Shobana Rajan, M.D.  Attending Anesthesiologist, Albany Medical Center,  Albany, NY
Shaheen Shaikh, M.D.  Assistant Professor of Anesthesiology, University of Massachusetts Medical center,  Worcester, MA.

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A 55-year-old man who is scheduled to undergo carotid endarterectomy (CEA) has a persistent myocardial filling defect at three hours on a dipyridamole-thallium scan. Which of the following statements is correct?

(A) Coronary autoregulation is effective in this segment
(B) Coronary revascularization should precede CEA
(C) Isoflurane is contraindicated
(D) Myocardial infarction is impending
(E) There is a segment of nonviable myocardium
In the setting of severe coronary disease, perfusion reserve may be reduced from flow-limiting stenosis, endothelial dysfunction, and adrenergic stimulation.

When coronary arteries are narrowed by atherosclerotic disease, coronary auto regulation attempts to normalize myocardial blood flow by reducing the resistance of distal perfusion beds.

Fixed perfusion defects are usually due to myocardial scarring. Due to exhausted perfusion reserve in those areas of severe stenosis and blocked arteries, coronary auto regulation is unlikely to be effective.

Coronary revascularization should precede CEA

Results from the randomized, prospective CARP (Coronary Artery Revascularization Prophylaxis) trial demonstrated that an aggressive strategy of prophylactic coronary artery revascularization before vascular surgery in patients with stable cardiac symptoms does not improve short-term outcome or long-term survival. At 2.7 years after randomization, mortality was 22% and 23% in patients with and without revascularization, respectively. Postoperative MI, defined by increased troponin levels, occurred in 12% and 14% of patients with and without revascularization, respectively. The best approach to managing coexisting severe carotid and coronary disease remains uncertain.

Early studies indicated that isoflurane caused coronary steal and should therefore be avoided in patients with coronary heart disease. Subsequently, more detailed trials have disputed this and have shown that as long as coronary perfusion pressure is maintained, isoflurane does not cause coronary steal or myocardial ischemia.

Studies using EEG and regional cerebral blood flow (rCBF) measurements suggest that the critical rCBF (the rCBF below which EEG changes of cerebral ischemia occur) is lower for isoflurane than for halothane or enflurane. Sevoflurane is a good alternative because its critical rCBF in patients undergoing endarterectomy is similar to that determined with isoflurane and it may facilitate more rapid emergence.

Impending myocardial infarction has 3 common presentations:

- Onset of ischemic cardiac pain in a patient previously free of symptoms.
- Intensification of angina of effort in a patient with previous angina of several months' or years' duration.
- Recurrence of pain, at rest or on slight provocation, in a patient who has been pain free after a previous myocardial infarction.
Thallium201 is a monovalent cation with biologic properties similar to those of potassium. 201 Tl is a well-suited radionuclide for differentiation of normal and ischemic myocardium from scarred myocardium.

After initial uptake into the myocyte, an equilibrium is created between the intracellular and extracellular concentrations of thallium. After blood levels diminish during the redistribution phase, the equilibrium favors egress of thallium out of the myocyte. On the basis of that equilibrium, thallium concentration diminishes over time in zones of normal uptake while diminishing more slowly in zones with less initial thallium uptake (zone of ischemia).

Fixed perfusion defect is due to decreased blood flow to an area of heart muscle, due to permanently damaged muscle (essentially a scar in the heart muscle). When scarred myocardium is present, the initial rest or stress thallium defect persists over time; such deficits are termed irreversible or fixed defects.

Reference: Nuclear cardiology: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 16, 271-319
2. A 75-yr-old woman with significant carotid artery stenosis is scheduled for general anesthesia for repair of a fractured hip. Which of the following is the greatest disadvantage of using propofol as an induction agent in this patient? 

A) Decreased arterial blood pressure  
B) Pain during IV injection  
C) Prolonged apnea after induction  
D) Prolonged awakening  
E) Prolonged elimination half life
The most prominent effect of propofol is a decrease in arterial blood pressure during induction of anesthesia. Independent of the presence of cardiovascular disease, an induction dose of 2 to 2.5 mg/kg produces a 25% to 40% reduction of systolic blood pressure. Similar changes are seen in mean and diastolic blood pressure. The decrease in arterial blood pressure is associated with a decrease in cardiac output and cardiac index (±15%), stroke volume index (±20%), and systemic vascular resistance (15% to 25%). Left ventricular stroke work index also is decreased (±30%).

