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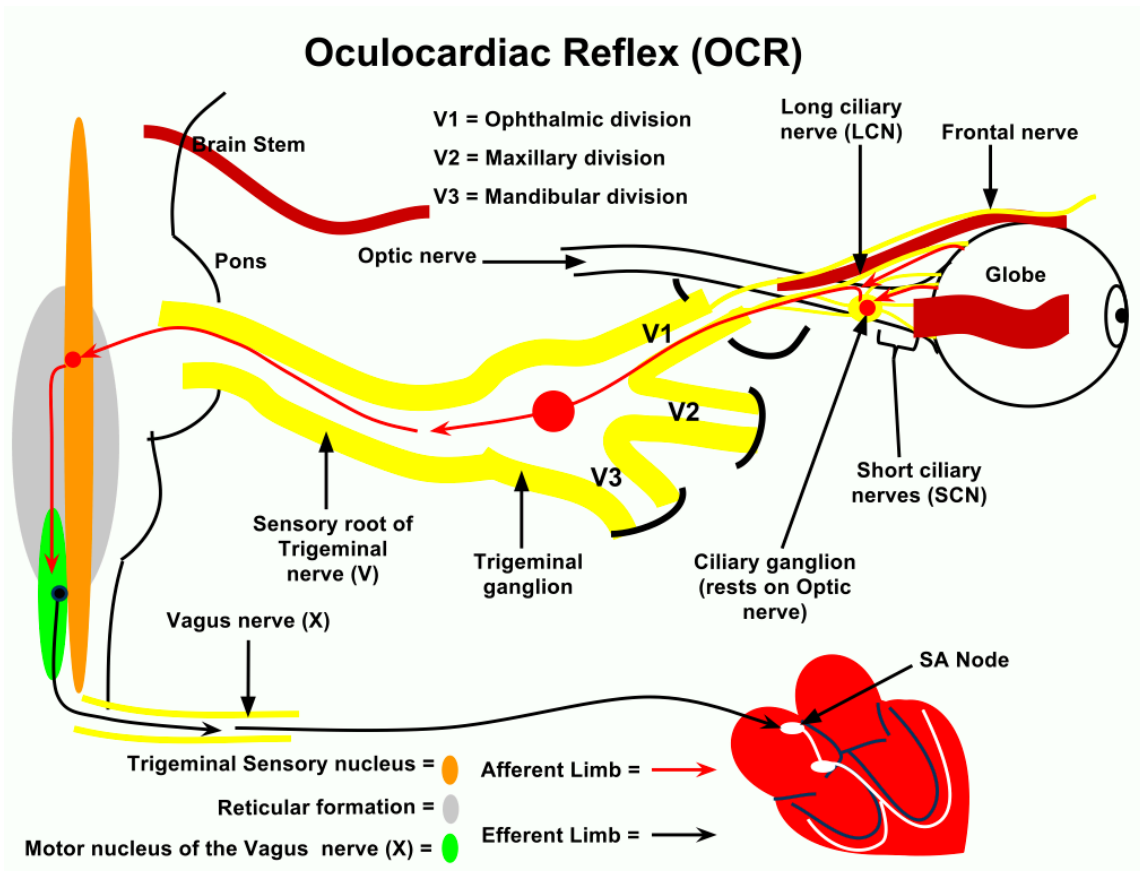
Sample question #1

A 52-year-old female is undergoing an anterior orbitotomy. When traction is applied to the eye, her heart rate suddenly decreases from 75 to 40 bpm. Which of the following are the **CORRECT** afferent and efferent limbs of this reflex, respectively?

- A. Optic nerve, oculomotor nerve
- B. Oculomotor nerve, vagus nerve
- C. Trigeminal nerve, vagus nerve**
- D. Optic nerve, facial nerve
- E. Vagus nerve, trigeminal nerve

This scenario depicts the oculocardiac reflex (OCR) which includes the trigeminal (afferent) and vagus (efferent) nerves (**C**).

