, resulting in reflex arterial vasodilation (parasympathetic mediated) and subsequent vagal mediated bradycardia.

This is true. Sitting position leads to peripheral vasodilation and venous pooling, thus reducing venous return to the heart leading to empty ventricles and contributing to the BZR.

4. ALL ARE TRUE FOR MANAGEMENT/PREVENTION OF TRIGEMINAL CARDIAC REFLEX EXCEPT:

A. Communication between the surgeon and the anesthesiologist.
B. Regional anesthesia.
C. Preoperative anticholinergics.
D. Epinephrine
A. COMMUNICATION BETWEEN THE SURGEON AND THE ANESTHESIOLOGIST.

This is true. As TCR can happen even with the slightest manipulation of the trigeminal nerve, the surgeon should keep the anesthesiologist in the loop and keep him/her informed of the proximity of the nerve during dissection. Close monitoring of the HR and MABP by the anesthesiologist allows communication with the surgeon to interrupt the manipulation to avoid catastrophe and cessation of the reflex.

This is true. Local anesthetic infiltration/block of the nerve can provide prophylaxis against development of TCR. Peribulbar block using bupivacaine in patients for retinal detachment surgery provided significant reduction in the incidence and severity of the TCR. Blocks were more efficacious than topical application to prevent oculocardiac reflex component of TCR.


C. PREOPERATIVE ANTICHOLINERGICS.

This is false. Prophylactic administration of atropine or glycopyrrolate preoperatively does not provide protection against development of TCR. Cholinergic blockade reduces but does not totally prevent the lowering of the HR or BP.


This is true. If bradycardia and hypotension in TCR still persist after cessation of the stimulus, pharmacologic treatment is initiated. TCR results from both activation of the vagal cardio-inhibitory fibers and inhibition of the adrenergic vasoconstriction, hence may respond to epinephrine in addition to atropine/glycopyrrolate.

5. A 75 YEAR OLD FEMALE UNDERGOES CRANIOTOMY. DURING BURR HOLE PATIENT DEVELOPED HYPOTENSION, BRADYCARDIA AND ST ELEVATION. ALL ARE TRUE REGARDING CORONARY ARTERY SPASM (CAS) DURING NEUROSURGICAL CASES, EXCEPT:

A. Vagally mediated.
B. Female patients may be more susceptible
C. Stimulation of thalamus precipitates microvascular spasm
D. Occurs predominantly in patients with coronary artery disease
This is true. CAS during neurosurgical cases is secondary to vagal tonic reflex which is part of TCR--- peripheral TCR manifests as hypertension, tachycardia and ECG changes. The dura is supplied by the meningeal branch of the maxillary N and hence during burr hole a dural stretch can incite TCR and CAS.

B. FEMALE PATIENTS ARE MORE SUSCEPTIBLE

This is true. Female patients tend to have narrow coronaries and short cardiac cycle, which puts them at high risk of any CAS. Also after menopause the hormonal protection against heart disease declines.

C. STIMULATION OF THALAMUS PRECIPITATES CORONARY ARTERY SPASM.

This is true. Stimulation within the medial dorsal nucleus of thalamus and the substantia nigra cause increase in HR, BP. Hence in DBS surgery stimulation of the thalamic nuclei could incite CAS. Cardiac ischemic changes could also occur with the sympathetic surge caused by thalamic and hypothalamic stimulation.

D. OCCURS PREDOMINANTLY IN PATIENTS WITH CORONARY ARTERY DISEASE.

This is false. CAS can occur in patients undergoing neurosurgical procedures in the vicinity of the trigeminal nerve with no cardiac history. Most of the events are transient but can progress to myocardial infarction, ventricular fibrillation and asystole if the inciting factor is not removed or with improper management.