Anesthetic management goals for carotid endarterectomy include protection of the heart and brain from ischemic injury. Propofol appears safe and superior in terms of hemodynamic stability in carotid endarterectomy and prevention of myocardial ischemia in two studies listed below. Both the studies studied hemodynamic stability and incidence of myocardial ischemia during carotid endarterectomy and compared propofol with isoflurane.

Induction of anesthesia with propofol is often associated with pain on injection. Pain on injection is reduced by using a large vein (antecubital vein), avoiding veins in the dorsum of the hand, and adding lidocaine to the propofol solution or changing the propofol formulation. Pretreatment with opiates, nonsteroidal anti-inflammatory drugs, ketamine, esmolol or metoprolol, magnesium, , a clonidine-ephedrine combination, dexamethasone, dexmedetomidine and metoclopramide all have been tested with variable efficacy. However pain on injection is not a reason to avoid propofol at induction of anesthesia during a carotid endarterectomy.

Apnea occurs after administration of an induction dose of propofol; the incidence and duration of apnea depend on dose, speed of injection, and concomitant premedication. An induction dose of propofol results in a 25% to 30% incidence of apnea from the respiratory depressant effects of propofol. The duration of apnea occurring with propofol may be prolonged to more than 30 seconds. The incidence of prolonged apnea (>30 seconds) is increased further by addition of an opiate, either as premedication or just before induction of anesthesia. However, since we are going to manually control the patient’s respiration after induction, apnea will not be a problem during induction of anesthesia.

After a single bolus dose, whole blood propofol levels decrease rapidly as a result of redistribution and elimination. The initial distribution half-life of propofol is 2 to 8 minutes.

The context-sensitive half-time for propofol for infusions of up to 8 hours is less than 40 minutes. Because the required decrease in concentration for awakening after anesthesia or sedation with propofol is generally less than 50%, recovery from propofol remains rapid even after prolonged infusion.

The rapidity with which the drug level decreases is directly related to the time of infusion (i.e., the longer the drug is infused, the longer the half-time). Propofol, has a significantly short half-time (4-7 hrs) and this makes them more suitable for prolonged infusion.

3. EEG changes of cerebral ischemia during carotid endarterectomy occurs when regional cerebral blood flow is

A. 45-50 ml/min/100g
B. 35-40 ml/min/100g
C. 25-30 ml/min/100g
D. 15-20 ml/min/100g
E. 5-10 ml/min/100g

go to Q 4
Carotid endarterectomy requires temporary clamping leading to cerebral ischemia. The ipsilateral cerebral hemisphere will be dependent on collateral blood flow from the vertebral artery and contralateral carotid artery through the circle of Willis. Ischemic EEG changes may be seen with shunt malfunction, hypotension or cerebral emboli. Typical regional cerebral blood flow is 50-55ml/min/100g of brain tissue. EEG deterioration would not occur.
This is not the correct answer. EEG changes have not been observed at 35-40 ml/min/100g.
The above value is incorrect. EEG changes have not been observed at 25-30 ml/min/100g.
Changes in EEG are usually seen with flows between 15-20 ml/min/100g. However, analysis of xenon decay curves for regional cerebral blood flow along with EEG monitoring have provided some important insights. The critical rCBF values depend upon the volatile agent used along with nitrous oxide and is approximately 20, 15, 10 and 10 ml/100g of brain tissue for halothane, enflurane, sevoflurane and isoflurane respectively. The changes usually consist of replacement of the faster background activity with higher amplitude theta and lower amplitude irregular delta components. The changes usually reverse with placement of a shunt.

E. 5-10 ml/min/100g

In patients anesthetized with isoflurane critical regional cerebral blood flow for EEG changes measured was approximately 10-11ml/100g/min. Isoflurane and sevoflurane lower the critical regional cerebral blood flow at which EEG changes occur. In areas of focal cerebral ischemia such as carotid or middle cerebral artery occlusion, cerebral auto regulation of blood flow is lost and flow is dependent on perfusion pressure and blood volume.

Messick JM Jr et al, Anesthesiology, 1987 Mar; 66(3); 344-9
Grady RE et al, Anesthesiology 1998 Apr; 88(4): 892-7
4. A 59 year old man undergoing unilateral carotid endarterectomy under general anesthesia develops a heart rate of 31 beats/min during the surgical procedure. Which of the following is responsible for this effect.

A. Carotid body stimulation
B. Receptors in the carotid sinus
C. Autonomic hyperreflexia
D. Bezold-Jarisch reflex

[To Q 5]
A. Carotid body stimulation

The carotid body is a small cluster of chemoreceptors located near the bifurcation of the carotid artery. The carotid body detects changes in the composition of arterial blood flowing through it, partial pressure of oxygen and of carbon dioxide. Furthermore, it is also sensitive to changes in pH and temperature.