The afferent limb of the OCR is via the TRIGEMINAL nerve (cranial nerve V), primarily via the ophthalmic division (V1). The impulse travels along the long and short ciliary nerves (LCN and SCN). The SCN synapses on the ciliary ganglion, which rests above the optic nerve. The impulse continues through the trigeminal ganglion via V1, and continues to the sensory nucleus of the trigeminal nerve. The synapse between the afferent and efferent limbs is at the visceral motor nucleus of the vagus nerve in the reticular formation of the brain stem. The efferent limb is via the VAGUS nerve (cranial nerve X), which then synapses on the sinoatrial node of the heart.



The OCR can be triggered by pressure on the globe; traction on the extraocular muscles, conjunctiva, or orbital structures; regional ophthalmic anesthesia such as a retrobulbar or peribulbar block; ocular trauma; or pressure on tissue within the orbital apex after enucleation. The reflex typically triggers sinus bradycardia, though other arrhythmias including junctional rhythm, AV blockade, multifocal PVCs, ventricular tachycardia, or asystole may occur.

The optic nerve (II) and oculomotor (III) nerve describe the afferent and efferent limb of the pupillary reflex (**A**).

The optic nerve (II) and the facial nerve (VII) describe the afferent and efferent limb of the corneal blink reflex (**D**).

BOTTOM LINE: The trigeminal nerve (ophthalmic division, V1) provides the afferent limb of the OCR. The vagus nerve (X) provides the efferent limb.



Evoked Potential: During ophthalmic surgery, the first line of treatment for bradycardia is to discontinue manipulation of the eye until the heart rate returns to baseline. Prophylactic atropine or glycopyrrolate prior to ocular manipulation may be of benefit if preexisting bradycardia or beta-blockade is present.

Source:

Barash, *Clinical Anesthesia*, 6th edition, p. 1327.
Standing, *Gray's Anatomy*, 40th edition, pp. 276, 667-669.

Sample question #2

Which of the following local anesthetics has the **LOWEST** cardiac-to-CNS dose toxicity ratio and the **HIGHEST** relative potency for cardiac toxicity?

- A. Ropivacaine
- B. Lidocaine
- C. Mepivacaine
- D. Bupivacaine**
- E. Levobupivacaine

Bupivacaine (**D**) has the lowest cardiac-to-CNS dose toxicity ratio (2:1) AND the highest relative potency for cardiac toxicity amongst the local anesthetics listed above. This cardiac toxicity is due to bupivacaine's stronger affinity for both resting and inactivated sodium channels within the myocardium compared with other local anesthetics.

All local anesthetics slow cardiac conduction to some degree. They may produce both direct and indirect cardiotoxicity through blockage of the INTRACELLULAR portion of cardiac SODIUM ion channels. This results in impairment of cardiac automaticity and conductance of cardiac action potentials. This can be observed on ECG as prolongation of the PR interval and widening of the QRS complex. Bupivacaine has been shown to dissociate from sodium channels during diastole at a rate much slower than lidocaine. This slower dissociation impairs complete recovery of sodium channels following hyperpolarization.

Higher serum levels of local anesthetics are required for cardiotoxicity compared to those required to cause CNS toxicity. Local anesthetic potential for causing cardiotoxicity is directly proportional to potency of the agent, with greater cardiotoxic potential favoring agents with greater potency and higher lipid solubility. For example, bupivacaine's cardiac-to-CNS dose toxicity ratio is 2:1, whereas lidocaine's ratio is 7:1 (**B**). The lower ratio associated with bupivacaine indicates that the cardiotoxicity may occur at serum levels that are relatively closer to levels sufficient for CNS toxicity. This is why bupivacaine increases the frequency of reentrant cardiac arrhythmias at serum concentrations slightly above the seizure threshold.

Local anesthetic	Cardiac-to-CNS toxicity dose ratio
Bupivacaine	2.0
Levobupivacaine	2.0
Ropivacaine	2.0
Mepivacaine	7.0
Lidocaine	7.0

Levobupivacaine (**E**) is a single-optical isomer of bupivacaine. It has an improved safety profile with less potential for CNS toxicity and cardiovascular collapse as the levo-isomer has a reduced affinity for brain and cardiac tissue. The cardiac-to-CNS toxicity dose ratio is, however, similar to bupivacaine (see table below).

Ropivacaine (**A**) and mepivacaine (**C**) have also been shown to have reduced potential for causing CNS and cardiotoxicity. Ropivacaine, however, has a similar cardiac-to-CNS toxicity dose ratio to that of bupivacaine. Mepivacaine (like lidocaine) has a higher ratio than bupivacaine.

BOTTOM LINE: Bupivacaine has the lowest cardiac-to-CNS dose toxicity ratio (2:1) AND the highest relative potency for cardiac toxicity of all local anesthetics. Bupivacaine's higher relative lipophilicity and potency promote stronger affinity for both resting and inactivated sodium channels within the myocardium.

Source:

Barash, Clinical Anesthesia, 6th edition, pp. 540-544.

Sample question #3

According to the ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation, which of the following patient descriptions has the strongest recommendation for perioperative use of beta blocker therapy?

- A. Low risk surgery, one clinical risk factor
- B. Vascular surgery, one clinical risk factor
- C. Intermediate risk surgery, currently on beta blockers for hypertension**
- D. Intermediate risk surgery, two clinical risk factors
- E. Vascular surgery, two clinical risk factors

A patient undergoing intermediate risk surgery that is on preoperative beta blockers (**C**) has the highest class of evidence for perioperative beta blocker use (Class I, level C). Patients on preoperative beta blockers should continue to use them throughout the perioperative period no matter the type of surgery.

To understand this answer it is important to know there are two types of evidence: class (I, IIa, IIb, II) and level (A, B, C). Below is a reproduction of the table used in the article ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation.

	Class I Benefit >>>Risk Procedure/treatment SHOULD be performed or administered	Class IIa Benefit>>Risk Additional studies with focused objectives needed. IT IS REASONABLE to perform procedure / administer treatment	Class IIb Benefit ≥ Risk Additional studies with broad objectives needed. Additional registry data would be helpful. Procedure / treatment MAY BE CONSIDERED	Class III Risk ≥ Benefit No additional studies needed. Procedure / treatment should NOT be performed or administered SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL
Level A Multiple population risk evaluated	Recommendation that procedure or treatment is useful/effective. Sufficient evidence from multiple randomized trials or meta analysis	Recommendation in favor of treatment or procedure being useful/effective. Some conflicting evidence from multiple randomized trials or meta analysis.	Recommendation usefulness/efficacy less well established. Greater conflicting evidence from multiple randomize trials or meta analysis.	Recommendation that procedure or treatment not useful/effective and may be harmful. Sufficient evidence from multiple randomized trials or meta analysis.
Level B Limited population risk strata evaluated	Recommendation that procedure or treatment is useful/effective. Limited evidence from single randomized trial or non-randomized studies	Recommendation in favor o treatment or procedure being useful/effective. Some conflicting evidence from single randomized trial or non- randomized studies.	Recommendation usefulness/efficacy less well established. Greater conflicting evidence from single randomized trial or non-randomized studies	Recommendation that procedure or treatment not useful/effective and may be harmful. Limited evidence from single randomized trial or non- randomized studies.
Level C Very limited population risk strata evaluated	Recommendation that procedure or treatment is useful/effective. Only expert opinion, case studies, or standard of care.	Recommendation in favor of treatment or procedure being useful/effective. Only diverging expert opinion, case studies, or standard of care.	Recommendation useful/efficacy less well established. Only diverging expert opinion, case studies, or standard of care.	Recommendation that procedure or treatment not useful/effective and may be harmful. Only expert opinion case studies, or standard of care.

Patients undergoing vascular surgery that have ischemia on preoperative testing are the only types of patients with a higher class and level of evidence for perioperative beta blocker therapy, Class I Level B, but that was not an answer choice.

Low risk surgery, one clinical risk factor (**A**): no recommendation

Vascular surgery, one (**B**) or two (**E**) clinical risk factors – Class IIa Level B

Intermediate risk surgery, one or more clinical risk factors (**D**) – Class IIb Level C

Vascular surgery, no risk factors – Class IIb Level B

BOTTOM LINE: The strongest recommendations (Class I) for perioperative use of beta blockers are for patients undergoing any type of surgery who are taking preoperative beta blockers or for patients with active cardiac risk factors undergoing vascular surgery who are not already on beta blockers.