Carotid body denervation may occur after carotid endarterectomy as a result of surgical manipulation. Unilateral loss of carotid body function may result in an impaired ventilatory response to mild hypoxemia and is rarely of clinical significance. Bilateral carotid endarterectomy is associated with loss of the normal ventilatory and arterial pressure responses to acute hypoxia and an increased resting partial pressure of arterial CO2. In this situation, the central chemoreceptors are the primary sensors for maintaining ventilation, and serious respiratory depression may result from opioid administration.

Miller’s textbook of anesthesia, ch 69; pp 2155, ed 8
B. Receptors in the carotid sinus

Carotid sinus baroreceptors are present at the bifurcation of the common carotid artery. Manipulation of the carotid sinus during carotid surgery activates this reflex, leading to bradycardia, AV block, or even cardiac asystole. The afferents are carried via the glossopharyngeal nerve to the medulla oblongata and efferent impulses lead to the activation of the cardiac vagal efferent nerve fibers. Sustained bradycardia should be treated promptly by cessation of surgical manipulation and atropine. Infiltration of the carotid sinus with local anesthetic, e.g. 1% lidocaine can be used to prevent these episodes.

Carotid stenting procedures-related dysautonomia (CAS-D) (hypertension, hypotension, and bradycardia) is a frequently reported problem occurring in the periprocedural period. Alterations in autonomic homeostasis result from carotid sinus baroreceptor stimulation, which occurs particularly at the time of balloon inflation. The response can be profound enough to induce asystole or even complete cessation of postganglionic sympathetic nerve activity and needs to be watched for and treated.

Autonomic hyperreflexia is a syndrome of vascular instability commonly bradycardia, hypertension, facial flushing occurring after spinal cord injury usually above T7. The trigger to this response is usually a cutaneous, proprioceptive or visceral stimulus.
D. Bezold-Jarisch reflex

This is not the correct answer.
Reflex cardiovascular depression with vasodilation and bradycardia has been variously termed vasovagal syncope, the Bezold–Jarisch reflex and neurocardiogenic syncope. This response may occur during regional anesthesia, hemorrhage or supine inferior vena cava compression in pregnancy; these factors are additive when combined.
Treatment includes the restoration of venous return and correction of absolute blood volume deficits. Ephedrine is the most logical choice of single drug to correct the changes because of its combined action on the heart and peripheral blood vessels. Epinephrine must be used early in established cardiac arrest, especially after high regional anaesthesia.

Incorrect
Try again
A. Barbiturate coma produces greater cerebral protection than hypothermia to 17 deg C
B. Barbiturates decrease the cerebral metabolic rate for oxygen by decreasing cerebral blood flow
C. Somatosensory evoked potentials and the EEG are equally sensitive to suppression by barbiturates
D. When administered in doses sufficient to produce an isoelectric EEG, barbiturates decrease the cerebral metabolic rate for oxygen by 50%
Barbiturates reduce the metabolic activity concerned with neuronal signaling and impulse traffic (EEG activity) not the metabolic activity corresponding to basal metabolic function associated with providing energy for cellular homeostasis. The only way to suppress baseline metabolic activity related to cellular activity is through hypothermia which ensures greater cerebral protection than barbiturates.
Consequent to the reduction in CMRO2, a parallel reduction in cerebral perfusion occurs, leading to decreased cerebral blood flow (CBF) and ICP and not the vice versa. The ratio of CBF to CMRO2 remains unchanged.

Even though the mean arterial pressure (MAP) decreases, barbiturates do not compromise the overall CPP because the CPP = MAP − ICP. In this relationship, ICP decreases more than MAP after barbiturate administration and thereby preserves CPP.

B. Barbiturates decrease the cerebral metabolic rate for oxygen by decreasing cerebral blood flow

Incorrect
Barbiturates can cause an isoelectric EEG at a dose when evoked potentials are still preserved. SSEPs primarily monitor the integrity of the dorsal spinal columns. SSEPs are preserved when barbiturates are administered at regular doses. However higher blood levels can affect the latency and the amplitude.
D. When administered in doses sufficient to produce an isoelectric EEG, barbiturates decrease the cerebral metabolic rate for oxygen by 50%.

When the EEG became isoelectric, a point at which cerebral metabolic activity is roughly 50% of baseline, no further decreases in CMRO2 occurred. These findings support the hypothesis that metabolism and function of the brain are coupled. The effect of barbiturates on cerebral metabolism is maximized at a 50% depression of cerebral function, thus leaving all metabolic energy for the maintenance of cellular integrity.

Millers anesthesia ch 17, 387-422, Ed 9