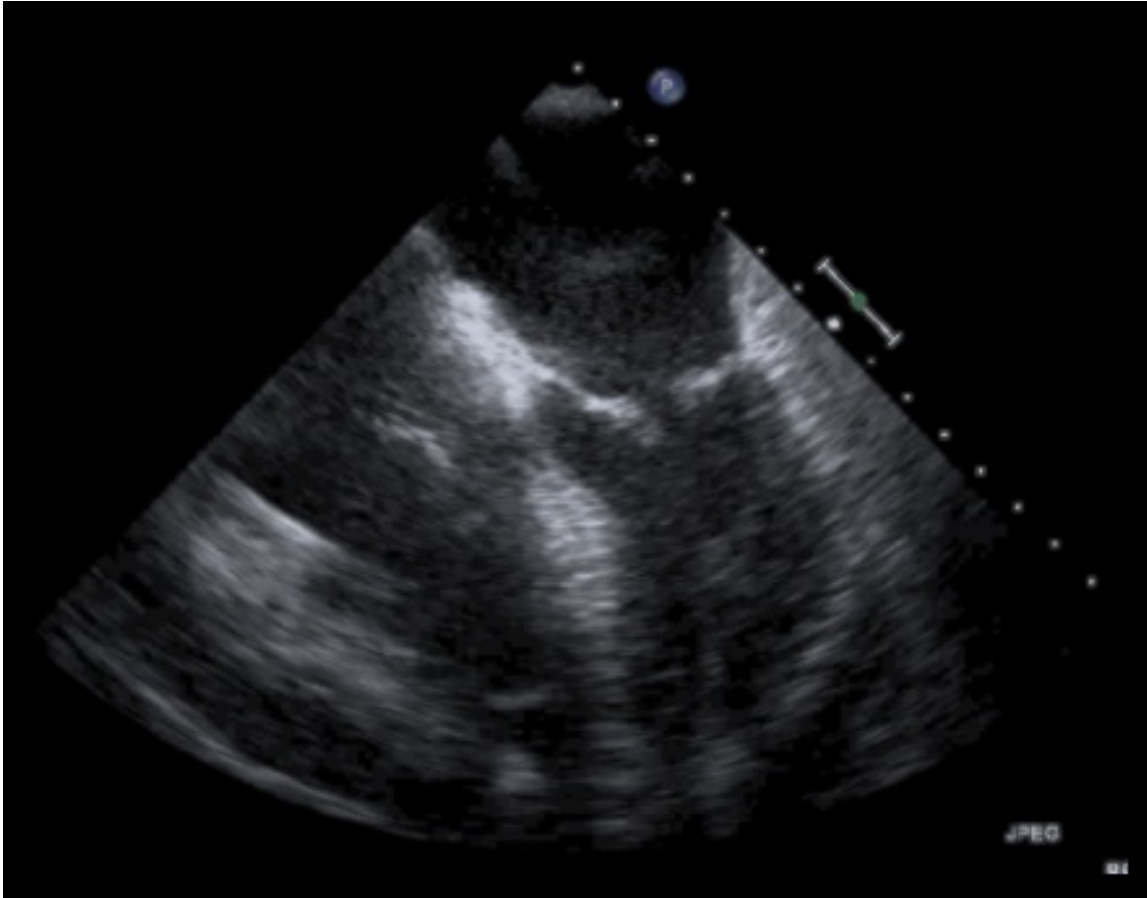
Source:

Fleisher L, et al. ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to

Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *Circulation*. 2007; 116: e418-e499. (See especially e460).

Sample question #4

Which of the following diagnoses can be **CORRECTLY** made from the TEE image shown below?

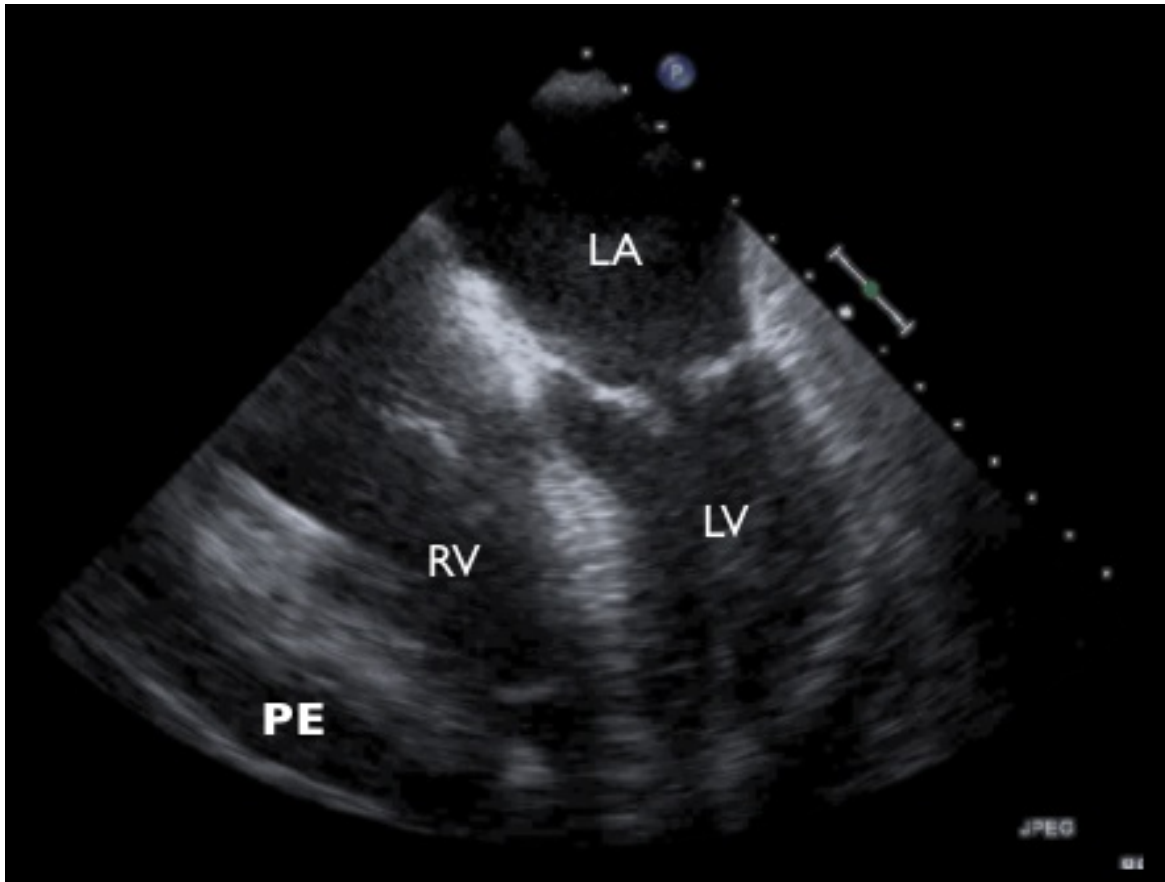


- A. Hypovolemia
- B. DeBakey Type 1 aortic dissection
- C. Papillary fibroelastoma
- D. Atrial myxoma
- E. Pericardial effusion**

A pericardial effusion (**E**) is present in the lower left corner of the image (see below).

Pericardial effusion is an important diagnostic consideration in patients presenting with tachycardia, low cardiac output, and high filling pressures. In theory, pericardial tamponade can be differentiated from myocardial failure by the presence of pulsus paradoxus (a drop of at least 10 mm Hg (or >9%) in systolic arterial blood pressure on inspiration), however, TEE can be essential

to perioperative diagnosis. Pericardial effusion and hematoma are characteristically identified as echo-free spaces between the epicardium and the pericardium.



PE = Pericardial effusion, LV = Left ventricle, RV = Right ventricle, LA = Left atrium

Echocardiographic signs of pericardial tamponade

Moderate to large pericardial effusion (shown in image)

Right and/or left ventricular collapse during diastole

Right and/or left atrial during systole

Decreased left ventricular filling with inspiration

Increased right atrial and ventricular filling with inspiration

The image shown in the question is not diagnostic for the remaining answer choices (A,B,C,D).

BOTTOM LINE: Echocardiography is a useful tool in diagnosing pericardial effusion and guiding intraoperative drainage. Drainage of a pericardial effusion (diagnostic finding) causing pericardial

tamponade (physiologic finding) results in propulsion of forward cardiac flow and a de-equalization of intracardiac pressures.

Source:

Wilson, Trauma: Critical Care, 1st edition, pp. 393-396.

Perrino, A Practical Approach to Transesophageal Echocardiography, 2nd edition, pp. 161-162, 355t.

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